UNITED EUROPEAN GASTROENTEROLOGY

ueg week

sharing the future of digestive health

Postgraduate Teaching Programme (PGT)

Syllabus

Postgraduate Teaching Programme
Overview

Saturday, October 19, 2019

	Hall 6 Voting Room	Room A2	Room C3 Voting Room	Room F3 Voting Room
09:00 – 10:30	**Plenary I**			
	Update on diverticulitis			
	10:30 – 11:00 Coffee Break			
11:00 – 13:00	**Video Case Session**	**Parallel Sessions**		
	Endoscopic retrograde cholangiopancreatography (ERCP)	Gastric polyps and submucosal lesions	How to use drugs in IBD	Chronic pancreatitis
	13:00 – 14:00 Lunch Break			
14:00 – 16:30	**Video Case Session**	**Parallel Sessions**		
	Diagnosis and management of early neoplasia of the upper GI tract	Monitoring of disease activity in IBD	Complications of severe acute pancreatitis	Advanced colorectal cancer

Sunday, October 20, 2019

	Hall 6 Voting Room	Room A1	Room A2	Room A3	Room C1	Room C2	Room C3 Voting Room	Room F1	Room F2	Room F3 Voting Room
08:30 – 10:30	**Parallel Sessions**		**Parallel Sessions**				**Parallel Sessions**		**Parallel Sessions**	
	Ulcerative colitis: Current management		Neuroendocrine tumours				How to improve quality in endoscopy		Obstruction and ileus	Therapy update: Faecal transplantation
	10:30 – 11:00 Coffee Break									
11:00 – 13:00	**Clinical Case-Based Sessions**									
	Constipation: To modify behaviour or anatomy?	Anorexia and unexplained weight loss	Oesophageal cancer: Challenges and controversies	Challenging the small bowel: Diagnosis and management of small bowel tumours in 2019	Autoimmune pancreatitis: From diagnosis to treatment	Acute liver failure: An era of expectation	Peptic ulcer disease	Proctology: Solutions for daily problems	Oropharyngeal swallowing disorders: Why gastroenterologists should be interested!	Evaluation and management of patients with alcohol-related liver disease: A multidisciplinary approach
	13:00 – 14:00 Lunch Break									
14:00 – 16:00	**Plenary II**									
	Complications of liver cirrhosis									

Update on diverticulitis

09:00-10:30 / Hall 6

Diverticulitis: Where are we now and where are we going?

Not available

EAES & SAGES Guidelines on diverticulitis

Patricia Sylla, United States

Learning Objectives:

- Review key findings of the 2018 EAES and SAGES consensus conference on the management of acute diverticulitis
- Review updated recommendations regarding management of acute diverticulitis based on review of the best available evidence
- Explore areas of persisting disagreement with adoption of consensus recommendations into clinical practice

Abstract:

Since the EAES 1999 consensus conference on diverticular disease, a growing number of studies and trials on the management of acute diverticulitis (AD) have been published reflecting significant changes in the medical and surgical management of uncomplicated and complicated AD. This presentation will review the most notable results from the recent 2018 SAGES and EAES consensus conference on AD management. Specially, updated recommendations regarding the epidemiology, diagnosis, management of uncomplicated and complicated AD, emergency and elective operative AD management will be reviewed, as well as the level of evidence in support of these recommendations. Areas of controversy, ie where consensus could not be achieved based on members' disagreement with expert recommendations will be highlighted, as well as recommendations with a low likelihood of changing members' current practice. Future research will focus on elucidating the reasons for low adherence to clinical recommendations supported by high level of evidence, and uncovering strategies to increase adoption.

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Kohler L, Sauerland S, Neugebauer E (1999) Diagnosis and treatment of diverticular disease: results of a consensus development conference. the Scientific Committee of the European Association for Endoscopic Surgery. Surg Endosc 13(4):430-436. Francis NK, Sylla P, Abou-Khalil M, Arolfo S, Berler D, Curtis NJ, Dolejs SC, Garfinkle R, Gorter-Stam M, Hashimoto DA, Hassinger TE, Molenaar CJL, Pucher PH, Schuermans V, Arezzo A, Agresta F, Antoniou SA, Arulampalam T, Boutros M, Bouvy N, Campbell K, Francone T, Haggerty SP, Hedrick TL, Stefanidis D, Truitt MS, Kelly J, Ket H, Dunkin BJ, Pietrabissa A (2019) EAES and SAGES 2018 consensus conference on acute diverticulitis management: evidence-based recommendations for clinical practice. Wexner SD, Talamini MA (2019) EAES/SAGES consensus conference on acute diverticulitis: a paradigm shift in the management of acute diverticulitis. Surg Endosc 33(9):2724-2725. Surg Endosc 33(9):2726-2741.

Disclosure:

Nothing to disclose

ESCP Guidelines on diverticulitis

Marja A. Boermeester, Netherlands

Learning Objectives:

- Patients with acute uncomplicated diverticulitis (except those immunocompromised) do not require antibiotics routinely
- Routine (endoscopic) follow-up is not recommended in asymptomatic patients after acute diverticulitis
- Primary anastomosis with or without diverting ileostomy can be performed in selected patients (hemodynamically stable, immunocompetent) with Hinchey III or IV diverticulitis

Abstract:

Acute diverticulitis and its treatment are surrounded by believes and traditions, and contradicting guidelines [1] EAES and SAGES 2018 consensus conference on acute diverticulitis has been published this year. The European Society of Coloproctology (ESCP) initiated a new evidence-based guideline on diagnosis and treatment of diverticulitis using GRADE; the final version of the guideline will be launched in a few months. Here, a pre-release look at the concept guideline.

Traditionally, all patients were treated with antibiotics but benefits of this strategy have never been proven. The Scandinavian randomized AVOD trial [2] in 2012 and the short- and long-term results of the Dutch DIABOLO trial [3,4] have shown no benefits of antibiotics on outcome, quality of life or in costs.

Most guidelines have not been updated and still recommend routine colonoscopy after acute diverticulitis. A recent systematic review including a meta-analysis alters recommendations concerning follow-up after acute diverticulitis. [5] An important conclusion of this meta-analysis is that acute diverticulitis does not enhance the risk of future colorectal carcinoma. Moreover, the risk of colorectal carcinoma being present at initial presentation is comparable among patients with CT-diagnosed acute diverticulitis and asymptomatic controls.

When a patient is diagnosed with perforated diverticulitis and has a purulent (Hinchey III) or faecal (Hinchey IV) peritonitis, emergency surgery is indicated. Many surgeons still believe that Hartmann's procedure is the best treatment. However, randomized trials have compared sigmoid resection and primary anastomosis with Hartmann's procedure. [6-9] Morbidity or mortality are comparable for resection with primary anastomosis and sigmoid resection with end colostomy. Primary anastomosis gives a lower stoma rate, but also the number of permanent stomas is reduced. This applies to patients who are hemodynamically stable and immunocompetent.

References:

[1] Vennix S, Morton DG, Hahnloser D, Lange JF, Bemelman WA. Systematic review of evidence and consensus on diverticulitis: an analysis of national and international guidelines. Colorectal Dis 2014;16(11): 866-878. [2] Chabok A, Pahlman L, Hjern F, Haapaniemi S, Smedh K, Group AS. Randomized clinical trial of antibiotics in acute uncomplicated diverticulitis. Br J Surg 2012;99:532-9. [3] Daniels L, Unlu C, de Korte N, et al. Randomized clinical trial of observational versus antibiotic treatment for a first episode of CT-proven uncomplicated acute diverticulitis. Br J Surg 2017;104:52-61. [4] Van Dijk ST, Daniels L, Ünlü Ç, de Korte N, van Dieren S, Stockmann HB, Vrouenraets BC, Consten EC, van der Hoeven JA, Eijsbouts QA, Faneyte IF, Bemelman WA, Dijkgraaf MG, Boermeester MA; Dutch Diverticular Disease (3D) Collaborative Study Group. Long-Term Effects of Omitting Antibiotics in Uncomplicated Acute Diverticulitis. Am J Gastroenterol. 2018 Jul;113(7):1045-1052. [5] Rottier SJ, van Dijk ST, van Geloven AAW, Schreurs WH, Draaisma WA, van Enst WA, et al. Meta-analysis of the role of colonoscopy after an episode of left-sided acute diverticulitis. Br J Surg. 2019;106(8):988-97. [6] Binda GA, Karas JR, Serventi A, Sokmen S, Amato A, Hydo L, et al. Primary anastomosis vs nonrestorative resection for perforated diverticulitis with peritonitis: a prematurely terminated randomized controlled trial. Colorectal Dis. 2012;14(11):1403-10. [7] Bridoux V, Regimbeau JM, Ouaissi M, Mathonnet M, Mauvais F, Houivet E, et al. Hartmann´s Procedure or Primary Anastomosis for Generalized Peritonitis due to Perforated Diverticulitis: A Prospective Multicenter Randomized Trial (DIVERTI). J Am Coll Surg. 2017;225(6):798-805. [8] Oberkofler CE, Rickenbacher A, Raptis DA, Lehmann K, Villiger P, Buchli C, et al. A multicenter randomized clinical trial of primary anastomosis or Hartmann´s procedure for perforated left colonic

diverticulitis with purulent or fecal peritonitis. Ann Surg. 2012;256(5):819-26; discussion 26-7. [9] Lambrichts DPV, Vennix S, Musters GD, Mulder IM, Swank HA, Hoofwijk AGM, et al. Hartmann´s procedure versus sigmoidectomy with primary anastomosis for perforated diverticulitis with purulent or faecal peritonitis (LADIES): a multicentre, parallel-group, randomised, open-label, superiority trial. Lancet Gastroenterol Hepatol. 2019;4(8):599-610.

Disclosure:
Nothing to disclose

Gastric polyps and submucosal lesions

11:00-13:00 / A2

Epidemiology, histopathological features and genetic predisposition

Lodewijk Brosens, Netherlands

Learning Objectives:

- Knowledge of the different types of epithelial polyps in the stomach
- Understand the etiology, pathogenesis and prognosis of different gastric polyps
- Know the main somatic genetic alterations in different types of gastric polyps and association with hereditary conditions

Abstract:
Gastric polyps are diagnosed in 1-4% of gastroscopies in the general population. Most polyps are of epithelial origin, but subepithelial lesions such as neuroendrocrine tumors, pancreatic heterotopia, lymphoma and mesenchymal tumors can also present as polyps.
There are geographical differences in prevalence of various gastric polyps, mainly due to differences in Helicobacter pylori (HP) infection. In countries with high rates of HP infection, hyperplastic and adenomatous polyps are more prevalent. In Western countries, with low rates of HP infection, fundic gland polyps (FGP) are the most prevalent type of polyp (up to 77% of all gastric polyps).
Hyperplasic polyps are the second most common gastric polyp in Western countries(~15% of all gastric polyps). Most hyperplastic polyps arise in the antrum and are a hyperproliferative response to tissue injury and typically occur in patients with Helicobacter pylori or autoimmune chronic gastritis, atrophy and intestinal metaplasia.
Gastric adenomas account for < 1% of all gastric polyps and are the third most common type of gastric polyp in Western patients. Gastric adenomas are further subclassified as gastric foveolar-type, intestinal-type, and pyloric gland adenoma.
Gastric hamartomatous polyps exclusively occur in the setting of rare hamartomatous polyposis syndromes, but gastric hamartomatous polyps are hard to differentiate from gastric hyperplastic polyps. Knowledge of the clinical context is essential to diagnose a gastric hamartomatous polyp.
The vast majority of gastric polyps are sporadic, but some gastric polyps indicate an underlying syndrome. Gastric polyps can manifest in each of the gastrointestinal polyposis syndromes, but also in Lynch syndrome and in a few rare not primarily gastrointestinal conditions. While some of these syndromes are clearly associated with an increased risk of gastric cancer, others are not. Recognition of syndromic gastric polyps is important for individual patient management.

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Management of gastric polyps: a pathology-based guide for gastroenterologists. Carmack SW, Genta RM, Graham DY, Lauwers GY. Nat Rev Gastroenterol Hepatol 2009;6:331-41. Pathology and Genetics of Syndromic Gastric Polyps. Brosens LA, Wood LD, Offerhaus GJ, Arnold CA, Lam-Himlin D, Giardiello FM, Montgomery EA. Int J Surg Pathol. 2016 May;24(3):185-99 The current spectrum of gastric polyps: a 1-year national study of over 120,000 patients. Carmack SW, Genta RM, Schuler CM, Saboorian MH. Am J Gastroenterol 2009;104:1524-32. Sporadic Fundic Gland Polyps With Low-Grade Dysplasia: A Large Case Series Evaluating Pathologic and Immunohistochemical Findings and Clinical Behavior. Levy MD, Bhattacharya B. Am J Clin Pathol 2015;144:592-600. Gastric lesions in patients with autoimmune metaplastic atrophic gastritis (AMAG) in a tertiary care setting. Park JY, Cornish TC, Lam-Himlin D, Shi C, Montgomery E. Am J Surg Pathol 2010;34:1591-8. Hyperplastic polyps of the stomach: associations with histologic patterns of gastritis and gastric atrophy. Abraham SC, Singh VK, Yardley JH, Wu TT. Am J Surg Pathol 2001;25:500-7. Gastric adenomas: intestinal-type and gastric-type adenomas differ in the risk of adenocarcinoma and presence of background mucosal pathology. Abraham SC, Montgomery EA, Singh VK, Yardley JH, Wu TT. Am J Surg Pathol 2002;26:1276-85. Some observations on pyloric gland adenoma: an uncommon and long ignored entity! Vieth M, Montgomery EA. J Clin Pathol 2014;67:883-90. Histological and molecular classification of gastrointestinal polyps. Haumaier F, Sterlacci W, Vieth M. Best Pract Res Clin Gastroenterol. 2017 Aug;31(4):369-379. Evaluation and management of gastric epithelial polyps. Castro R, Pimentel-Nunes P, Dinis-Ribeiro M. Best Pract Res Clin Gastroenterol. 2017 Aug;31(4):381-387.

Disclosure:
Nothing to disclose

Clinical presentation and endoscopic assessment of gastric lesions

Christian Gerges, Germany

Learning Objectives:

- Types of Gastric Lesions
- Endoscopic Assessment
- Classification & Management

Abstract:

Definition: Sessile or pedunculated lesions that originate in the epithelium or submucosa and protrude into the lumen. The most common and important polyps dicussed today are fundic gland polyps, hyperplastic polyps, adenomatous polyps and gastric cancer. Evaluation of gastric polyps should always follow a standardized concept evaluating four main important aspects: Size,Surface, Location and Number. Biopsy at first EGD is always recommended to exclude dysplasia, AC and FAP. Usually no polypectomy for sporadic FGP is required. Young patients with multiple FGP without using PPI or with dysplasia in biopsy should undergo colonoscopy to exclude FAP. Hyperplastic Polyps are second most common gastric polyp (30-93% of all benign GP) and associated with HP and inflammatory disorders in general. The neoplastic potential is controversially discussed; but there is an increased risk of synchronous cancer elsewhere in gastric mucosa.

Therefore intensive inspection of the whole stomach using WLI and NBI is necessary. HP eradication should be performed if present. Single large lesion >0,5cm should be removed. A min of 6 Biopsies from surrounding mucosa should be taken. Adenomas represent 3-26% of all gastric polyps and are strongly associated with atrophic gastritis and intestinal metaplasia. They are true neoplasms and precursors of gastric cancer and associated with synchronous and metachronous gastric adenocarcinoma. They should always be removed en-bloc. In case of gastric cancer Intensive endoscopic assesment is mandatory including the use of Paris classification for risk evaluation of submucosal invasion and lymphnode infiltration.

Take your time cleaning and looking using WLI and NBI if possible, polyps should be biopsied at first egd unless Endoscopic Resection is indicated anyway. Precise Classification for suspicious lesions is crucial. Endoscopic resection of early gastric cancer should be performed en-bloc in tertiary referral centers.

References:

1. Andrew F Goddard, Rawya Badreldin, D Mark Pritchard, Marjorie M Walker, Bryan Warren, on behalf of the British Society of Gastroenterology The management of gastric polyps GUT 2010;59:1270-6 2. Castro R, Pimentel-Nunes P, Dinis-Rebeiro M, Best Practice & Research Clinical Gastroenterology 2017 3. YASSER H. SHAIB, MASSIMO RUGGE,‡ DAVID Y. GRAHAM,* and ROBERT M. GENTA; Management of Gastric Polyps: An Endoscopy-Based Approach PERSPECTIVES IN CLINICAL GASTROENTEROLOGY AND HEPATOLOGY 2013;11:1374-1384 4. Endoscopic classification review group. Endoscopy 2005;37:570-8 5. Zheng Z, BMC Cancer 2016, 16:92 6. Rugge M, Hum Pathol 2005;36:228-233 7. Capelle LG, GIE 2010;71:1150-1158 8. Price AB JGH 1991;6:209-22 9. Weston BR, J Clin Gastroenterol 2003; 36:399-402*

Disclosure:

MTE Consultant

Pathology perspective: What are subepithelial lesions?

Michael Vieth, Germany

Learning Objectives:

- Learning about the histo-anatomical layers of the upper GI-tract
- Identifying the tumor by its characteristics according to location
- Identifying tumors by biopsy and histology

Abstract:

Fortunately, most lesions are benign and can be biopsied first. A prerequisite is that gastroenterologists and pathologists know and understand what they are dealing about. First, the knowledge of the histo-anatomical layers of the wall (eg. Stomach) is necessary.

Esophagus: Melanomas are to be expected within the esophageal epithelium. Leiomyoma and Granular cell tumors are located within the muscularis mucosa whereas GISTS are to be found within the muscularis propria

Stomach: NET are found in the mucosa and submucosa (new WHO : highest grade of KI67 or count of mitosis should be used for grading!), whereas Granular cell tumors are to be found originating from the muscularis mucosae. An Inflammatory fibroid polyp (Vanek, mostly stomach, adults, only) is located in the submucosa, whereas GIST (often stomach, various locations have been described, metastases up to 30% of the cases), Glomus tumors (rarey in the GI tract, mostly benign), Schwannoma (rarely esophagus or colon, “lymphoid cuffs” as typical feature in the stomach), and plexiform fibromyxoma (stomach, only, fusiongene MALAT1-GLI1 like in Gastroblastoma (malignant!) are to be found in the muscularis propria.

Small bowel: Gangliocytic paraganglioma and inflammatory fibroid polyps are to be found in the submucosal layer, whereas GIST, Ganglioneuromas and clear cell sarcoma-like tumors are to be expected within the muscularis propria. Within the mesenterium: sclerosing mesenteritis, inflammatory-myofibroblastic tumor (subsequently described outside the lungs and regarded as spectrum of lesions called “plasma cell granulomas”, target for specific therapy e.g. ceritinib), mesenteric fibromatosis (may be component of Gardner Syndrome, can be associated with APC gene mutations) can be found.

In conclusion the diagnosis depends on the layer of the wall. Immunochistochemistry or Molecular Pathology may lead to confusing results if not correlated to the exact site within the wall and the morphology of the lesion.

References:

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Disclosure:

Nothing to disclose

Gastric subepithelial lesions: Minimal-invasive surgery as standard treatment!

Not available

Gastric subepithelial lesions: Endoscopic resection always first-line!

Not available

How to use drugs in IBD

11:00-13:00 / C3

Treatment aims of therapeutic drug monitoring

Shomron Ben-Horin, Israel

Learning Objectives:

- Understand factors influencing drug levels
- Learn how to use 'reactive TDM' to better manage patients with loss of response
- Learn the place of 'proactive TDM' to control disease activity and prevent relapses

Abstract:

Drug levels of biologics are influenced by distribution and clearance. Clearance is impscted by many factors, including immunogenicity (formation of anti-drug antibodies), the degree inflammation ('inflammatory sink'), drug loss in feces, reduction of clearance by FcRn binding and is also correlated with weight and albumin levels. For most biologics, an association of drug levels with clinical outcomes and with suppression of inflammation has been demonstrated, and low drug levels are generally predictive of worse outcome later on. However, the direction of the association has proven more difficult to prove, has more inflammation - for whatever reason - can in itself reduce drug levels.

For anti-TNF agents, TDM has proven useful in guiding management of loss of response. In this 'reactive TDM' strategy, low drug levels with absent or low-titer anti-drug antibodies (ADA) indicate that dose-increase is the optimal management, after non-adherence has been ruled out. Low drug with high-titer ADA indicate the need to switch to another drug of the same class, whereas an adequate drug level usually indicates the need to swap to a drug from another class (with different mechanism of action) but also indicate the need to carefully verify the absence of other causes for clinical symptoms. The TDM approach has not yet been shown beneficial for the newer biologics, i.e. ustekinumab and vedolizumab.

Recent trials sought to prove that a proactive approach, whereby drug doses increases in asymptomatic patients with low drug levels may improve their later outcomes. The results of two seminal trials were negative, although in one of these trials a proactive approach reduced the number of clinical flares during follow-up. In the recent pediatric PAILOT trial, a proactive dose-adjustment of adalimumab based on TDM and inflammatory markers was superior to conventional therapy for improving long-term outcomes of CD patients. More trials on 'proactive TDM' are eagerly awaited.

References:

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Disclosure:

Janssen, Takeda, Celltrion, Abbvie, Pfizer: Consultancy, speaker fees, & research support GSK, Ferring: Consultancy/speaker fees.

Positioning of drugs in ulcerative colitis

Iris Dotan, Israel

Learning Objectives:

- Therapy in ulcerative colitis should be chosen according to several factors, including treatment target, disease severity and extent, patient factors and global considerations
- For induction of remission in patients with moderate to severe UC, steroids, anti-TNF, anti-integrins, JAK inhibitors, calcineurin inhibitors and anti-IL-23 agents are effective
- Emerging head-to head studies and comparative analyses may provide guidance in therapeutic decisions

Abstract:

Ulcerative colitis (UC) is a chronic inflammatory disease of the colonic mucosa. Moderate to severely active UC is traditionally treated with mesalamine, steroids and immunomodulators for maintenance. In recent years the efficacy of multiple agents for treating moderately to severely active UC was demonstrated. Those include anti-TNF, anti-integrins, JAK inhibitors, calcineurin inhibitors and anti-IL-23 agents. Effective therapy is meaningful in achieving advanced treatment goals and preventing short and long term complications. These benefits should be weighed against side effects that may be associated with these potent agents. A number of factors have to be considered to choose the optimal therapy for a patient with moderately to severely active UC. Those include target of treatment, specifically induction of remission or its maintenance, patient factors such as age, disease severity and extent, childbearing/pregnancy, BMI, extraintestinal manifestations, comorbidities and risk factors (i.e. cardiac failure, demyelinating disease, risk for infection, malignancies, thrombotic risk).Global factors such as patient preference, route of administration, local availability and cost effectiveness should be taken into account as well. Currently no biomarker may direct specific treatment choice over the other. In recent years, head to head comparisons, and comparative effectiveness and network meta analyses reports, provide further information that may be used in therapeutic decision making.

References:

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Disclosure:
Pfizer, Takeda, Abbvie, Janssen, Genentech/Roche, Celltrion, Celgene, Nestle, Ferring, Rafa Laboratories, Arena, Neopharm, Gilead, Sublimity, Given Imaging/Medtronic, Falk Pharma, Altman Research

Positioning of drugs in Crohn's disease

Matthieu Allez, France

Learning Objectives:
- To review the different therapeutic options in the management of CD according to disease severity and extent
- To review the factors which may influence the choice between the different options
- To compare the efficacy and safety of biologics which are available for CD

Abstract:
Crohn's disease (CD) is a heterogeneous disease associated with a chronic inflammatory process of segments of the digestive tract, which can progressively induce destructive damages. Therapies including immunomodulators and biologics are used to induce and maintain remission, restore quality of life and to avoid long-term bowel damage. The presence of active inflammation due to CD should be confirmed before initiating or changing medical therapy. Options are classically discussed according to disease severity and extent. Budesonide or systemic steroids can be used for induction of remission in patients with mild and non-extended luminal disease, with escalade to immunomodulators and/or biologics if remission has not been achieved. Surgery is also an option in patients with resistance to therapies and local complications. Biologics may be used early in the course of the disease in patients with severe disease or features suggesting a poor prognosis. Those include anti-TNF, ustekinumab and vedolizumab, with new drugs and targets being in development. The choice between the different options is based on a different parameters including disease profile and phenotype, presence of extra-intestinal manifestations and co-morbidities, benefit/risk expected ratio, patient preferences and projects, smoking discontinuation or not, local constraints including drug availability and rules of usage. Ideally, choice of first- and second-line biologics should be personalized and driven by likelihood of response based on biomarkers. Head-to-head clinical trials will provide rationale data for therapeutic decisions. Real World Data and network meta-analysis can provide evidence on the comparative effectiveness of biologics.

References:
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Disclosure:
Pfizer, Janssen, Celgene, Genentech, Roche, Takeda, Abbvie, MSD, Ferring, Falk, Mayoli, Tillots, Innate Pharma, Biogen, Amgen

Pre- and postsurgical management in Crohn's disease

Joline de Groof, Netherlands

Learning Objectives:
- Preoperative management and timing of surgery
- Perioperative drugs and risk of postoperative complications
- Enhanced recovery and postoperative management

Abstract:
Crohn's disease can affect any part of the gastrointestinal tract, however the terminal ileum is most commonly affected. Patients are nowadays treated according to the step-up approach, starting with prednisolone for induction after which maintenance treatment will be introduced with an immunomodulator. If necessary treatment can be escalated to anti-TNF. Surgery is still generally considered a last resort option when all medical options have failed. However a recent RCT showed comparable quality of life in patients with terminal ileitis following anti-TNF and ileocecal resection. The potential benefit of any medication should be weighed against the additional surgical risk should this medication fail to achieve symptomatic relief. This is also the case in patients with acute severe Crohn's colitis. These patients should be under daily surveillance by a multidisciplinary team. Any clinical deterioration or failure to improve within approximately 1 week despite optimal medical treatment should prompt consideration for emergency surgery. Malnutrition is a significant risk factor for postoperative complications, therefore nutritional status should be optimised prior to surgery. Also preoperative medical therapy (e.g. prednisolone and anti-TNF) is a significant risk factor for postoperative complications.
Unfortunately it is unknown up till now what the safest period of discontinuation before surgery is in order to reduce the risk of postoperative complications. Regarding postoperative management patients will be treated following the principles of fast track rehabilitation. Numerous studies have demonstrated the advantages of enhanced recovery protocols, with a shorter hospital stay and lower overall complication rates.
Prophylactic treatment with thiopurines or anti-TNF following surgery is recommended in patients with at least one risk factor for recurrence e.g smoking, previous intestinal surgery, penetrating disease at index surgery or perianal disease.

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Disclosure:
Nothing to disclose

Adjustment and optimisation of medical treatment over the disease course

James Lindsay, United Kingdom

Learning Objectives:

- Understand the natural history of disease and how this impacts drug therapy
- Appreciate the management of loss of response to therapy
- Appreciate that the risks of therapy can vary over the course of disease

Abstract:

The chronic intestinal inflammation of both Crohn´s disease and ulcerative colitis may result in disease progression and complications if undertreated. Therefore the goals of therapy are to induce and then maintain both clinical and mucosal disease remission throughout the whole disease course. This has translated into the concept of "treat to target" therapeutic approaches with the end goals being restoration of a normal quality of life and prevention of disease progression and complications. However, the ideal target to aim for and the most appropriate strategy to achieve this target are not certain. Despite this, it is clear that regular monitoring of disease activity using both symptom based an objective assessments couple with optimisation of medical therapy reduces disease complications. It is important to understand that the requirement of both conventional and biological / small molecule therapies may vary during the course of a patient´s disease. In addition, The risk benefit profile of the drugs that are used will also vary, such that optimisation considering safety as well as efficacy is important. These treatment decisions often require involvement of the full multidisciplinary team and must involve input from patients.

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Chronic pancreatitis

11:00-13:00 / F3

Genetics in chronic pancreatitis: Pathophysiology and clinical implications

Julia Mayerle, Germany

Learning Objectives:

- To understand the necessity of genetic testing in chronic pancreatitis
- To recognize the increased risk of pancreatic cancer in patients with hereditary pancreatitis in contrast to patients with idiopathic chronic pancreatitis
- Management and surveillance of hereditary and idiopathic chronic pancreatitis

Abstract:

Chronic pancreatitis is a disease of the pancreas in which recurrent inflammatory episodes result in replacement of pancreatic parenchyma by fibrous connective tissue. This fibrotic reorganisation of the pancreas leads to a progressive exocrine and endocrine pancreatic insufficiency. In addition, characteristic complications arise, such as pseudocysts, pancreatic duct obstructions, duodenal obstruction, obstruction of the bile ducts, malnutrition and pain syndrome. In 2015 the incidence was given with 22/100,000 population and the prevalence is 10 times higher. Non-alcohol and non-tobacco associated chronic pancreatitis account for 20 to 50% of cases. Hereditary chronic pancreatitis is a rare cause of chronic pancreatitis, most commonly due to mutations of PRSS1, encoding for the trypsin-1 (also known as cationic trypsinogen). The prevalence of hereditary chronic pancreatitis is around 0.3 per 100,000 individuals in Western countries.

The inheritance pattern is autosomal dominant with an incomplete penetrance (up to 80%).

Numerous studies have shown an association between mutations in SPINK1 and the risk of developing recurrent acute and chronic pancreatitis, although the mechanisms remain

unknown. SPINK1 encodes for a trypsin inhibitor secreted by acinar cells. Variants of CTRC, encoding chymotrypsin-C (involved in the degradation of prematurely activated trypsin-1), were associated with a significantly increased risk of chronic pancreatitis. Mutations in the genes coding for calcium-sensing receptor and carboxypeptidase A1 were also associated with a small increase in risk of developing chronic pancreatitis. Inactivating mutations in CFTR (encoding cystic fibrosis transmembrane conductance regulator), which also cause cystic fibrosis, are associated with chronic pancreatitis. Finally, variants in noncoding regions of the PRSS1, PRSS2, CTRB1 and 2, and CLDN2 loci affect the risk of sporadic and alcoholic pancreatitis.

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Disclosure:

Nothing to disclose

Long-term management of patients with chronic pancreatitis: What to expect, and how to monitor and treat

Marcel Vasile Tantau, Romania

Learning Objectives:

- Pre-treatment planning and prognostic assessment
- Endoscopic therapy - when and how?
- Post-therapeutic follow-up - defining expectations

Abstract:

Chronic pancreatitis (CP) is defined by the replacement of normal pancreatic parenchyma by fibrous connective tissue as a result of recurrent aggressions.

What to expect in CP?

The first step after diagnosing CP by EUS, MRI or CT should be to find the etiology, as disease courses vary greatly. Alcoholic patients tend to develop calcifications, exocrine and endocrine insufficiency. The second step should be to assess the staging of the disease and to screen for complications. CP treatment requires lifestyle modifications, pain management, endoscopic and/or ESWL treatment and, in special circumstances surgery.

Favorable long-term outcome is expected in patients with a short disease duration, no main pancreatic duct obstruction, cessation of smoking and alcohol consumption, complete removal of obstructive pancreatic stones and strictures resolved with stenting.

How to monitor and treat?

Asymptomatic and uncomplicated patients do no need for endoscopic therapy. Treated patients should be followed-up at 6 to 8 weeks to assess clinical response.

Radiopaque stones >5mm in diameter should be initially treated using ESWL Smaller or radiolucent stones should be initially treated by ERCP.

Electrohydraulic or laser lithotripsy under direct pancreatoscopic control of pancreatic stones is an temptimg option under evaluation.

Single 10-Fr plastic stent for one uninterrupted year or multiple side-by-side plastic stents can be placed to treat symptomatic pan creatic strictures. Fully covered self-expendable metal stents appears safe and effective. Endosonographic-guided celiac plexus blocks are preferred over percutaneous blocks.

Endoscopic treatment is considered the first line of treatment for cysts that are within endoscopic reach. Biliary strictures can be treated by placing multiple side-by-side plastic stents or a fully covered stents. Approximately four ERCPs over a 12 month period are usually required for complete therapy of common bile duct strictures.

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Disclosure:

Nothing to disclose

Surgery first: Skip non-effective endoscopic interventions

Marja A. Boermeester, Netherlands

Learning Objectives:

- Surgery is superior to endoscopy in terms of mid-term and long-term pain relief in patients with painful CP
- Surgery early in the disease process of CP is favored over surgery in a more advanced stage of disease to achieve optimal long-term pain relief. Also, long-term QoL is improved after early surgery (<3 years from onset) compared to surgery at a more advanced stage of disease
- In patients with (a) uncomplicated painful CP in (b) the early stage of disease and (c) a dilated main pancreatic duct with distal obstruction, endoscopic treatment may be used as first-line treatment after failed medical therapy if clinical response is evaluated at 6-8 weeks and under the condition that failure of or repeated need for endoscopic therapy triggers referral for surgery

Abstract:

In the recent international evidence based (HaPanEU) guidelines, a multidisciplinary step-up approach to treatment of pain in chronic pancreatitis was recommended [1], although specific trials incorporating this approach have yet to be performed. The first step is conservative therapy, including life style management (e.g. cessation of alcohol use and smoking), dietary advice, and pain medication. The approach to pain management of the World Health Organization is widely accepted for pain treatment in CP, although it has never been formally evaluated. If patients have persistent pain despite appropriate conservative measures including optimization of pain medication, subsequent interventional endoscopy or surgery is recommended.

Surgery is reported to have a good long-term effect although approximately 10% of patients will not respond and this risk is larger in patients with a high number of endoscopic procedures prior to surgery [2,3]. Two RCTs have compared endoscopic and surgical management in patients with painful obstructive CP [4,5]. Both studies showed superiority of surgical over endoscopic management.

In long term follow-up these results remained stable after more than 6 years, with still 80% pain relief in the surgery group versus 38% pain in the endoscopy group [6]. Complete pain relief is seen in 53% of patients after surgery versus 25% after endoscopy [6]. It is also noted that 47% (9/16) of the endoscopic treated patients required delayed surgery, and complete pain relief was found in only 2/9 (22%) patients with delayed surgery compared with the 8/15 (53%) who had primary surgery.

The recent ESCAPE trial has shown that early surgery for patients with symptomatic chronic pancreatitis and a dilated pancreatic duct who have recently started using opioids provides better pain relief with less interventions and at lower costs than the current step-up practice including endoscopy, with comparable pancreatic function and quality of life.

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Disclosure:
Nothing to disclose

Endoscopy first: Surgery only as a last resort

Jacques Deviere, Belgium

Learning Objectives:
- Discuss the place of interventional therapy in CP pain management
- Understand clinical indications
- Understand morphological indications

Abstract:
Chronic pancreatitis: minimal intervention first.
Pain associated with chronic pancreatitis may be due to a main pancreatic duct (MPD) obstruction induced by stones and/or strictures, often located in the head of the pancreas. Extracorporeal shockwave lithotripsy (ESWL) and endotherapy (ET) are first line interventional management in this clinical setting.
After a pre-therapeutic planning which usually includes a magnetic resonance cholangiopancreatography (MRCP) and a computed tomography (CT) scan without contrast injection, ESWL can be used alone without endoscopic management in order to fragment the stones. These stones, made of calcium carbonate, can be destroyed in millimetric fragments using high power, fluoroscopy guided lithotripsy.
In case of associated symptomatic strictures of the MPD, an endoscopic sphincterotomy of the pancreas has to be performed associated with dilation and placement of stents bypassing the stricture.
In order to obtain a long term calibration of such strictures, stents have to be left in place for a minimum of one year (with regular exchange) and more recently, multiple plastics stents have been proposed in order to gain a better calibration. These plastic stents (7 or 8.5 French) are usually inserted side by side. There is currently no indication for routine placement of fully covered self-expandable metal stent into the pancreas. Such a policy allows to relieve pain on the long term after stent removal in approximately two thirds of the patients. If a clinically symptomatic stricture recurs after stent removal, these patients, managed into a multidisciplinary environment, may be offered surgery. Measurement of outcomes is important as most pain relapses after endotherapy occur within one year after initial treatment, a feature which contrasts with pour relapsing after surgery which usually occurs after a median of six to seven years.

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Nothing to disclose

Optimal medical pain management: When endoscopy and surgery are not an option

Asbjørn M. Drewes, Denmark

Learning Objectives:
- An understanding of pain mechanisms to guide medical management of chronic pancreatitis
- Recognize the many pharmacological options to treat chronic pancreatitis
- Learn how to use pharmacological treatment in the most rational way

Abstract:
Pharmacological treatment remains a challenge as it is poorly documented and mainly based on empirical knowledge from somatic pain conditions. This may be problematic, as many aspects of the neurobiology differ significantly from somatic pain. On the other hand, the variability in phenotypic presentation of different pain syndromes is found to be greater between patients than between different pain syndromes and this supports current practice. The novel and improved understanding of pain etiology in chronic pancreatitis, proposing that visceral neuropathic pain in present in a large part of patients requires a paradigm shift in pain management. Abstinence from alcohol and smoking should be strongly advised and pancreatic enzyme therapy and antioxidants may be helpful as initial treatment.
Current guidelines for pharmacological management recommends a simple stepwise escalation of analgesic drugs with increasing potency until pain relief is obtained. In the first step, non-opioid drugs such as paracetamol or non-steroidal anti-inflammatory drugs (NSAIDs) should be used. If these drugs are insufficient, weak opioids such as codeine or tramadol are added at the second step. In the third step, weak opioids are replaced by strong opioids. At each stage of analgesic ladder, it is possible to give adjuvant drugs such as tricyclic antidepressants or anticonvulsants. Side effects are simultaneously monitored and handled. Of note analgesic treatment can seldom stand alone, and in modern pain treatment the ladder is only used a guide to oral medications where treatment includes e.g., invasive management, supportive care and nursing (multimodal analgesia). Finally, it should not be forgotten that the individual experiences and manifestations of pain are influenced by a complex series of interactions involving sensory, pathophysiological, affective, socio-cultural, behavioural and cognitive elements that may also **need management.**

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Monitoring of disease activity in IBD

14:00-16:30 / A2

Which are the evidence to promote disease monitoring in Crohn's disease and in ulcerative colitis?

Laurent Peyrin-Biroulet, France

Learning Objectives:

- What is the role of biomarkers for monitoring UC and CD patients?
- Should we include histological healing in the disease monitoring strategies of UC?
- Should we include transmural healing in the disease monitoring of CD?

Abstract:

The main objectives in inflammatory bowel disease (IBD) are to avoid disease complications and preserve the patient's quality of life. Historically the primary objective of treatment in therapeutic trials and clinical practice in CD was to induce and maintain symptomatic remission. This approach failed to clearly modified the natural course of CD. With the Selecting Therapeutic Targets in Inflammatory Bowel Disease (STRIDE) consensus, treatment goals in CD have moved to "deep remission" which is defined by reaching both symptomatic and endoscopic remission (defined as no ulceration at ileocolonoscopy). Biomarkers were not targets in STRIDE but only adjunctive measures of inflammation. The CALM trial allowed a prospective validation of tight control strategies based on careful and continuous surveillance of the disease activity by biomarkers (mostly fecal calprotectin), and early therapeutic optimization or change of treatment if necessary. Two controlled trials also showed that 5-ASA optimization based on measurement of fecal calprotectin leads to better outcomes in ulcerative colitis (UC). Over the mast decade, a fecal calprotectin level less than 250 was considered as therapeutic success. With more ambitious therapeutic goals (transmural healing in CD and histological healing in UC), normalization of fecal calprotectin levels could be recommended in the near future. In summary, early disease control and close monitoring might be the best way to change the disease course. In this context, the role of prognostic factors and first-line treatment appears less significant.

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Disclosure:

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How to increase patients' acceptance and participation in disease monitoring: The "classical" way

Johan Burisch, Denmark

Learning Objectives:

- To understand the differences in the perception about treatment goals between patients and physicians
- To learn that shared decision-making is associated with better adherence and patient satisfaction
- To be introduced to decision aids and patient-reported outcome measures, that can facilitate patient interaction

Abstract:

Inflammatory bowel diseases (IBD) are complex and it is difficult to predict which patients will respond to which therapies or how aggressive the disease will be. Multiple treatment options exist with no head-to-head trials for comparative effectiveness data, and therefore patients and physicians often find themselves in clinical decision-making situations with no clear correct solutions. Furthermore, the perception of disease severity differs between patients and physicians. Improving patients understand of and adherence to treatment and monitoring plans involves shared decision-making, which involves explaining different clinical options and taking explicit steps to elicit patients' values and preferences. This approach has been shown to increase patient satisfaction and adherence in patients with chronic diseases such as IBD. Furthermore, shared decision-making increases patient understanding of disease management plans, and such patients are subsequently more likely to be more accepting of it and willing to share in and follow their treatment and monitoring schedules. Measures to facilitate shared decision-making include well-developed, comprehensive written materials (decision aids) or questionnaires capturing patient-reported outcome measures such as disability. Challenges to this approach include time pressure and lack of manpower in daily clinical practice.

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How to increase patients' acceptance and participation in disease monitoring: The technical, web-based approach

Not available

Disease monitoring with biomarkers and endoscopy: How to best combine them?

Peter Bossuyt, Belgium

Learning Objectives:

- Learn how to select the optimal tool for disease monitoring before the start of treatment
- Learn to assess the disease activity with the correct interval
- Learn how to proactively monitor and adjust the treatment

Abstract:

Clinical symptoms are suboptimal for the monitoring of IBD. Underlying subclinical inflammation is associated with progression to complicated disease in Crohn's disease (CD) as in ulcerative colitis (UC) and leads to worse outcome over time. For this, strict monitoring of the disease activity can impact on the long term outcome of the disease. The advantage of biomarkers compared to endoscopy is the ubiquitous availability, low

invasiveness and low cost. On the other hand more data are supporting the role of endoscopy and particularly the presence or absence of endoscopic remission as predictor of further disease course in a treat to target strategy, although most studies are retrospective or observational. Before starting treatment it is important to have a baseline assessment of the disease activity. This includes biomarkers (i.e. C-reactive protein CRP, faecal calprotectin (FC)) and endoscopy. Based on this baseline "staging", the ideal monitoring tool can be selected. One should absolutely avoid to monitor biomarkers that are not elevated at baseline. In UC it is suggested to have an early endoscopy evaluation 8-12 weeks after treatment initiation. The presence of endoscopic remission at that stage is predictive for sustained clinical remission over time. If endoscopic remission is achieved then further follow-up can be done based on biomarkers. Serial FC measurements in patients with UC in remission are helpful in predicting clinical relapse, since FC progressively raises 2-3 months before clinical relapse. In CD monitoring of biomarkers with subsequent treatment optimization (if persistent inflammation is present), leads to better long term outcomes. Endoscopy should be performed after 6-12 months to confirm endoscopic remission. In the specific situation of an ileocaecal resection in CD, endoscopy after 6-12 months is the gold standard. FC measurement after 3 months can select patients that warrant an earlier endoscopic evaluation.

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Disease monitoring with gastroenterologist ultrasound: The point-of-care procedure!

Gerhard Rogler, Switzerland

Learning Objectives:

- Ultrasound is useful in the monitoring of IBD patients to evaluate whether symptoms are inflammation associated and for postoperative relapses
- There are objective parameters to be measured by bowel ultrasound
- Ultrasound is useful for the evaluation of therapeutic success in IBD therapy

Abstract:

Gastroenterologist ultrasound is established for the monitoring of therapy success in patients with UC and CD in many countries. Ultrasound has several advantages over other imaging techniques:
It allows transmural evaluation of the bowel wall (in contrast to endoscopy and similar to CT/MRI)
It provides a rapid information on the origin of symptoms or therapeutic success without delay or preparation (in contrast to endoscopy, MRI or CT scan)
It is not associated with X-ray exposure (in contrast to CT scans, similar to MRI)
It allows the evaluation of motility (live on-site in contrast to endoscopy, CT scan and MRI, which is Important to evaluate functional impairment)
It allows demonstrating findings to the patient "live", which frequently improves patient adherence to therapy.
There are objective parameters in bowel ultrasound that can easily be measured such as bowel wall thickness. Their assessment should be requested as a quality measure.
Ultrasound, CT, and MRI have a similar high diagnostic accuracy at the initial presentation of terminal ileal CD [1]. The relevance of measuring bowel wall thickness has been demonstrated by a number of studies and sensitivities of 75-94% with specificities of 67-100% have been reported [1]. A recent meta-analysisindicated that a threshold of > 3 mm bowel wall thickness as cut-off value has a sensitivity and specificity 88 and 93% respectively for acute inflammation in CD, while a cut-off level of >4 mm is associated with a sensitivity and specificity of 75% and 97% respectively [2].
Imaging for the monitoring of therapeutic success and disease control (or "mucosal healing") is an essential component of IBD patient care. The schedule of monitoring should depend on the disease course (mild versus severe) and the treatment used. Ultrasound may substitute for endoscopy in many instances for the monitoring of IBD patients.

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Disclosure:
Nothing to disclose

Disease monitoring with medical imaging: MRI - the golden standard!

Jordi Rimola, Spain

Learning Objectives:
- To review the strengths and limitations of MRE in monitorization of Crohn's disease
- To discuss the potential treatment endpoints measured by MRE
- To show how MRE may guide treatment optimization in clinical practice and in research

Abstract:
In the era of biological drugs, the most common target to treat is healing of severe inflammatory lesions in Crohn's disease (CD), as its achievement has been associated with better outcomes 1. Therefore, accurate and reliable tools to monitor inflammatory lesions of the intestine and potential CD's related complications are essential for guiding therapeutic decisions2. For assessment of efficacy outcomes after therapeutic intervention, MRE ensures an objective assessment of all intestinal segments by providing a full map of lesions3. The existence of fully validated MRE scoring systems for the small and large bowel4-7 allows the implementation of MRE in clinical research offering the possibility to establish well defined endpoints and avoiding operator dependent variability8.
On the other hand, the progressive nature of CD over time is well established. To advance in the assessment of therapeutic interventions on damage progression it is necessary to separate the long-term effects from the short-term effects on symptoms that these treatments may afford. With the purpose of measuring digestive tract damage and not severe inflammatory lesions in CD, and facilitate the assessment of disease progression over time, an international initiative recently developed the Lémann index9 that could be measured by MR. This index integrates stricturing and penetrating lesions in all segments of the intestine together with previous resected intestinal segments using MRI facilitating the objective measurement on long-term damage.

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* Reviews lectures for the non-expert.*

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Complications of severe acute pancreatitis

14:00-16:30 / C3

Initial medical management of severe acute pancreatitis: Hydration, nutrition, antibiotics

Enrique de-Madaria, Spain

Learning Objectives:

- To know current evidence for and against aggressive fluid resuscitation and the use of lactated Ringer solution as fluid of choice
- To learn when and how patients with acute pancreatitis may benefit nutritional support
- To learn when antibiotics are indicated and which antibiotics

Abstract:

Currently we lack a specific treatment to manage the early phase of acute pancreatitis (AP), so patients rely on supportive care. Aggressive fluid resuscitation is considered by many experts as a cornerstone in the treatment of AP, but the evidence from randomized controlled trials (RCTs) is scarce. Two studies from the same group suggested that aggressive fluid resuscitation in severe AP is associated to worse outcomes including decreased survival (1, 2). One RCT suggested that aggressive resuscitation may fasten clinical recovery and decrease inflammatory response in predicted mild AP (3). Those 3 studies have some design flaws so new quality RCTs are needed. Lactated Ringer solution, when compared to normal saline, seems to have an anti-inflammatory effect, according to 2 RCTs (4, 5), but the effect on important clinical endpoints like organ failure, local complications or survival is unknown. Naso-jejunal and nasogastric tube feeding are linked to similar outcomes in AP according to 3 RCTs. A well-designed RCT did not showed improved outcomes for early enteral nutrition in predicted severe AP when compared to on-demand enteral nutrition (trying oral intake after 3 days of fasting and reserving tube feeding to patients unable to resume oral intake) (6), so enteral feeding is mainly recommended in patients with prolonged intolerance to oral intake. Well-designed RCTs investigating the effect of prophylactic antibiotics on outcomes of AP have yielded negative results (7), so currently antibiotics are not used to prevent infection of pancreatic necrosis, but the empiric use of antibiotics when such a complication is suspected should be encouraged (8). Carbapenems, quinolone, metronidazole, and high dose cephalosporins are known to penetrate well in pancreatic tissue (8).

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Disclosure:

Nothing to disclose

Pancreatic collections: How to assess, which and when to treat? The clinician's and the radiologist's view

Marianna Arvanitakis, Belgium
Maria Antonietta Bali, Belgium

Learning Objectives:

- Differentiating between types of pancreatic collections (based on morphological features and time from onset of acute pancreatitis)
- Imaging modalities: knowing which to use and when
- Understanding the indications for intervention

Abstract:

In most cases of acute pancreatitis (AP), the outcome is rapidly favorable. However, acute necrotizing pancreatitis (ANP) may develop in up to 20% of cases and is associated with significant rates of early organ failure (38%), need for intervention (38%), and death (15%).

Pancreatic collections related to AP are best defined in the revised Atlanta criteria and include acute (peri)pancreatic fluid collections(PFCs; within the first 4 w, with no well-defined wall, usually resolving spontaneously); acute necrotic collections(within the first 4 w of onset of ANP, containing variable amounts of fluid and necrotic tissue); pancreatic pseudocysts(≥4 w,fluid collection in the [peri]pancreatic tissues, surrounded by a well-defined wall, containing no solid material); and walled-off necrosis(WON; after ≥4 w of onset of ANP, encapsulated collection containing partially liquefied [peri]pancreatic necrotic tissue).

Contrast-enhanced CT is considered as the first-line imaging modality on admission when indicated and up to the 4th week from onset in the absence of contraindications. The CT severity index is the preferred imaging severity score, which combines assessment of surrounding collections as well as parenchymal necrosis. MRI may be used instead in patients with contraindications to contrast-enhanced CT, and after the 4th week from onset when invasive intervention is considered because the contents (liquid vs. solid) of pancreatic collections are better characterized by MRI and evaluation of pancreatic duct integrity is possible.

Interventions for pancreatic collections include radiological, endoscopic and minimally invasive surgical techniques for drainage and debridement. Intervention should be proposed for certain indications such as clinically suspected or proven infected necrosis, persistent organ failure, organ compression and persistent pain. Delaying interventions beyond 3-4 weeks have better outcome, if the patient's clinical situation allows it.

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Disclosure:

Nothing to disclose

Walled-off necrosis: Step-up management

Geoffroy Vanbiervliet, France

Learning Objectives:

- Be familiar with the indications for the treatment of the walled-off necrosis (WON)
- To know the outcomes of the different techniques available for the treatment of the walled-off necrosis (WON)
- To know what step-up management is for walled-off necrosis (WON) and its current validated algorithm

Abstract:

In 2019, the necrotic evolution after acute pancreatitis is still a severe complication, which is difficult to manage. It is considered that 20% of patients after acute pancreatitis will develop walled-off necrosis (WON) and among them the mortality rate can reach up to 30%, especially after infection. This difficulty of management explains why all international recommendations (from ESGE and AGA) highlight the need for expert center-based management and multidisciplinary approach in order to optimize the treatment of these vulnerable patients. The intervention and drainage and/or debridement of WON is indicated in patients with infected necrosis, as this group presents the highest risk of death. Drainage and/or debridement may be required in patients with sterile WON in case of persistent abdominal pain, nausea, vomiting, and nutritional failure or with associated complications including gastrointestinal luminal obstruction, biliary obstruction, recurrent acute pancreatitis, fistulas, or persistent systemic inflammatory response syndrome (SIRS). The treatment of symptomatic WON has evolved significantly over the past 10 years. The high complication rates and mortality rates after open surgery have made it necessary to develop new less invasive approaches. Several randomized trials have shown that minimally invasive step-up approach, based on endoscopic, percutaneous approaches or VARD surgery, significantly reduces the risk of death compared to open surgery during pancreatic necrosectomy. Recently the endoscopic step-up approach was found not superior to the surgical step-up approach in reducing major complications or death. Nevertheless, the rate of pancreatic fistulas and length of hospital stay were lower in the endoscopy group explained that the endoscopic step-up approach was choose as treatment preference in the last international guidelines.

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Disclosure:

Boston Scientific Corporation Cook Medical Corporation

Beyond collections: Abdominal compartment syndrome and vascular complications

Wolfgang Huber, Germany

Learning Objectives:

- Definitions and aetiology of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS)
- Pathophysiological effects of IAH and ACS
- Diagnosis and treatment of IAH and ACS

Abstract:

The abdomen is considered as a closed anatomic space with only few compliant parts including the abdominal wall and the diaphragm. Any increase in intra-abdominal volume (e.g. ascites, blood, edema) necessarily results in an increase in intra-abdominal pressure (IAP).

Intra-abdominal hypertension (IAH) is defined as a sustained increase in IAH ≥12mmHg. The incidence of IAH in critically ill is about 25%. Even in these patients IAH is frequently unrecognized.

IAH results in increased morbidity and mortality. IAH reduces intra-abdominal perfusion pressure (IAPP) and directly impairs the function of intra-abdominal organs such as kidneys, liver and bowel. Furthermore, IAH results in extra-abdominal organ impairment, in particular in circulatory failure and affection of respiratory function.

Organs within other physiologic compartments (head, chest and extremities) are frequently affected by organ fialure as a consequence of IAH. This phenomen has been termed polycompartment syndrome. Circulatory disfunction in IAH is characterized by decreased venous return and cardiac output as well as in increases in systemic vascular resistance. Since IAH artificially increases central venous pressure, the decreased venous return frequently is obscured unless more sophisticated haemodynaic techniqes are used.

Among several techniques to measure IAP including direct intra-peritoneal and intra-gastric measurement, indirect measurement of the intravesical pressure (Foley catheter) is most frequent and recommended by the WSACS (World Society of Abdominal Compartment Syndrome). The pressure transducer should be zeroed at the iliac crest in the mid-axillary line.

Therapeutic measures to decrease IAP include evacuation of intraluminal contents (naso-gastric and rectal tubes; urinary catheter; prokinetic agents; reduction in oral feeding; enemas), evacuation of intra-intra-abdominal contents (paracentesis, drainage), improvement of abdominal wall compliance (sedation, relaxation, avoidance of constrictive dressings), optimized fluid resuscitation (advanced haemodynamic monitoring; monitoring of micro-circulation and IAPP). In case of an abdominal compartment syndrome (ACS: IAP >20mmHg and new organ dysfunction) surgical abdominal decompression should be considered.

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Disclosure:
Nothing to disclose

Advanced colorectal cancer

14:00-16:30 / F3

Modern imaging to optimise clinical decision-making: The surgeon's view

Pim Burger, Netherlands

Learning Objectives:
- Describe the prognostic value of novel imaging features
- Discuss the role of imaging in treatment planning
- Discuss the role of imaging in "watch-and-wait"

Abstract:
The selection of neo-adjuvant treatment and type of surgery is highly dependent on the initial tumor staging. In the past years MRI has become the leading modality for both local staging of the tumor as well as nodal staging. T-stage and N-stage can be assessed by MRI with high sensitivity and specificity. Other features that are evaluated are: distance to the mesolectal fascia, location of possible involved surgical margins, ExtraMural Venous Invasion (EMVI), and vascular deposits (N1c). Furthermore, MRI plays a major role in the evaluation of tumor after neo-adjuvant treatment, allowing a "watch-and-wait" approach in patient with a clinical complete response.
For the patient to benefit the most from state-of-the-art imaging it is mandatory that patients are discussed in a MDT where discussion of imaging is incorporated in discussing the treatment plan. In this session both the surgeon's point of view as well as the radiologist point of view on modern imaging will be discussed.

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Disclosure:
Nothing to disclose

Modern imaging to optimise clinical decision-making: The radiologist's view

Joost Nederend, Netherlands

Learning Objectives:
- Describe the prognostic value of novel imaging features
- Discuss the role of imaging in treatment planning
- Discuss the role of imaging in "watch-and-wait"

Abstract:
The selection of neo-adjuvant treatment and type of surgery is highly dependent on the initial tumor staging. In the past years MRI has become the leading modality for both local staging of the tumor as well as nodal staging. T-stage and N-stage can be assessed by MRI with high sensitivity and specificity. Other features that are evaluated are: distance to the mesolectal fascia, location of possible involved surgical margins, ExtraMural Venous Invasion (EMVI), and vascular deposits (N1c).
Furthermore, MRI plays a major role in the evaluation of tumor after neo-adjuvant treatment, allowing a "watch-and-wait" approach in patient with a clinical complete response.
For the patient to benefit the most from state-of-the-art imaging it is mandatory that patients are discussed in a MDT where discussion of

imaging is incorporated in discussing the treatment plan. In this session both the surgeon's point of view as well as the radiologist point of view on modern imaging will be discussed.

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Disclosure:
Nothing to disclose

The use of neo-adjuvant treatments to downstage advanced colorectal tumours

Rob Glynne-Jones, United Kingdom

Learning Objectives:
- To understand the potential role of neoadjuvant treatment for down-staging microsatellite stable (MSS) and microsatellite instable MSI colorectal cancer (CRC)
- To recognize the current limitations of clinical staging in CRC
- To gain insight into the immuno-biology and different therapeutic strategies for patients with deficient mismatch repair (d-MMR) and proficient mismatch repair (p-MMR)

Abstract:
Surgery is the mainstay of treatment for colorectal cancer (CRC). Recent data suggests more radical resections produce better long-term outcomes. Postoperative adjuvant chemotherapy has traditionally been offered to patients, who have had a curative resection, based on adverse pathological features in the specimen (mainly TNM stage) to reduce the risk of subsequent metastatic disease. Adjuvant chemotherapy in rectal cancer is more controversial.
Neoadjuvant chemotherapy (NACT) is standard of care in the management of some adenocarcinomas (breast/ oesophago-gastric /pancreatic). This strategy aims to reduce the overall tumour burden and facilitate a curative (R0) surgical resection, or even allow more conservative surgery or a watch-and-wait approach in rectal cancer. The main aim of this strategy aims is to deal with micro-metastatic disease at other sites.
The strategy has been used in colon cancer for tumours invading adjacent organs or involving extensive regional lymph nodes. Severe toxicity can compromise surgery. Besides down-staging, the approach offers insight into mechanisms of disease resistance to specific cytotoxic agents, and might identify predictive biomarkers. Response or failure to respond to a NACT regimen might also define the choice of drugs or even the need for adjuvant chemotherapy. Preliminary results in colon cancer in the context of treatment with an immune checkpoint blockade in patients with dMMR suggest neoadjuvant immunotherapy might provide a survival benefit over adjuvant therapy.
In rectal cancer, oncologists increasingly recommend NACT as a component of multimodality treatment- the 'total neoadjuvant treatment' (TNT) approach, which is administered as induction ie prior to, or consolidation after RT. NACT is also given as an alternative to CRT.
The aims of the strategy and potential risks to NACT are reviewed. The ideal appropriate population for NACT, the optimal regimen, and the duration of treatment, are discussed.

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Disclosure:
Rob Glynne-Jones has received honoraria for lectures and advisory boards in the last 3 years by Merck, Servier, Sanofi-Aventis and Bristol Myers Squibb. He has also in the past received unrestricted grants for research from Roche.

Weighing the pros and cons of colorectal surgery in the frail and elderly

Harm Rutten, Netherlands

Learning Objectives:
- Frailty is not a well defined condition and should always be individualized. Irrespective of age and comorbidity
- Excess postoperative mortality and one-year mortality after surgical treatment is not much higher anymore elderly than in their younger counterparts. Differences between countries are probably the result of better perioperative care rather thab patient factors
- Acute surgery is still associated with high risks and should be avoided or minimalized if possible

Abstract:
Curative treatment for colorectal cancer is accompanied by acceptable morbidity and low mortality rates for all patients regardless of age. However, earlier studies on this topic showed that morbidity in elderly patients is most often higher than in their younger counterparts, and the risk for mortality is increased if a complication occurs. Recent studies show a major decrease in one-month and one-year mortality in the elderly population. For the period 2006-2012, 9.1% patients aged ≥75 years died in the first 90 days compared to 4.6% in the most recent period. After correction of excess mortality (relative survival), one-year survival rates were almost equal for younger and elderly patients, with rates of 95.5% and 94.3%, respectively. Despite the fact that elderly had significantly more comorbidities than younger patients in these periods, showing that age and comorbidities alone are not enough to predict short-term mortality. Even for patients above 80 years of age these findings were confirmed. In a population based study from The Netherlands, including patients surgically treated for CRC between 2009 and 2013, improvement was seen in one-month and one-year mortality for

patients ≥75 years old over the years. The reported mortality rates in elderly ≥75 years were, 2.9-6.2% and 11.7-15.0%, respectively for one-month and one-year mortality. Another recent population-based cohort study across four North European countries also showed somewhat higher 30-day, 90-day and overall one-year mortality rates for octogenarian colon and rectal cancer patients. For colon cancer patients ≥80 years the reported one-month and one-year mortality rates of 5.5-11.4% and 17.1-23.6%, respectively. For the elderly rectal cancer patients these rates were 4.7-7.5% and 13.6-22.1%, respectively. This study also showed that short-term mortality improved over the years, but varied substantially between different countries.

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Disclosure:
Nothing to disclose

Decision making in management of locally recurrent rectal cancer: The surgeon's view

Pim Burger, Netherlands

Learning Objectives:
- What are the minimal requirements for a locally recurrent rectal cancer MDT?
- What is the evidence for neoadjuvant treatment and aggressive surgery?
- Which new developments/trials are coming?

Abstract:
The MDT making decisions in locally recurrent rectal cancer has to deal with a much more heterogeneuous patient group than when its dealing with primary rectal cancer. Previous treatment, such as adjuvant chemotherapy, neoadjuvant(chemo-) radiotherapy and repeated neoadjuvant treatment for the recurrent cancer complicate the interpretation of imaging and decision making.
Furthermore, patients may be in a poor clinical condition and synchronous metastatic disease is common. The consequences of choices made in the MDT are crucial for the chance of cure and to avoid unnecessary harm. What are the arguments for neoadjuvant therapies such as induction chemotherapy and reirradiation of the pelvis? What is the role of intraoperative radiotherapy and how aggressive should surgery be in frail patients with a high chance of systemic relapse?

References:

Total pelvic exenteration for locally advanced and locally recurrent rectal cancer in the elderly. Hagemans JAW, Rothbarth J, Kirkels WJ, Boormans JL, van Meerten E, Nuyttens JJME, Madsen EVE, Verhoef C, Burger JWA. Eur J Surg Oncol. 2018 Oct;44(10):1548-1554. Factors affecting outcomes following pelvic exenteration for locally recurrent rectal cancer. PelvEx Collaborative. Br J Surg. 2018 May;105(6):650-657. Preliminary results of a cohort study of induction chemotherapy-based treatment for locally recurrent rectal cancer. van Zoggel DMGI, Bosman SJ, Kusters M, Nieuwenhuijzen GAP, Cnossen JS, Creemers GJ, van Lijnschoten G, Rutten HJT. Br J Surg. 2018 Mar;105(4):447-452.

Disclosure:
Nothing to disclose

Decision making in management of locally recurrent rectal cancer: The radiologist's view

Joost Nederend, Netherlands

Learning Objectives:
- What are the minimal requirements for a locally recurrent rectal cancer MDT?
- What is the evidence for neoadjuvant treatment and aggressive surgery?
- Which new developments/trials are coming?

Abstract:
The MDT making decisions in locally recurrent rectal cancer has to deal with a much more heterogeneuous patient group than when its dealing with primary rectal cancer. Previous treatment, such as adjuvant chemotherapy, neoadjuvant(chemo-) radiotherapy and repeated neoadjuvant treatment for the recurrent cancer complicate the interpretation of imaging and decision making. Furthermore, patients may be in a poor clinical condition and synchronous metastatic disease is common. The consequences of choices made in the MDT are crucial for the chance of cure and to avoid unnecessary harm. What are the arguments for neoadjuvant therapies such as induction chemotherapy and reirradiation of the pelvis? What is the role of intraoperative radiotherapy and how aggressive should surgery be in frail patients with a high chance of systemic relapse?

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study of induction chemotherapy-based treatment for locally recurrent rectal cancer. van Zoggel DMGI, Bosman SJ, Kusters M, Nieuwenhuijzen GAP, Cnossen JS, Creemers GJ, van Lijnschoten G, Rutten HJT. Br J Surg. 2018 Mar;105(4):447-452.

Disclosure:
Nothing to disclose

Ulcerative colitis: Current management

08:30-10:30 / Hall 6

Treatment targets

Julian Panés, Spain

Learning Objectives:

- Review the components and markers that are indicative of remission in UC
- Describe the methods for establishing a definition for remission in patients with UC
- Understand the differences between the definitions used for remission in clinical practice and clinical trials

Abstract:

The overall target of medical management in patients with ulcerative colitis is to induce and maintain remission with the long-term goals of preventing disability, colectomy, and colorectal cancer. The first and irrevocable target is to obtain clinical remission defined as a resolution of clinical symptoms, including cessation of rectal bleeding and improvement in bowel habits, since persistence of symptoms, even if mild, induces a deterioration in quality of life. Second, endoscopic healing, frequently defined as an endoscopic Mayo score of zero or one, is a relevant target; endoscopic healing has been shown to greatly improve long-term clinical remission, decrease risk of colectomy, and limit corticosteroid use. It has become evident that histologic activity may persist in the presence of endoscopic remission, and persistence of endoscopic activity even after achieving endoscopic healing is associated with higher risk of relapse; histologic activity is also the factor with a strongest association with the risk for colorectal neoplasia in ulcerative colitis. Complete normalization of the colonic mucosa at the molecular level may not be part of the definition of remission, since potent activation of anti-inflammatory pathways have been demonstrated in the presence of histologic remission, and may be required to achieve this outcome. Early therapy using a treat-to-target approach, which implies identification of a predefined target, followed by optimization of therapy and regular monitoring until the goal is achieved, is critical in preventing adverse long-term outcomes. The selection of medications is guided by disease severity and extent. A rapid step-up approach based on ulcerative colitis severity and treatment response while closely monitoring intestinal inflammation is recommended.

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Disclosure:

Julián Panés has received consulting fees from AbbVie, Arena Pharmaceuticals, Boehringer Ingelheim, Celgene, Ferring, Genentech/Roche, GoodGut, GSK, Janssen, MSD, Nestlé, Oppilan, Pfizer Inc, Progenity, Takeda, Theravance, TiGenix and Topivert

Optimal use of conventional treatment modalities

Peter Irving, United Kingdom

Learning Objectives:

- To understand the ongoing role for conventional therapy in ulcerative colitis
- To review the opportunities for optimising 5-ASA, steroids and thiopurines
- To understand how to recognise failure of conventional therapy

Abstract:

Conventional therapy for ulcerative colitis comprises 5-ASA, steroids and thiopurines. 5-ASAs have a well-established role in the induction and maintenance of remission in patients with mild-moderate ulcerative colitis (UC). However, therapy may be delivered not only orally but also rectally, either as suppositories or as enemas, and either alone or in combination. Furthermore, different release characteristics of oral preparations present possibilities for treatment optimisation.

Steroids remain a mainstay of treatment for active UC but have no role in maintenance. Poorly absorbed steroids have a better side effect profile than oral systemic steroids whilst intravenous steroids are more potent and are the initial treatment choice for acute severe UC.

Whilst thiopurines have no role in the induction of remission in UC, they are an effective treatment in steroid-dependent patients. Intolerance is relatively common but can be overcome in the majority of patients and effectiveness can be improved with optimisation using metabolite measurement. Shunting of thiopurines towards methylated metabolites can be corrected by using allopurinol in combination with a reduced dose of thiopurines and is associated with a decrease in side effects and improved response.

Finally, optimal use of conventional therapy includes recognition of treatment failure and the need to move on to other treatments.

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Disclosure:

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Individualising treatment: Biologicals and small molecules

Joana Torres, Portugal

Learning Objectives:

- To review the most important points regarding mechanism of action, efficacy and safety of biologicals and small molecules, that can help individualizing clinical-decision making
- To appropriately identify UC patients that will benefit the most from each therapeutic class, including those at higher risk for complications
- To review the role of biomarkers in predicting/individualizing response to therapy and in therapy monitoring

Abstract:

The therapeutic arsenal in UC is expanding. Besides Anti-TNF, other biologicals (vedolizumab, and recently ustekinumab) and small molecules (JAK inhibitors, S1P receptors modulators) are entering the therapeutic arena, offering promising alternatives. Individualizing therapy implies choosing the best drug for each patient, and maximizing its efficacy during follow-up. Several drug and patient-related factors need to be taken into account, such as efficacy, rapidity of onset, mode of administration,safety, patient comorbidities, age, and preferences. Only head-to-head trial is available comparing VDZ with ADA, showing higher efficacy for the anti-integrin. Indirect comparisons of efficacy data (with all the inherent limitations) indicate that IFX and VDZ may be the most effective drugs to achieve clinical remission in biological-naïve patients, while tofacitinib and VDZ may be the most effective for the Anti-TNF-experienced patients. A higher rate of infections, cancer (melanoma) and lymphoma (especially when used in combination therapy) have been reported with Anti-TNF, warranting special monitoring. The gut-selectivity of anti-integrins makes them attractive options in special populations such as the elderly population, or in those with a past history of cancer. Tofacitinib, a JAK1 inhibitor, has a rapid onset of action, low immunogenicity and is administered orally, making it an attractive option; however a safety alert for pulmonary embolism has been recently issued, potentially limiting its use in some populations.

It is clear that the need for individualizing therapy in ulcerative colitis is greater than ever. No biomarker is able to predict response to therapy before its initiation, although there is active research in this setting. However, therapeutic drug monitoring and individualizing therapy on a treat-to-target approach offer the possibility to improve outcomes and should be implemented in clinical practice.

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Disclosure:

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Optimising surgical outcome: Peri-operative treatment, timing and techniques

Marc Ferrante, Belgium
André D'Hoore, Belgium

Learning Objectives:

- To recognize failure of medical therapy as an indication for surgery
- To promote a staged surgically procedure in case of refractory ulcerative colitis
- To optimize outcome through a multidisciplinary approach

Abstract:

Surgery is a valid and safe treatment modality for patients with ulcerative colitis. The three main indications include:
1) acute severe colitis,
2) dysplasia or cancer, and
3) refractory disease. According to the ECCO guidelines, refractory ulcerative colitis includes both steroid dependent and steroid refractory disease, as well as immunomodulator, biologic, or small molecule refractory disease.

With the expanding armamentarium of treatments for moderate-to-severe ulcerative colitis, the need for (elective) surgery has decreased in the last decade. However, postponing surgery in patients with ongoing disease activity increases the risk of postoperative morbidity and mortality. Therefore, a multidisciplinary approach is mandatory to define individualized treatment goals and decide on appropriate timing and type of surgery.

A restorative proctocolectomy with ileal pouch-anal anastomosis is currently regarded the gold standard for surgery in patients with refractory ulcerative colitis. As an attempt to avoid postoperative complications, a staged procedure is preferred with construction of the pouch only in a second phase (classical three stage or modified two-stage procedure). In a first step a minimally invasive (laparoscopic, single port access) total colectomy with end-ileostomy is performed. Furthermore, preoperative systemic steroids should be tapered as much as possible and the malnourished patient should receive preoperative care.

Despite the revolution in medical treatment for ulcerative colitis, the most important adagio in daily clinical practice has remained the same: Save lives, not colons!

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Disclosure:

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A D'Hoore: Research grant: Johnson & Johnson; Consultancy: Takeda Speakers fee: Takeda, Johnson & Johnson

Neuroendocrine tumours

08:30-10:30 / A2

Mechanisms of carcinogenesis: Classification and diagnosis

Ulrich-Frank Pape, Germany

Learning Objectives:

- Suspicion and diagnosis of a neuroendocrine neoplasia (NEN)
- Definition of the neuroendocrine entity
- Definition of WHO-grading and TNM-staging

Abstract:

Neuroendocrine (NE) tumors (NET) or - more generally termed as appropriately suggested by the WHO - NE neoplasms (NEN) comprise a truly heterogenous group of epithelial neoplasms of the gastroenteropancreatic (GEP) system with common characteristic features. The corner stone histopathological markers are chromogranin A (CgA) and synaptophysin (SYN) - both marker molecules of secretory vesicles for peptide hormones or amine transmitters, NEN are derived from either a pluripotent stem cell or a multipotent endorcine precursor cell, which also determines the extent of differentiation, but dedifferentiation may occur as well. When diagnosing a NEN, it is essential to specify its proliferative capacity (i.e. WHO-grading). This can be achieved by either determining mitotic rate or Ki67-index. If < 3% NEN are considered NE tumors (NET) of grade 1 (G1), while if 3-20% as NET G2, and >20% as NEN G3. NEN G3 according to WHO can now be termed NET G3 if their conventional histomorphology resembles that of NET G1/G2 but their Ki67-index is above 20%; if poorly differentiated they are termed NE carcinomas (NEC, implying G3 grade inherently). NEC bear a significantly worse prognosis than NET, while NET G1 fare better than NET G2 which fare better than NET G3.
At least as important is UICC-TNM-staging, which is specifically defined for NEN of any GEP-organ.
For clinical diagnosis of a NEN it is thus essential to define
- presence of a clinically manifest hormone hypersecretion syndrome
- the primary tumor organ within the GEP system
- the histopathological entity (i.e. CgA and SYN) and differentiation (NET or NEC)
- the proliferative capacity (i.e. WHO-grading) and
- staging according to UICC-TNM-classification.

For clinical staging the usual clinical techniques of direct, indirect or functional imaging can be applied depending on the clinical situation. By these means a NEN can and should be clearly defined and appropriate management strategies can be tailored accordingly.

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The role of advanced endoscopy in diagnosis and treatment of GEP-NETs

Horst Neuhaus, Germany

Learning Objectives:

- Classifying gastrointestinal neuroendocrine neoplasms (NENs)
- Endoscopic diagnosis and characterization of NENs
- Indications and selective endoscopic treatment of NENs

Abstract:

Background: Since diagnostic endoscopies of the GI tract are widely used, gastric, duodenal, and colorectal neuroendocrine neoplasms (NENs) are increasingly discovered, mostly as an incidental finding1. Their prognosis is dependent on the primary site, histological classification and stage. Whether a curative endoscopic treatment can be performed depends on tumor size, degree of infiltration and presence of lymph node metastases.
Gastric NEN: According to the German guideline2 gNEN type I/II, 1-2cm without additional risk factors (RF) (T1, G1, Ki-67 < 10%, L0, V0) should be resected endoscopically, gNEN < 1cm can be controlled. For gNEN types I/II < 2cm with G2 differentiation no consistent studies are available, therefore, depending on patient characteristics an individual decision should be made (endoscopic or surgical approach). gNEN Type III < 1cm should be resected endoscopically to obtain an optimal histopathological assessment. In the absence of RF (no deep sm invasion, G 1/2, L0, V0), additional surgery is not required.
Duodenal NEN: Well differentiated, non-functional dNEN ≤1cm without additional RF (T1, Ki-67 < 2%, G1, L0, V0), can be resected endoscopically. dNEN with RF (>2cm, Ki-67 >2%, G3, L1/V1) and gastrinomas require surgery, therefore, prior to endoscopic treatment of a small duodenal lesion suspicious for dNEN, histology acquisition, determination of serum gastrin levels, an EUS as well as a special anamnesis concerning gastrinoma and MEN1 must be performed2.
Rectal NEN: Rectal NEN which are restricted to the mucosa/submucosa, have a maximum diameter of 1cm an do not have additional risk factors (G1, L0, V0) can be resected endoscopically. For small (< 1cm) lesions with G2 differentiation and lesions between 1-2cm in size and G1 differentiation, no uniform studies and guidelines are available. Therefore, depending on further risk factors, age and comorbidities, a decision should be made between a local endoscopic or a radical surgical procedure2.

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Disclosure:

Nothing to disclose

Systemic treatment options for symptoms and tumour growth control in GEP-NETs

Philippe Ruszniewski, France

Learning Objectives:

- To indicate the available systemic treatment options in patients with digestive neuroendocrine tumors
- To be able to list the randomized clinical trials having shown efficacy of systemic treatments in patients with digestive neuroendocrine tumors
- To describe the principle of peptide-receptor radiotherapy, and its application to digestive NETs

Abstract:

Treatment of symptoms: Treatment of Zollinger-Ellison syndrome relies on proton pump inhibitors. Diazoxide is generally effective to control the consequences of insulin hypersecretion. SST analogs are used for VIPoma and glucagonoma. Patients with carcinoid syndrome should receive SST analogs, while telotristat might be useful in case of refractory diarrhea.
Treatment of tumoral process:
Somatostatin analogs: Their antitumor efficacy has been demonstrated in the phase III PROMID and CLARINET trials. (advanced, G1 or G2 (Ki67 < 10%) entero-pancreatic neuroendocrine tumor, not progressive and with liver involvement < 50%).
Systemic chemotherapy: Remains the treatment of reference of metastatic pancreatic NET: streptozotocin with doxorubicin or 5-fluorouracile, but also 5-fluorouracile-dacarbazine (or their oral prodrugs capecitabine and temozolomide) or oxaliplatine (FOLFOX). Chemotherapy is poorly effective in patients with intestinal NET.
Targeted therapies: Sunitinib is an oral inhibitor of the tyrosine kinases and has demonstrated significantly prolonged PFS in patients with advanced progressive pancreatic NET (median 11.1 vs 5.5 months). The RADIANT-3 phase III trial demonstrated that everolimus (Akt/mTOR pathway inhibitor) prolonged PFS in patients with advanced progressive pancreatic NET (11 vs 4.6 months). Everolimus (but not sunitinib) has also demonstrated efficacy in intestinal NET.
Peptide receptor radionuclide therapy (PRRT): It consists in the intravenous injection of a SST analog radiolabelled with 177Lutetium. The NETTER-1 trial demonstrated prolonged PFS in comparison with double-dose octreotide, in patients with metastatic well-differentiated intestinal neuroendocrine tumors (> 30 months, vs 8.4 months). PRRT can be also proposed in patients with progressive, metastatic pancreatic NET, mainly with liver involvement < 50% and/or with extrahepatic metastases and with strong uptake at SST isotopic imaging.

References:

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Disclosure:

Scientific advisor to Novartis, Ipsen, AAA and Keocyt.

How to improve quality in endoscopy

08:30-10:30 / C3

Why and what should we measure to assess quality in upper GI endoscopy?

Raf Bisschops, Belgium

Learning Objectives:

- Understand the need and rationale to measure quality in upper GI endoscopy
- Understand the evidence supporting the use of key performance measures
- Understand how to conduct a quality survey for upper GI endoscopy practise in your unit

Abstract:

ESGE and UEG have identified quality of endoscopy as a major priority. Like for colonoscopy, also quality in upper GI endoscopy differs significantly. Gastric cancers and precursor lesions are frequently missed: in one series, 7.2% of patients with gastric cancer did not have the lesion detected at previous endoscopy within 1 year (1). Inspection time for lesion detection is important : slow endoscopists with an inspection time of seven minutes or more detected significantly more high risk lesions in the stomach in comparison to fast endoscopists (14% versus 6%) (2). Similarly, in most epidemiological studies for Barrett's oesophagus, it is shown that up to 36 % of cancers are detected within 1 year of diagnosis indicating that these lesions were probably missed during the first endoscopy (3). In patients referred with Barrett's dysplasia without clearly visible lesions, expert eyes can detect lesions in 75% of cases, even including lesions that need straight referral for surgery (4).
In 2016, ESGE and UEG have published quality measures (QMs) for upper GI endoscopy (5). The development of these QMs was as much as possible based on available evidence (5). At this stage it is not entirely clear if all PMs will be useful for auditing upper GI practice. Selection of QMs may differ according to the prevalence of a disease (eg Barrett of stomach cancer). After this first step of development of performance measures, the next step would be implementation. Here lies a major role for national societies to assist individual endoscopist to overcome barriers that were identified in an ESGE survey (administrative, financial and motivational). National societies may facilitate surveys and emphasize the simplicity of auditing based on a small sample of +/- 200 gastroscopies. In the end, we owe it to our patients to overcome individual or financial barriers to ensure that endoscopy services are of the highest quality.

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Why and what should we ensure to assess quality in lower GI endoscopy?

Monika Ferlitsch, Austria

Learning Objectives:
- Knowing quality measures in lower GI endoscopy
- Monitoring quality measures
- Improving quality measures

Abstract:
Efficacy of screening colonoscopy depends on the quality of examination. Low Adenoma detection rate (ADR) is associated with a higher risk of interval cancer and should be at least 25%. Further, intubation of the cecum should be ensured in 90% of examinations, as the risk of interval is increasing with lower cecal intubation rate (CIR). However, a clean colon is necessary for high ADR & CIR. A study by Clark et al. compared the miss-rate of adenomas >5mm comparing boston bowel preparation score (BBPS) of 1vs.2 and showed a 10,7% higher miss rate among BBPS 2. Therefore, monitoring these measures is crucial. In Austria data are entered via electronical report by endoscopists themselves and quality measures are calculated automatically. A benchmark report is provided to all participants as feedback of their performance. Since 2007 ADR, CIR & adequate bowel preparation increased and current mean ADR is 24.2%, compared to 18.5% in 2007, representing the importance and benefit of a screening program. Further, annually random samples of pictures of coecum, polyps and polypectomy site are compared with the entered datasets. To ensure the cleanness of endoscopes sample of the working, air and the water channel of the endoscope and rinsing fluid of the washing machine are also investigated at least 1/year.

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Disclosure:
Nothing to disclose

Importance of sterilisation and radioprotection: The clinician's perspective

Daniel Blero, Belgium

Learning Objectives:
- To know adverse effects of X-rays utilization
- To be aware of the main measures to protect patients and staff members from X-rays exposure
- To implement measure(s) that improve X-rays manipulation

Abstract:
The use of X-rays in gastroenterology is mainly related to the performance of therapeutic acts such as ERCP, dilatations and placement of stents. However, X-rays induce two major types of "adverse" effects:
- deterministic effects, related to a dose effect, responsible for cell death in tissues, the potential consequences of which include radiodermatitis, cataract or impaired fertility
- stochastic effects, independent of the dose, related to the occurrence of somatic carcinogenic or even teratogenic mutations (in the pregnant woman).
The use of ionizing radiation is framed by European legislation translated into laws in member countries. It obliges users and staff members exposed to such radiation to undergo training and attend continuing education courses. The goal is to stimulate the use of the lowest possible doses to obtain a usable quality image. In order to achieve this, an understanding of the technical capabilities of the device with which one works is essential. Limiting the time of fluoroscopy, bringing the patient closer to the receiver, using the functions of replay of the sequences of fluoroscopy and the capture of reference images are some measures making possible to reduce the administration of X-rays. The distance to the source, installation of protective panels around the machine, wearing of glasses, thyroid protector and apron are the main measures limiting the exposure of the staff. Finally, users are also required to measure and record the dose at which the staff members but also the patients are exposed. Follow-up of these measurements compared to reference doses levels listed either nationally or in the literature also reduces the doses administered during digestive endoscopy examinations.

References:
Dumonceau et al : Radiation protection in digestive endoscopy: ESGE Guidelines. Endoscopy 2012;44:408-24. Kelsey et al: Radiation safety in the endoscopy setting. Gastroenterol Nurs 2001;24:143-6.

Disclosure:
Nothing to disclose

Importance of sterilisation and decontamination: The nurse's perspective

Helen Griffiths, United Kingdom

Learning Objectives:
- To be aware of endoscope-related infections risks
- To know the current disinfection goals and process
- How to assess and check that disinfection has been done properly

Abstract:
Endoscopists assume that the endoscope they are handed has been appropriately decontaminated, but can you honestly say that you understand the process and the pathway by which the instruments you use are reprocessed? Do you understand the associated risks if the pathway fails and your part in supporting the teams (nurse and technicians) responsible for this critical role? There has been an increased worldwide focus on infection associated with endoscopic decontamination. This is following an alarming increase in the incidence of endoscopy-associated infections and deaths between 2012 and 2015 associated with Carbapenemase-resistant Enterobacteriaceae.
A human factors approach to decontamination starts with an understanding of things that support or hinder the way people work. So there are critical points that every endoscopist needs to consider regarding decontamination to be assured that the endoscope that they pick up is fit for purpose and safe. Successful decontamination of endoscopes is everyone's business not just nurses.

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Beilenhoff U et al. Reprocessing of flexible endoscopes and endoscopic accessories used in gastrointestinal endoscopy: Position Statement of the European Society of Gastrointestinal Endoscopy (ESGE) and European Society of Gastroenterology Nurses and Associates (ESGENA) - Update 2018. Endoscopy 2018; 50: 1205-1234.

Disclosure:
Nothing to disclose

Improving safety in your endoscopy unit

Siwan Thomas-Gibson, United Kingdom

Learning Objectives:

- Understand the challenges regarding safety in endoscopy
- Describe ways of improving safety and reducing error
- Understand the importance of learning from error to improve safety

Abstract:

Safety in Endoscopy is a priority in every endoscopy unit, so what's new? In 2019 we face several challenges that require us to review ways in which safety could be improved. This presentation outlines three main challenges that I see in 2019 and will describe how to learn from error and adverse events to improve safety.

There is an increasing demand for endoscopy throughout the western world. In the United Kingdom alone more than 2.5 million endoscopic procedures were performed in 2016. There are many drivers for this increasing demand.

The focus on early diagnosis and screening for gastrointestinal cancer has increased.

Advancing technology has improved our ability to detect pathology and has blurred the boundaries between surgery and endoscopy such that endoscopists are now able to offer an increasingly complex array of therapeutic procedures. Demand for these increases as clinicians and patients choose a less invasive non-surgical approach for many gastrointestinal disorders. Many of the patients we see are of advanced age with multiple co-morbidities.

Finally, with increasingly complex pathways how can the endoscopy community communicate with eachother better? It is almost impossible to keep up with every guideline, position statement, new technology, classification system, therapeutic intervention... And how can we learn from eachothers errors and safety incidents to prevent the same error from happening time and again? In an age where communication should be easier we are perhaps overwhelmed with information.

The UK Joint Advisory Group for GI endoscopy has developed a strategy which aims to Improve Safety and Reduce Error in Endoscopy (ISREE). This has five work streams which can be developed and delivered at endoscopy unit or at regional or national level:

· Prevention of patient safety incidents
· Training in non technical and team skills
· Promoting incident reporting
· Learning from incidents
· Supporting endoscopists in difficulty

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Disclosure:

I have received educational support funds from Norgine, Aquilant and Olympus.

Obstruction and ileus

08:30-10:30 / F2

Causes and investigation of ileus

Tom Moreels, Belgium

Learning Objectives:

- To understand the pathophysiology of paralytic (postoperative) ileus
- To be able to clinically assess and to perform the diagnostic workup of the patient at risk for paralytic ileus
- To be able to distinguish paralytic ileus from other GI hypomotility pathologies in clinical practice

Abstract:

Paralytic ileus is defined as constipation and intolerance of oral intake due to non-mechanical factors that disrupt normal gastrointestinal propulsive motor activity. One of the most prevalent and best-known forms of paralytic ileus is postoperative ileus. However, paralytic and postoperative ileus remain both a research as well as a clinical challenge. The current presentation highlights its pathophysiology and clinical work-up in a concise and comprehensive way.

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Disclosure:

Nothing to disclose

Surgical management of acute intestinal obstruction

Not available

Endoscopic management of acute intestinal obstruction

Gareth Corbett, United Kingdom

Learning Objectives:

- Understanding of the options available to treat intestinal obstruction endoscopically
- To have the knowledge base for endoscopic interventions in practical terms
- Understanding of the technique of endoscopic self expanding metal stent insertion for the treatment of intestinal obstructing lesions

Abstract:

Diseases which cause obstruction of the gastrointestinal tract have endoscopic interventional options which can provide either an alternative to surgical intervention, or as a bridge to this. These conditions can be divided into neoplastic, inflammatory and functional disorders. The main endoscopic therapies available are balloon dilatation, self-expanding metal stent (SEMS) insertion and endoscopic decompression.

Up to 85% of patients presenting with colonic obstruction have a colorectal cancer as the underlying cause [1]. Compared to surgery, colonic stenting is non-inferior in terms of mortality and length of stay and reduces the rates of stoma formation [2].

For patients with pancreatic or gastric cancer surgical resection is not an option for 80-85% and 40% respectively [3]. 15-20% of patients with inoperable pancreatic cancer will present with gastric outlet obstruction. The traditional palliative option for this is gastrojejunostomy. Endoscopic SEMS has been shown to be no different in terms of technical success and complications. Length of stay after SEMS appears shorted than after surgery, but survival appears longer after surgery [4].

In patients with short strictures, either inflammatory or post-surgical, pneumatic dilatation has been shown to be safe and effective as an alternative to surgery [5].

For patients presenting with obstruction secondary to sigmoid volvulus or pseudo-obstruction endoscopic decompression may be performed. In patients with sigmoid volvulus an emergency decompression as a bridge to surgery is a safe and effective treatment to prevent colonic gangrene [6]. For patients who are unfit for surgery repeated endoscopic decompressions or percutaneous endoscopic colostomy (PEC) could be considered [7]. For patients with pseudo-obstruction the role of endoscopy is more controversial, but appears to be helpful in preventing acute complications such as perforation secondary to colonic ischaemia [8].

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Disclosure:

Nothing to disclose

Therapy update: Faecal transplantation

08:30-10:30 / F3

FMT procedure, regulation and stool banks

Maria J.G.T. Vehreschild, Germany

Learning Objectives:

- To understand the different technical options available for FMT
- To understand advantages and disadvantages of available options
- To understand current developments in the regulation of FMT products

Abstract:

The clinical application of fecal microbiota transfer (FMT) for the treatment of recurrent Clostridioides difficile infections (rCDI) has increased rapidly since the first publication of a randomized controlled trial in 2013. In the following years, ilts efficacy has been extensively confirmed, not only in further randomized clinical trials, but also in daily clinical practice. In 2013, the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) included the microbiota transfer FMT in the guidelines for the treatment of recurrent CDI with an AI recommendation. Developments in application technique include frozen and freeze-dried preparations.

Across trials, response is similar at approximately 80%. In this presentation, different factors potentially determining treatment failure and success will be discussed. Furthermore, an overview on current safety data will be given and recommendations with respect to different treatment approaches will be made on the basis of safety considerations.

Finally, current international developments in the regulation of FMT will be presented and discussed, including the concept of commercial and non-commercial stool banks, as well as the consequences of classsification of FMT products as drugs.

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Disclosure:

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FMT in clinical practice for C. difficile infection: Pro

Ed Kuijper, Netherlands

Learning Objectives:

- FMT is effective and safe for treatment of patients with multiple recurrent C. difficile infections
- FMT should be coordinated by national stool banks
- A national registry is recommended for follow-up of patiients and donors

Abstract:

Faecal Microbiota Transplantation (FMT) has been demonstrated as the preferred first therapeutic intervention for patients with multiple recurrent Clostridioides difficile infections (rCDI), as published in various guidance documents. The effectiveness of FMT treatment (higher than 90%) has not been approached by other treatments for rCDI, though no studies have been performed with bezlotoxumab and fidaxomicin (or extended fidaxomicin) as comparison. Several studies on cost-effectivess including a long term follow-up of 1 year after FMT to analyse readmissions and complications, also indicate that FMT is the prefered option for multiple rCDI. The costs of one FMT treatment as determined in a budget impact analysis at the Netherlands Donor Feces Bank (NDFB), are approximately 1000 Euro, not including the administration by colonoscopy or nasoduodenal route. An important prerequisite to apply FMT is the

setting of stool banks, approved by national authorities. In 2019, experts from different countries established new guidelines for stool banks that provide safe and effective FMT products for the treatment of rCDI and for use in trials assessing other indications. Stool banks are non-profit organisations and have standard operation procedures for screening of donors, preparation of the product, storage and administration to patients. The long term effects of FMT are unknown. Since FMT is also mentioned in the guidelines for treatment of children with rCDI, a national registry should be used by the stool banks to detect possible adverse events in patients during a folllow-up of 10 to 15 years. Donors are also included in the folllow-up to recognise diseases of which transmission to donors could be possible. In summary, FMT will remain as the first treatment choice for patients with rCDI, unless new regulations will classify donor feces suspensions as a drug or pharmaceutical product.

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Disclosure:

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FMT in clinical practice for C. difficile infection: Con

Mark Wilcox, United Kingdom

Learning Objectives:

- FMT remains an experimental therapy at present, especially as we do not know the long term effects of manipulation of the human gut microbiome
- Serious untoward events have been reported following FMT
- What are the proven alternatives for FMT for the treatment of recurrent C. difficile infection?

Abstract:

We know that the microbiome of the human gut is involved in the pathogenesis of multiple diseases, and there is increasing evidence that its roles may be fundamental/far reaching for human health/disease. While FMT has been shown to be an effective option in the management of patients with multiple recurrences of C. difficile infection, it is important to recognise that this remains an experimental therapy at present, especially as we do not know the long term effects of manipulation of the human gut microbiome. Notably, serious untoward events have been reported following FMT. There is no systematic collection of controlled outcomes/adverse events following FMT. There are multiple alternatives to FMT for the treatment of recurrent C. difficile infection, including agents that have undergone more rigorous safety assessments. These include options that can avoid the use of FMT.

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Disclosure:

I have received: Consulting fees from Actelion, Astellas, bioMerieux, Da Volterra, Merck, Meridian, Pfizer, Sanofi-Pasteur, Seres, Singulex, Summit, Synthetic Biologics, Valneva, Vaxxilon & VenatoRx; Lecture fees from Alere, Astellas, Merck, Pfizer & Singulex; and Grant support from Actelion, Alere, Astellas, bioMerieux, Da Volterra, Merck, MicroPharm, Morphochem AG, MotifBio, Paratek, Sanofi-Pasteur, Seres, Summit & Tetraphase.

FMT for IBS

Andreas Petersen, Denmark

Learning Objectives:

- The composition of the gut microbiota in IBS patients of all subtypes is different from healthy controls
- Putative mechanisms explaining a possible role of microbiota in the development of IBS include altered metabolic activity of the microbiota, mucosal immune activation, impaired mucosal barrier function and a disturbed gut-microbiota-brain axis
- Meta-analysis of results from RCTs does not suggest a benefit of FMT for global IBS symptoms, however, this could possibly be explained by differences in patient subgroup and FMT modality

Abstract:

Irritable bowel syndrome (IBS) is the most common gastrointestinal disorder, affecting up to one in five people [1]. In accordance with the Rome IV criteria [2], IBS is characterized by abdominal pain and altered bowel function [2]. The exact cause of IBS is still unknown. A meta-analysis has confirmed a gut microbial dysbiosis in IBS in comparison with healthy controls [3]. The putative mechanisms that explain the role of microbiota in the development of IBS include altered metabolic activity, mucosal inflammation, impaired mucosal barrier function, and a disturbed gut-microbiota-brain axis [4]. Therefore, modulation of the intestinal microbiota has been suggested as a strategy for managing IBS symptoms [5]. Worldwide, interest in fecal microbiota transplantation (FMT) as therapy for several diseases, including IBS, is growing rapidly. Feces from healthy donors contain more than a hundred different types of bacteria, along with e.g. parasites, viruses, fungi, and bacteriophages. In recurrent Clostridium difficile infections, FMT has shown excellent effects. FMT has a much higher cure rate than standard treatment [6] and studies have shown that FMT might restore intestinal microbial balance in treated patients [7]. FMT could therefore theoretically be a possible treatment for IBS patients. To date, four randomized placebo-controlled studies and few non-placebo studies have evaluated whether FMT is effective in IBS patients [8, 9, 10]. Of the four placebo-controlled studies, involving in all 254 participants, two shows a positive effect and two, in fact, a negative effect of FMT. These differences could possibly be explained by patient subgroup and FMT modality. However, current evidence from RCTs does not suggest a benefit of FMT for IBS. Considering IBS as a rather benign condition and FMT as a treatment with possible unknown side effects caution is recommended, both when considering FMT as a treatment option in IBS and when designing new studies.

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Disclosure:
Nothing to disclose

FMT in IBD

Severine Vermeire, Belgium

Learning Objectives:
- To learn what is the current evidence for FMT in ulcerative colitis, Crohn´s disease and pouchitis
- To review the European Guidelines on FMT with respect to indications, and screening
- To understand future applications of microbiota-focussed therapies

Abstract:
Faecal microbiota transplantation (FMT) is a successful therapy for patients suffering from refractory Clostridium difficile infections.
While comprehensive culture- and molecular-based analyses of the microbiota of IBD patients have failed to identify consistent enrichment of pathogenic species, several studies have shown decreased microbial diversity of the intestinal ecosystem in IBD patients. Given this observed dysbiosis, faecal transplantation has also been suggested as a treatment option for patients with refractory IBD. A number of placebo-controlled randomized trials have been performed which together suggest that FMT may increase clinical and endoscopic remission rates in UC. However, the number of studies, the included patients and the quality of the evidence remains overall limited and further work is needed.
A number of open questions also remain such as the preferred route and frequency of administration, what the donor should look like and which subphenotype of disease would be most suited for this type of therapy. In addition, no studies assessed long-term maintenance of remission in UC or CD.

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Disclosure:
S Vermeire received consultancy from Prodigest and Actobio

Constipation: To modify behaviour or anatomy?

11:00-13:00 / Hall 6

Behavioural modification of defecation disorders

Jan Borovicka, Switzerland

Learning Objectives:

- To understand the role of functional defecation disorders in the pathophysiology of constipation
- To identify the benefit of biofeedback-aided treatments in patients with constipation
- To be aware of the evidence of biofeedback-treatment in randomized controlled trials

Abstract:

The behavioral modification of defecation disorders is based on the understanding of the pathophysiology of functional defecation disorders which are recognized as an important element in the diagnosis of constipation. To diagnose dyssynergic defecation (DD) there must be objective features of impaired evacuation, as demonstrated by 2 of the following 3 tests: Abnormal balloon expulsion test, Abnormal anorectal evacuation pattern with manometry or anal surface EMG, Impaired rectal evacuation by imaging. Symptoms per se are not reliable for DD as shown by Rao et al. Therefore physiologic assessments of defecation are important to guide further treatment. Biofeedback in case of DD is considered as a second line treatment due to the restricted availability but also due to the often insufficient adherence of patients. Biofeedback therapy provides sustained improvement of bowel symptoms and anorectal function in constipated subjects with DD, whereas standard therapy is largely ineffective. Chiarioni et al. studied 104 patients with DD and constipation in a randomized controlled trial and could show that biofeedback is more effective than polyethylene glycol plus 5 weekly counselling sessions. Benefits as obtained by biofeedback were sustained at 12 and 24 months. Depending on the therapist's skill and the patient's motivation the response to biofeedback therapy may vary considerably from one center to the other. Aided by mostly visual or less common auditory feedback of anorectal and pelvic floor pressure (manometry-based devices) or muscle activity (EMG-based devices), patients are motivated to increase intra-abdominal pressure and relax the pelvic floor during defecation. When rectal sensation is reduced, e.g. in patients with rectal prolapse, sensory retraining with stepwise inflation of rectal balloons may also be provided. Rao et al. showed that home-based and office-based biofeedback as a therapy for DD improved bowel symptoms and physiology with similar efficacy.

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Disclosure:

Nothing to disclose

Surgical management of constipation

Charles H. Knowles, United Kingdom

Learning Objectives:

- To understand the main indications for surgery for constipation
- To understand the need for triple assessment and MDT review before surgery is contemplated
- To understand that available procedures have a benefit vs. harm profile that makes few (if any) very attractive

Abstract:

The management of most patients with constipation should not include surgery for reasons of irreversibility, unpredictable functional outcomes and potential harm, as well as the basic fact that surgery (excepting neuromodulation +/- future cell therapies) can only alter gross anatomy rather than neuromuscular function.

There are 2 main pathophysiological targets for surgery: generalised colonic transit delay and defined dynamic structural causes of obstructed defaecation. The former represent about 5% of patients undergoing specialist investigations, the latter perhaps 10%. There is no place for surgery for patients with normal physiology, those with IBS, or those with functional defecation disorders.

The talk highlights the importance of triple assessment (symptoms & signs, examination and specialist investigations) and MDT decision making (including appreciation of all pelvic floor compartments). For patients with generalised slow-transit constipation, the historical index surgical procedure is colectomy. The talk discusses the rare indications (and many contra-indications) for this approach, its potential benefits and its substantial harm profile. It also covers variations in approach and extent of resection i.e. alternatives to the best-studied but radical procedure of colectomy and ileorectal anastomosis.

For patients with mechanical defecation disorders there are 2 common surgical targets, alone or in combination: rectocele and rectoanal intussusception. As with colectomy, the indications and contraindications as well as realistic outcomes and harms (e.g. current mesh issues) of the numerous procedures (hitching, reinforcing and excisional) are summarised in overview.

Finally, the talk covers treatments that have been applied to patients more generally. These include sacral neuromodulation (now proved to have little or no benefit in randomised trials); anterograde irrigation procedures (in the speaker's min often overlooked) and stoma.

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Disclosure:
Nothing to disclose

Anorexia and unexplained weight loss

11:00-13:00 / A1

Malnutrition: Causes and diagnostic criteria

André Van Gossum, Belgium

Learning Objectives:

- To define nutrition disorders and nutrition related conditions
- To explain the new GLIM criteria for the diagnosis of malnutrition
- To determine the tools for assessing the body composition

Abstract:
Malnutrition can be defined as "a state resulting from lack of intake or uptake of nutrition that leads to altered body composition (decreased fat free mass) and body cell mass leading to diminished physical and mental function and impaired clinical outcome from disease". Undernutrition is considered as a synonym of malnutrition.
Malnutrition may be classified as: 1) disease-related malnutrition with inflammation (acute or chronic); 2) disease-related malnutrition without inflammation; 3) malnutrition/undernutrition without disease (socio-economic/psychologic problems or hunger-related malnutrition.
The international community in the field of clinical nutrition (ESPEN, ASPEN, FELAPE, PENSA) has recently proposed some new criteria for diagnosing malnutrition.
The first step should be to screen the patients who are at risk of malnutrition using some validated tools (NRS-2002, MUST, MNA).
The selected criteria for defining malnutrition are: 3 phenotypic: weight loss, BMI, muscle mass and 2 etiologic: decrease of food intake or malabsorption and inflammation.
Some cut-offs for the phenotypic criteria have been proposed for grading the malnutrition.
A patient is considered to be malnourished when having one abnormal phenotypic criteria and one abnormal etiologic criteria.
Validation studies are needed to adapt these criteria.
Fat free mass and fat mass can be estimated by bio-impedance analysis (BIA) or DXA-scan, standard anthropometric measurements, such as mid-arm-circumference, calf-measurement or skinfold to thickness are potential alternatives although subject to measurement variability.
Computerized tomography (CT) is being increasingly used to evaluate muscle mass depletion, especially in patients with digestive disorders who require an abnormal CT exam.
Biochemical markers, e.g. serum concentrations of visceral protein (albumin) should not be used as indicators of a patient's nutritional status.

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Disclosure:
Nothing to disclose

Motility disorders: Nutritional treatment

Francisca Joly, France

Learning Objectives:

- Sensitize to gastrointestinal disorders in patients with eating disorders
- Determine the nutritional support to be proposed in a patient with motility disorder in a context of eating disorders
- Prevent the refeeding syndrome

Abstract:

Anorexia nervosa (AN) is an eating disorder characterized by body schema disruptions, underweight, self-induced weight reduction, and endocrine impairments.Gastrointestinal alterations can be particularly bothersome by patients. Decreased gastric motility and delayed gastric emptying can result in further complications. Both rarely lead to acute gastric dilation, a surgical emergency that can result in gastric necrosis, perforation, shock, and death. Lower intestinal motility can be affected as shown by a prolonged gastrocecal transit time, by whole-gut transit time, and by pelvic floor dysfunction. Because most AN patients reported the desire to lose weight before the onset of GI symptoms, AN is rather the cause and not the consequence of abnormal GI functions and related symptoms. If most of these are reversible following treatment and weight regain, the presence of GI symptoms can make the nutritional approach difficult. Different approaches including oral feeding, enteral nutrition (EN), or parenteral nutrition (PN) should be tailored to each individual. In general, EN is always preferable to PN because of preservation of gut function, fewer infectious and metabolic complications. Patients with AN are high risk patients for refeeding syndrome. Refeeding syndrome is a potentially fatal condition, caused by rapid initiation of refeeding after a period of undernutrition. It is characterised by hypophosphataemia, associated with fluid and electrolyte shifts and metabolic and clinical complications. Awareness of refeeding syndrome and identification of patients at risk is crucial as the condition is preventable and the metabolic complications are avoidable. Refeeding should be started at a low level of energy replacement. Vitamin supplementation should also be started with refeeding and continued for at least 10 days. Correction of electrolyte and fluid imbalances before feeding should be done alongside feeding.

References:

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Disclosure:

Nothing to disclose

Oesophageal cancer: Challenges and controversies

11:00-13:00 / A2

Oesophageal cancer: Diagnosis and treatment

Prateek Sharma, United States

Learning Objectives:

- Discuss the role of resection in the diagnosis and staging of esophageal cancer
- List the various endoscopic therapies available for the treatment of Barrett's esophagus and cancer
- Summarize new insights about recent guidelines on Barrett's esophagus and cancer

Abstract:

Introduction: Barrett's esophagus (BE), a premalignant condition where the normal squamous epithelium of the esophagus transforms into columnar epithelium with intestinal metaplasia, is the most important risk factor for development of esophageal adenocarcinoma. The risk of this progression from non-dysplastic BE is relatively low with annual risk of 0.3 % to 0.5%, however, once adenocarcinoma develops the 5 year survival is 15 %.

The latest gastroenterology society guidelines recommend endoscopic eradication rather than esophagectomy for treatment of patients with early adenocarcinoma with endoscopic therapy. Previously, the gold standard of treatment used to be esophagectomy, however, due to high perioperative morbidity and mortality, it has been largely replaced by endoscopic techniques that are now the current mainstay of treatment.

Diganosis and Treatment: The initial diagnosis of esophageal cancer requires a meticulous and careful examination of the esophageal mucosa with high defintion endoscopy with or without electronic chromoendoscopy. The biopsy diagnosis of esopahgeal cancer is confirmed and then staged by endoscopic mucsal resection of the visible lesions in the esophagus (EMR). EMR removes the mucosal and sub-mucosal layers of tissue either by raising the targeted segment with sub-mucosal injection followed by resection (cap EMR) or the suction, banding and cut method (multi-band ligation). This allows for accurate diagnosis and differnetiation between T1(a) and T1(b) cancers.

EMR and and mucosal abaltion is generally combined, and recent data suggest this can be done safely within a single session.

Conclusions: An accurate diagnosis of early dysplastic disease is the first hurdle and can be helped by seeking histologic confirmation by a second pathologist. Studies have also shown that use of high defintion endoscopy and advanced imaging techniques can be effective in the endoscopic inspection for neoplasia. Endoscopic therapy has shown excellent results.

References:

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Disclosure:

Consultant: Olympus Grant support: US Endoscopy, Ironwood, Erbe

Changing epidemiology of oesophageal cancer and its impact

Isabelle Soerjomataram, France

Learning Objectives:

- Understand major causes of oesophageal cancers, their current and historical pattern
- Understand current landscape of oesophageal cancer and its substypes linked to changes in risk factors
- Understand the impact on characteristics of cases diagnosed with oesophageal cancer and their management

Abstract:

Survival of patients diagnosed with oesophageal cancer remains poor, as such that it is today the sixth most common cause of cancer mortality. Oesophageal cancer is predominantly categorised into two main histological subtypes: adenocarcinomas (AC) that are typically located in the lower third of the oesophagus and are linked to Barrett's oesophagus and squamous cell carcinoma (SCC) that develops mostly in flat cells

lining the upper part of the oesophagus. Changing pattern of major causes of oesophageal cancers has completely change the current landscape of cases diagnosed today with oesophageal cancer subtypes. While SCCs are highly linked with smoking and alcohol consumption, ACs mainly occur in patients with gastro-oesophageal reflux, which in turn is associated with obesity. Today global findings showed that although SCC is the more common type of oesophageal cancer globally, yet incidence rates of AC have surpassed SCC rates in high-income countries - changing completely clinical practice in terms of oesophageal cancer management. This session will provide a comprehensive view of current challenges and controversies in oesophageal cancers, in terms of its epidemiology and management followed by case presentation to illustrate common challenge that is found in clinical practice. Presentation on the most up-to-date diagnostic strategies and management of this disease will go through all modalities used to manage the different oesophageal subtypes. This is complemented with a presentation providing epidemiological perspective on the current burden of the disease, future projection as well as survival differences between countries highlighting impact on current and future clinical practice.

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1. Arnold M, Soerjomataram I, Ferlay J, Forman D. Global incidence of oesophageal cancer by histological subtype in 2012. Gut. 2015 Mar;64(3):381-7. 2. Eileen Morgan, Isabelle Soerjomataram, et al. International Trends in Oesophageal Cancer Survival by Histological Subtype: A population-based study from seven high-income countries 199.5-2014. Submitted.

Disclosure:
Nothing to disclose

Challenging the small bowel: Diagnosis and management of small bowel tumours in 2019

11:00-13:00 / A3

How to investigate the small bowel when a small bowel tumour is suspected

Marco Pennazio, Italy

Learning Objectives:
- Recognize clinical presentation
- Knowledge of patients at increased risk for small bowel cancer
- Appropriate diagnostic work-up

Abstract:
Small bowel tumours (SBTs) are rare and usually remain asymptomatic for years, The clinical presentation is often indolent and nonspecific. Most cases of SBTs are unexpectedly diagnosed in the course of a diagnostic workup of patients with suspected SB bleeding, unexplained iron-deficiency anaemia and persistent abdominal pain, or during the follow-up of patients at an increased risk to develop SBT (FAP, Peutz-Jeghers syndrome, Lynch syndrome, Crohn's disease, refractory celiac disease). Early diagnosis is of paramount importance. Although SBTs could be suspected by means of routine abdominal ultrasound or computed tomography (CT), at the present time, imaging tools specifically designed for the study of the SB include capsule endoscopy (CE), device-assisted enteroscopy (DAE) and SB cross-sectional imaging techniques [CT or magnetic resonance (MR) enterography].CE has demonstrated a high diagnostic yield by identifying SB mucosal lesions, including SBTs in 3-10% of patients receiving CE. CE is burdened by an increased risk of capsule retention and/or missing proximal SBTs. Capsule retention is generally asymptomatic and, as most patients with SBT will require surgery, retrieval of the retained capsule can happen at that time. Radiologic cross-sectional techniques have a high sensitivity in identifying SBTs (85-94%) but have a low specificity. They allow, in a single examination, both identification and staging of SBTs. DAE is more often used in clinical practice as confirmatory tool, when other less invasive tests identify relevant findings. DAE has the advantage to allow both a detailed evaluation of the SB surface with tissue sampling and endoscopic treatments or of placing tattoos for more precise identification of SBTs at the time of surgery. In patients with SBT, the result of a single diagnostic exam is often insufficient to provide a definite diagnosis. A balanced combination of different tests allows reaching a final diagnosis and drive further management.

References:
1) Pennazio M. Endoscopy. 2015;47:352-76. 2) Rondonotti E. Curr Opin Gastroenterol. 2018;34:159-164. 3) Johnston CA. Endoscopy Internat Open 2017; 05:E463-E470. 4) Chung CS. J Formos Med Assoc. 2018;117:705-710. 5) Elli L. Gastrointest Endosc.2017;86:264-273 6) Soyer P. Eur Radiol 2013; 23:388-399. 7) Faggian A. Gastroenterol Res Pract 2016; 2016:9686815.

Disclosure:
Nothing to disclose

Therapeutic aspects

Omar Faiz, United Kingdom

Learning Objectives:
- Clinical symptoms and diagnostic work-up
- Differential Diagnosis of underlying conditions
- Therapeutic management

Abstract:
The four principal differential diagnoses of small bowel tumours are discussed. The latter include: adenocarcinomas, lymphomas, sarcoma/gastrointestinal stromal tumours and neuroendocrine tumours. The surgical and adjuvant management of each of these tumours are presented in sequence.
The emphasis of the presentation is on neuroendocrine tumours and, in particular, their anatomical distribution, surgical intervention, the management of hepatic neuroendocrine (NETs) metastases and survival following treatment for NETs.

References:
Howe JR1, Cardona K, Fraker DL, Kebebew E, Untch BR, Wang YZ, Law CH, Liu EH, Kim MK, Menda Y, Morse BG, Bergsland EK, Strosberg JR, Nakakura EK, Pommier RF.The Surgical Management of Small Bowel Neuroendocrine Tumors: Consensus Guidelines of the North American Neuroendocrine Tumor Society. Pancreas. 2017 Jul;46(6):715-731. doi: 10.1097/MPA.0000000000000846. Strosberg JR1, Halfdanarson TR, Bellizzi AM, Chan JA, Dillon JS, Heaney AP, Kunz PL, O´Dorisio TM, Salem R, Segelov E, Howe JR, Pommier RF, Brendtro K, Bashir MA, Singh S, Soulen MC, Tang L, Zacks JS, Yao JC, Bergsland EK. The North American Neuroendocrine Tumor Society Consensus Guidelines for Surveillance and Medical Management of Midgut Neuroendocrine Tumors. Pancreas. 2017 Jul;46(6):707-714. doi: 10.1097/MPA.0000000000000850.

Disclosure:
Nothing to disclose

Autoimmune pancreatitis: From diagnosis to treatment

11:00-13:00 / C1

Autoimmune pancreatitis: From etiology to diagnosis

Matthias Löhr, Sweden

Learning Objectives:
- Being familiar with the two subtypes of autoimmune pancreatitis
- Learn how to diagnose autoimmune pancreatitis
- Understand the complexity of AIP 1 / IgG4-related diseases (IgG4-RD)

Abstract:
It is pivotal to know that such diagnosis exists! AIP is defined by histology, and different in clinical appearance and imaging: type I (lymphoplasmacytic sclerosing pancreatitis (LPSP) and type II (idiopathic duct-centric pancreatitis (IDCP)1. AIP type I is a systemic disease (IgG4-RD syndrome). The etiology is unknown. As an external trigger to autoimmunity, H. pyloriwas suggested based on"molecular mimikry" with human carbonic anhydrase II (CA-II)2, the lead enzyme of pancreatic ducts. The sequence in common contains a binding motif of a HLA-class-II-haplotype, a risk factor for AIP3. The H. pyloriCagA protein has a high homology to the telomerase subunit UBR2, expressed in the pancreas4. However, no H. pyloriDNA or RNA could be found in AIP5. Viruses have been implicated, especially Hepatitis E virus6. A murine leucemia retrovirus could mimik human AIP sharing the same protein defects and autoantibodies7.
A consensus was reached diagnosing AIP8, relying on serology and imaging. In AIP type 1, >80% of patients have elevated serum IgG/IgG4; some patients have only tissue expression of IgG4. Autoantibodies have been described for ductal (CA-II9) and acinar antigens (lactoferrin10, SPINK7, 11, UBR24, amylase12, trypsinogens7). Autoantibodies against CA-II, SPINK-1 and trypsinogens were confirmed independently13. Only tests for lactoferrin and CA-II are available in Europe. They are positive in up to 20% of AIP patients.
Imaging reveals pathognomonic signs of AIP: a homogenously swollen pancreas with smooth, contrast enhancing rim; contrast enhancement in the distal bile duct and kidney lesions14(AIP type I only). Changes can be subtle and AIP can present as acute pancreatitis or pancreatic cancer with a pancreatic tumor. Positive imaging and elevated IgG/IgG4 constitutes the diagnosis of AIP type I. If in doubt, a biopsy from the papilla or via EUS may help to establish the diagnosis, according to certain criteria15, also allowing for IgG4 immunostaining.

References:
1. Chari ST, et al. Histopathologic and clinical subtypes of autoimmune pancreatitis: the Honolulu consensus document. Pancreas 2010;39:549-54. 2. Guarneri F, Guarneri C, Benvenga S. Helicobacter pylori and autoimmune pancreatitis: role of carbonic anhydrase via molecular mimicry? J Cell Mol Med 2005;9:741-4. 3. Ota M, , et al. Two critical genes (HLA-DRB1 and ABCF1) in the HLA region are associated with the susceptibility to autoimmune pancreatitis. Immunogenetics 2007;59:45-52. 4. Frulloni L, et al. Identification of a novel antibody associated with autoimmune pancreatitis. N Engl J Med 2009;361:2135-42. 5. Jesnowski R, et al. Helicobacter pylori in autoimmune pancreatitis and pancreatic carcinoma. Pancreatology 2010;10:462-466. 6. Pischke S, et al. Hepatitis E virus: Infection beyond the liver? J Hepatol 2017;66:1082-1095. 7. Löhr JM, et al. Autoantibodies against the exocrine pancreas in autoimmune pancreatitis: gene and protein expression profiling and immunoassays identify pancreatic enzymes as a major target of the inflammatory process. Am J Gastroenterol 2010;105:2060-71. 8. Shimosegawa T, et al. International consensus diagnostic criteria for autoimmune pancreatitis: guidelines of the International Association of Pancreatology. Pancreas 2011;40:352-8. 9. Kino-Ohsaki J, et al. Serum antibodies to carbonic anhydrase I and II in patients with idiopathic chronic pancreatitis and Sjögren´s syndrome. Gastroenterology 1996;110:1579-1586. 10. Okazaki K, et al. Autoimmune-related pancreatitis is associated with autoantibodies and a Th1/Th2-type cellular immune response. Gastroenterology 2000;118:573-81. 11. Asada M, et al. Identification of a novel autoantibody against pancreatic secretory trypsin inhibitor in patients with autoimmune pancreatitis. Pancreas 2006;33:20-6. 12. Sanchez Castanon M, Zuliani V, Amodio A, et al. Role of Amylase-alpha2A Autoantibodies in the Diagnosis of Autoimmune Pancreatitis. Pancreas 2015;44:1078-82. 13. Felix K, Hauck O, Schnölzer M, et al. Identification of Novel Serum Autoantibodies for Differential Diagnosis of Autoimmune Pancreatitis and Pancreatic Ductal Adenocarcinoma. Pancreas 2016;45:1309-19. 14. Vujasinovic M, et al. Kidney Involvement in Patients with Type 1 Autoimmune Pancreatitis. J Clin Med 2019;8. 15. Detlef-

sen S, et al. Diagnosis of autoimmune pancreatitis by core needle biopsy: application of six microscopic criteria. Virchows Arch 2009;454:531-9.

Disclosure:
Nothing to disclose

Current treatment, side effects and long-term results

Jeanin E. van Hooft, Netherlands

Learning Objectives:
- When to start and how to guide first-line therapy with steroids
- Being familiar with maintenance treatment with low-dose steroids, immunomodulators and rituximab
- Monitoring the treatment and anticipating side effects

Abstract:
The first-line, standard-of-care approach for most patients diagnosed with autoimmune pancreatitis is steroid treatment. The usually recommended dosage for remission induction is 0.6 -1.0 mg/kg per day, resulting in daily starting doses of 30 to 40 mg. One should be aware that particular the high dose is potentially associated with significant adverse effects especially in the AIP population with advanced age and high incidence of (de novo) diabetes mellitus. Recent data suggest that a lower daily dose of 20 mg is as effective. In general, clinical improvement after the start of steroid therapy is rapid, and follow-up serological and/or radiological assessment should be done in 2 weeks after treatment initiation. A swift response to steroids provides further diagnostic confirmation. A poor response, however, should raise the possibility of other diagnoses, particularly cancer.
The aim of maintenance therapyis to reduce the incidence of relapses. However, up to 70% of patients will never experience a relapse. Therefor introducing maintenance therapy only after a first disease relapse has been advocated to avoid overtreatment. Drugs available for maintenance are: low-dose steroids, immunomodulators and rituximab. In western countries, the use of immunosuppressants is generally preferred to avoid the adverse events of the long-term use of steroids. The most studied of these drugs is azathioprine, which is generally administered at a dose of 2-2.5 mg/kg. Azathioprine maintains a vast majority (75-85%) of treated patients in sustained remission at 3 years. In eastern countries, a low-dose (±5 mg/per day) maintenance therapy with steroids is proposed based on a Japanese randomized controlled trial revealing a significant longer relapse free survival. The third therapeutic option, which is off-label for autoimmune pancreatitis, is rituximab. Recently data suggested rituximab treatment to be considered for patients with difficult-to-treat relapsing disease.

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Disclosure:
Nothing to disclose

Acute liver failure: An era of expectation

11:00-13:00 / C2

Decisions, decisions and so little time to think!

John O'Grady, Ireland

Learning Objectives:
- To understand the heterogeneity of acute liver failure
- To understand the assessment of prognosis and determination of the optimal management plan
- To understand the role of liver transplantation in the management of acute liver failure

Abstract:
The modern management of acute liver failure delivers survival rates of around 80% when all available treatment options are optimally deployed. Achieving this threshold is reliant on rapid and accurate decision making from the point of initial presentation. Initially, patients must be characterised in terms of aetiology and sub-classification of acute liver failure, leading to an early assessment of prognosis. A key decision relates to listing for liver transplantation and confirmation of suitability once an organ becomes available.

References:
General: Bernal W, Wendon J. Acute liver failure. NEJM 2013:369:2525-34.
O'Grady JG, Schalm SW, Williams R. Acute liver failure: redefining the syndromes. Lancet 1993;342:273-5.
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Larson FS, Schmidt LE, Beirnsmeier C et al. High volume plasma exchange in patients with acute liver failure: an open randomised controlled trial. J Hepatol 2016;64:69-78.
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Maloney PR, Mallory GW, Atkinson JLD, et al. Intracranial pressure monitoring in acute liver failure: institutional case series. Neurocritical Care 2016;29:86-93.
O'Grady JG, Alexander GJM, Hallyar KM, Williams R. Early indicators of prognosis in acute liver failure. Gastroenterology 1989;97:439-445.
O'Grady JG. Timing and benefit of liver transplantation in acute liver failure. J Hepatol 2014;60:663-70.

Disclosure:
Nothing to disclose

Extracorporeal liver support: Evidence, current and future strategies

Faouzi Saliba, France

Learning Objectives:
- When to transfer a patient with ALI-ALF to a transplant center
- Place and timing of artificial liver support in patients with ALF
- Outcome of patients with and without transplantation

Abstract:
Well-established criteria for LTx (the Clichy-Villejuif and King's College criteria have been validated for ALF patients. Three main large controlled randomized trial (RCT) evaluated extracorporeal liver support in this indication:
1. The HepatAssist®system (a hollow fibre bioreactor embedded with 7 billion porcine hepatocytes). 171 patients (147 patients with ALF and 24 others with primary graft non-function) were randomized to device treatment or control. No significant difference between the control and the treatment arms for 30-day survival (62% vs 71%; p=NS).
2. Larsen F et al. conducted a RCT in 182 ALF patients who received either standard medical therapy (SMT; 90 patients) or SMT plus High Volume Plasma Exchange (HVPE) for 3 days (92 patients). Hospital survival was 58.7% for patients treated with HVPE vs. 47.8% for the control group (HR: 0.56; 95%CI, 0.36-0.86; p = 0.0083). HVPE prior to LTx did not improve survival compared with patients who received SMT alone (CI 0.37-3.98; p = 0.75).

In this context, the EASL 2017 guidelines reported:
Liver support systems (biological or adsorbent) should only be used in the context of RCT. HVPE in RCT has been shown to improve transplant-free survival in patients with ALF, and to modulate immune dysfunction. HVPE may be of greater benefit in patients who are treated early and who will not ultimately undergo LTx.
3. A RCT in France compared albumin dialysis with MARS®, initiated within 12 hours of randomization, to conventional medical treatment in 110 patients with ALF. 75% of the study patients underwent transplantation within 24 hours. Six-month survival was 75.5% (95% CI, 60.8-86.2%) with conventional treatment and 84.9% (95% CI, 71.9-92.8%) with MARS® (p=0.28). Access to LT was significantly higher in the MARS group.
Conclusion: HVPE is a safe technique that improved transplant free survival and allows a bridge to LT. MARS® is a safe tool that could be used as a bridge to LT.

References:
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Disclosure:
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Peptic ulcer disease
11:00-13:00 / C3

The "normal" peptic ulcer
Madalina Ilie, Romania

Learning Objectives:
- To learn about the epidemiology and risk factors of peptic ulcer disease
- To learn about diagnosis and treatment of Helicobacter pylori infection
- To learn about the diagnosis and management of uncomplicated peptic ulcer disease

Abstract:
Peptic ulcer is defined as defect in the gastroduodenal mucosa that extends through the muscularis mucosa secondary to pepsin and gastric acid secretion. It usually occurs in the stomach and proximal duodenum and less often in the lower esophagus (Cameron ulcers), distal duodenum or jejunum (in Zollinger-Ellison syndrome). The prevalence of peptic ulcer disease (PUD) has been decreasing lately reflecting the change in risk factors rather than genetic factors. PUD is associated with 2 main risk factors: Helicobacter pylori (H. pylori) infection and use of nonsteroidal anti-inflammatory drugs (NSAIDs). The risk is even higher when these are associated with smoking, advanced age, comorbidities and co-therapy with corticosteroids, anticoagulants, and bisphosphonates. PUD may be asymptomatic, may present with dyspeptic symptoms or complications like bleeding or perforation. The diagnosis is suspected in patients with symptoms and risk factors and is established by upper endoscopy. Currently, the strategy "test-and-treat" for Helicobacter pylori is appropriate in patients younger than 55 years, in the absence of alarm symptoms. Most other patients require endoscopy. If the patient is confirmed to be both H. pylori and NSAIDs negative, biopsies from the ulcer and the surrounding mucosa should be obtained to exclude underlying malignancy/lymphoma, Crohn´s disease or unusual infectious agents. If the etiology of the ulcer remains unexplained, an underlying gastrinoma should be considered. Gastric and duodenal ulcers which are benign have smooth, regular edges, with a flat, smooth ulcer base often filled with exudate. Endoscopic features that suggest a malignant ulcer include: ulcerated mass protruding into the lumen, nodular, clubbed, fused folds surrounding the ulcer crater, irregular or thickened ulcer margins. All ulcers with malignant features should be biopsied. The mainstay of PUD management is represented by eradicating H. pylori and antisecretory therapy.

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Disclosure:
Nothing to disclose

The “complicated” peptic ulcer

Peter Bauerfeind, Switzerland

Learning Objectives:

- Epidemiology and etiology of peptic ulcer complications
- Treatment of peptic ulcer complications
- Prevention of rtecurrence of peptic ulcer complications

Abstract:

Ulcer complications are: bleeding, perforation, penetration and duodenal outlet obstruction. Most frequent is bleeding (~70%) followed by perforation (~10%) and outlet obstruction (~3%). I.v.proton pump inhibitors (PPI) should be given in all cases. Standard dose (esomeprazole or pantoprazole) is 80 mg bolus followed by 8 mg/h for 72 h. High doses of orally given PPI should be used as soon as oral intake is possible. Sensitivity of all biopsy based H.p. tests, urea breath test and of stool test is reduced in complications. Nevertheless, H.p. test at the first endoscopy is useful, since specificity is high. In case of a negative test, serology is useful (not affected during PUD complication). In H.p.-positive patients treatment should be started as soon as possible. Key diagnostic instrument is early endoscopy. 250 mg Erythromycin i.v. is recommended 2 h before endoscopy. Routine use of nasogastric or orogastric aspiration/lavage or the use of somatostatin i.v. is not recommended. Endoscopic treatment of PUD complication should be based on the Forrest (F) classification. FIa (active spurting) and FIb (active oozing) should be treated by combining epinephrine injection with a second hemostasis modality (contact thermal, mechanical therapy, or injection of a sclerosing agent. In case of an adherent clot (FIIb), clot removal followed by endoscopic hemostasis is recommended. A routine second look is recommended only in high-risk patients or high-risk ulcer. If hemostasis is not achieved or recurrent re-bleeding following the second attempt, an endoscopic salvage therapy with topical hemostatic spray or over-the-scope clip should be considered. If this fails, transcatheter angiographic embolization (TAE) or surgery is the next step. H.p. eradication and avoidance of NSAIDs is necessary to prevent recurrence. Patients with no H.p. need anti-secretory therapy when NSAID is continued (PPIs).

References:

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Disclosure:

Nothing to disclose

Proctology: Solutions for daily problems

11:00-13:00 / F1

Hemorrhoids, pilonidal sinus, fissures and warts

Dieter Hahnloser, Switzerland

Learning Objectives:

- Recognize from anal symptoms the most probable proctological diagnosis
- Initial management of pilonidal sinus, hemorrhoids, fissures and warts
- When is surgery for obstructed defecation syndrome needed?

Abstract:

Symptomatic hemorrhoids can bleed + protrude and cause itching, soiling. Grade I-II hemorrhoids are treated conservatively (stool softeners, rubber band ligation;RBL). Grade III-IV hemorrhoids require surgery. Recurrence @1year after hemorrhoidal arterial ligation was 30% and 49% after RBL (HubBLe trial). Traditional excisional hemorrhoidectomy compared to stapled hemorrhoidopexy demonstrated @24month better QoL, less symptoms, less re-intervention and was cost-effective (eTHoS tiral).

Constipation, pain at defecation and bleeding are suspicious for acute anal fissure. Stool softeners and local application of calcium antagonist ointment heal >90%.Chronic fissures are characterized by a skin tag, hypertroph anal papilla, inverted borders and/or a visible sphincter. Chronic fissures need surgical treatment with fissurcetomy or V-Y advancement flap. Lateral internal sphincterotomy should be avoided.

Pilonidal sinus cause pain, secretion and recurrent infections. Treatment should be definitive with fast return to normal physical activity. We recommend outpatient minimal invasive sinusectomy in local anesthesia. Wounds heal in 5w, after 1w return to work and 7.5% recurrence. Recurrent or extensive disease should be treated by wide excision and primary closure (Limberg flap; 3.1% recurrence).

Warts are related with anal intraepithelial neoplasia (AIN, =premalignant). The incidence of AIN and anal cancer is rising, especially in the high-risk population. Warts should be classified into low or high grade AIN if present. Ablative treatment includes excision, fulguration, and laser therapy. Significant controversy regarding screening, surveillance and vaccination exists.

Patients complaining of incomplete or unsuccessful defecation requiring digitation, prolonged episodes on the toilet could present obstructed defecation syndrome (ODS). Symptoms correlated with anatomical changes. Laparoscopic ventral mesh rectopexy has replaced transanal/-vaginal procedures.

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- Haemorrhoidal artery ligation versus rubber band ligation for the management of symptomatic second-degree and third-degree haemorrhoids (HubBLe): a multicentre, open-label, randomised controlled trial. Brown SR, Tiernan JP, Watson AJM, Biggs K, Shephard N, Wailoo AJ, Bradburn M, Alshreef A, Hind D; HubBLe Study team. Lancet. 2016 Jul 23;388(10042):356-364 Comparison of stapled haemorrhoidopexy with traditional excisional surgery for haemorrhoidal disease (eTHoS): a pragmatic, multicentre, randomised controlled trial. Watson AJ, Hudson J, Wood J, Kilonzo M, Brown SR, McDonald A, Norrie J, Bruhn H, Cook JA; eTHoS study group.Lancet. 2016 Nov 12;388(10058):2375-2385. Long-term follow-up after combined fissurectomy and Botox injection for chronic anal fissures. Scholz T, Hetzer FH, Dindo D, Demartines N, Clavien PA, Hahnloser D. Int J Colorectal Dis. 2007 Sep;22(9):1077-81. A systematic review and meta-analysis of the treatment of anal fissure. Nelson RL, Manuel D, Gumienny C, Spencer B, Patel K, Schmitt K, Castillo D, Bravo A, Yeboah-Sampong A. Tech Coloproctol. 2017 Aug;21(8):605-625. Sinusectomy for primary pilonidal sinus: less is more. Soll C, Dindo D, Steinemann D, Hauffe T, Clavien PA, Hahnloser D. Surgery. 2011 Nov;150(5):996-1001. Long-term follow-up for pilonidal sinus surgery: A review of literature with metanalysis. Milone M, Velotti N, Manigrasso M, Anoldo P, Milone F, De Palma GD. Surgeon. 2018 Oct;16(5):315-320. - Synthetic Versus Biological Mesh-Related Erosion After Laparoscopic Ventral Mesh Rectopexy: A Systematic Review. Balla A, Quaresima S, Smolarek S, Shalaby M, Missori G, Sileri P. Ann Coloproctol. 2017 Apr;33(2):46-51. doi: 10.3393/ac.2017.33.2.46. Epub 2017 Apr 28. Review. Current status of laparoscopic and robotic ventral mesh rectopexy for external and internal rectal prolapse. van Iersel JJ, Paulides TJ, Verheijen PM, Lumley JW, Broeders IA, Consten EC. World J Gastroenterol. 2016 Jun 7;22(21):4977-87. Anal intraepithelial neoplasia: diagnosis, screening, and treatment. Siddharthan RV, Lanciault C, Tsikitis VL. Ann Gastroenterol. 2019 May-Jun;32(3):257-263.

Practice parameters for the treatment of colonic diverticular disease: Italian Society of Colon and Rectal Surgery (SICCR) guidelines.Binda GA, Cuomo R, Laghi A, Nascimbeni R, Serventi A, Bellini D, Gervaz P, Annibale B; Italian Society of Colon and Rectal Surgery. Tech Coloproctol. 2015 Oct;19(10):615-26.

Disclosure:
Nothing to disclose

All you need to know about anal fistula

Not available

Oropharyngeal swallowing disorders: Why gastroenterologists should be interested!

11:00-13:00 / F2

Causes and diagnosis of oropharyngeal dysphagia: An update

Nathalie Rommel, Belgium

Learning Objectives:

- To understand the causes of oropharyngeal dysphagia in a variety of adult patients
- To understand the spectrum diagnostic instruments to assess of oropharyngeal dysphagia in adult patients
- To understand some of the pitfalls in current clinical diagnostic practise in the care of patients with dysphagia

Abstract:
Dysphagia of varying severity is common, hinders the provision of adequate nutrition and is associated with a wide range of morbidities[1,2]. Before treatment and management of dysphagia can commence, there is a need for a thorough assessment of swallowing function. Many different functional tests are available to assess oropharyngeal function, each with advantages and disadvantages[3,4]. Patients with dysphagia are typically investigated by a videofluoroscopic swallow study[5,6]. Although helpful clinically, the limitations of the technique are well known and relate to radiological exposure and the lack of measurable objective parameters. Currently the outcomes of a videofluoroscopic swallow study are largely descriptive. To make a videofluoroscopy diagnostically more useful, it can be combined with high resolution manometry impedance (HRM(I)), to assess pressure changes in the pharynx and upper oesophageal sphincter in relation to bolus flow during swallowing. This represents a significant advance over a standard videofluoroscopy because it (1) allows quantification of the pharyngeal movements in relation to bolus passage and to the opening of the upper esophageal sphincter and (2) allows determination of the pathological basis for swallowing disorders across the pharyngo-oesophageal segment. High resolution manometry (HRM) has revolutionalized the assessment of esophageal motor function , and has brought significant advances to understand esophageal motility disorders in adult[7-11] and pediatric patients[12,13]. HRM(I) is now increasingly applied to evaluate contractile function across the length of the pharynx and upper esophageal sphincter. The clinical interpretation of HRM(I) metrics for the diagnosis of pharyngeal motility disorders has recently been formalized in a core outcome set of metrics[14]. The advanced role of these functional tests will be discussed and illustrated with examples from daily clinical practice.

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Disclosure:
NR holds IP on AIM technology. No financial disclosures.

Treatment of oropharyngeal dysphagia in 2019

Philip Woodland, United Kingdom

Learning Objectives:

- To understand how investigations into oropharyngeal dysphagia can direct management decisions
- To understand treatment options (including allied professional therapy) for oropharyngeal dysphagia
- To understand endoscopic therapeutic options in oropharyngeal dysphagia

Abstract:

Treatment of oropharyngeal dysphagia is often an area treated with less confidence by gastroenterologists when compared to treatment of oesophageal dysphagia. This talk discusses how to approach treatment of oropharyngeal dysphagia. Discussed topics include the role of speech and language therapy, and the role of transcranial stimulation. The role of endoscopy in post-surgical/radiotherapy hypopharyngeal strictures, and for Zenker diverticulum will be addressed.

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Disclosure:

I receive consulting fees from Reckitt Benckiser UK and Dr Falk Pharma UK.

Evaluation and management of patients with alcohol-related liver disease: A multidisciplinary approach

11:00-13:00 / F3

Diagnosis and the role of histology in the management of patients with alcohol-related liver disease

Karoline Lackner, Austria

Learning Objectives:

- Types of alcohol-related liver disease (ALD)
- Clinical diagnosis of ALD
- Role of histology in the management of patients with ALD

Abstract:

The cornerstone of the clinical diagnosis of ALD is the documentation of regular alcohol overuse with a threshold of >20g/d for women and >30g/d for men and biochemical and/or radiological signs of liver injury [1, 2]. Most patients with ALD present at a late stage with advanced liver disease and severe symptoms related to liver failure either caused by acute decompensation of cirrhosis or as acute-on chronic liver failure (ACLF) often due to active drinking and severe alcoholic hepatitis. In the latter group the clinical diagnosis of ALD is often straight forward. However, the identification of the exact cause precipitating hepatic decompensation is important and guides decision making including treatment with corticosteroids and/or antibiotics. Therefore, liver histology can be very useful to confirm the clinical diagnosis and exclude other liver diseases which may be present in 10-20% of cases [2]. In addition, liver histology can provide important prognostic information and predict sepsis [3-5].
In the majority of individuals with alcohol overuse ALD follows an oligosymptomatic course with few or even no symptoms also despite the presence of severe liver injury. Advanced fibrosis may be found in up to 50% of patients [6]. Early detection of ALD and measures to achieve abstinence are pivotal to prevent disease progression, development of cirrhosis and its complications. Therefore, if alcohol overuse has been detected, screening for ALD is warranted even in the absence of symptomatic disease. This also applies for high-risk populations including patients of alcohol rehabilitation clinics, or for patients with extrahepatic manifestations of alcohol use disorders like injuries, cardiovascular diseases, pancreatitis, peripheral neuropathy or others. Also in patients with clinical suspicion of another or concomitant cause(s) of liver injury, histology plays an important role in the classification, staging as well as prognostication [2].

References:

[1] Seitz H K, Bataller R, Cortez-Pinto H, Gao B, Gual A, Lackner C et al. Alcoholic liver disease. Nat Rev Dis Primers 2018; 4:16-018-0014-7. [2] European Association for the Study of the Liver. Electronic address: easloffice@easloffice.eu, European Association for the Study of the Liver. EASL Clinical Practice Guidelines: Management of alcohol-related liver disease. J Hepatol 2018. [3] Spahr L, Rubbia-Brandt L, Genevay M, Hadengue A, Giostra E. Early liver biopsy, intraparenchymal cholestasis, and prognosis in patients with alcoholic steatohepatitis. BMC Gastroenterol 2011; 11:115. [4] Katoonizadeh A, Laleman W, Verslype C, Wilmer A, Maleux G, Roskams T et al. Early features of acute-on-chronic alcoholic liver failure: a prospective cohort study. Gut 2010; 59:1561-1569. [5] Mookerjee R P, Lackner C, Stauber R, Stadlbauer V, Deheragoda M, Aigelsreiter A et al. The role of liver biopsy in the diagnosis and prognosis of patients with acute deterioration of alcoholic cirrhosis. J Hepatol 2011; 55:1103-1111. [6] Lackner C, Spindelboeck W, Haybaeck J, Douschan P, Rainer F, Terracciano L et al. Histological parameters and alcohol abstinence determine long-term prognosis in patients with alcoholic liver disease. J Hepatol 2017; 66:610-618.

Disclosure:

Consultant histopathologist for Galmed Pharamceutical Ltd, Dr Falk Pharma GmbH, Allergan, and Histoindex

Burden of disease: The importance of early diagnosis and correct management

Helena Cortez-Pinto, Portugal

Learning Objectives:

- Learn what is presently the burden of ALD in Europe and the World
- How to diagnose ALD in earlier stages, and what are the best prognosticators
- How to manage ALD in an earlier stage and alcoholic hepatitis

Abstract:

Alcohol related liver disease (ALD) is the most frequent liver disease, responsible for 50% of cases of cirrhosis worldwide, according to WHO. Furthermore, ALD is now the most common indication for LT in US and Europe. It is also increasingly recognized the importance of overlap between harmful alcohol consumption, and metabolic factors in the risk of developing disease.

Although so frequent, ALD is diagnosed mostly in advanced stages. In a large multicontinental cohort of patients with either early or advanced liver disease, in the early disease group only 3.8% had ALD, contrasting with 29% of patients seen in an advanced stage. This late diagnosis is very detrimental for ALD patients.

However, there are good opportunities for ALD earlier diagnosis, including acute medical admissions, screening of patients in alcohol detoxification clinics and previous hospital admissions. Nonetheless, identification of harmful drinkers and early phases of liver disease will only be effective if measures to curtail alcohol consumption are implemented, and management of liver disease is initiated.

The use of noninvasive tests to detect earlier stages of ALD is attractive. In suspected ALD, doing blood tests, and ultrasound followed by liver stiffness measurement, enables the assessment of fibrosis stage in about 95% of patients. This may improve early recognition and follow-up of patients with ALD.In the setting of alcoholic hepatitis, several algorithms are used, such as MDF or MELD. Combining scores seems better for predicting outcome. New biomarkers, such as cytokeratin's and MicroRNA will be discussed.

ALD patients should be managed by a multidisciplinary team, including addiction therapists. Motivational interviewing and pharmacological agents to treat alcohol use dependence will be discussed. Besides from prednisolone, other treatments for alcoholic hepatitis are being investigated, such as IL-22 or GCSF. Liver transplantation in this setting is also increasingly recognized.

References:

Reviews; Seitz HK, Bataller R, Cortez-Pinto H, et al. Publisher Correction: Alcoholic liver disease. Nat Rev Dis Primers 2018;4:18. European Association for the Study of the Liver. Electronic address eee, European Association for the Study of the L. EASL Clinical Practice Guidelines: Management of alcohol-related liver disease. J Hepatol 2018. Key papers: 1. WHO. Global status report on alcohol and health 2018. Geneva: WHO; 2018. 2. Pimpin L, Cortez-Pinto H, Negro F, et al. Burden of liver disease in Europe: epidemiology and analysis of risk factors to identify prevention policies. J Hepatol 2018. 3. Cholankeril G, Ahmed A. Alcoholic Liver Disease Replaces Hepatitis C Virus Infection as the Leading Indication for Liver Transplantation in the United States. Clin Gastroenterol Hepatol 2018;16:1356-8. 4. Haldar D, Kern B, Hodson J, et al. Outcomes of liver transplantation for non-alcoholic steatohepatitis: A European Liver Transplant Registry study. J Hepatol 2019;71:313-22. 5. Eslam M, Sanyal AJ, George J. Toward More Accurate Nomenclature for Fatty Liver Diseases. Gastroenterology 2019;157:590-3. 6. Shah ND, Ventura-Cots M, Abraldes JG, et al. Alcohol-Related Liver Disease Is Rarely Detected at Early Stages Compared With Liver Diseases of Other Etiologies Worldwide. Clin Gastroenterol Hepatol 2019. 7. Atkinson SR, Vergis N, Louvet A, Thursz MR. Universal screening of acute medical admissions for excess alcohol consumption: What´s the misuse? J Hepatol 2017;67:448-50. 8. Moreno C, Mueller S, Szabo G. Non-invasive diagnosis and biomarkers in alcohol-related liver disease. J Hepatol 2019;70:273-83. 9. Gao B, Shah VH. Combination therapy: New hope for alcoholic hepatitis? Clin Res Hepatol Gastroenterol 2015;39 Suppl 1:S7-S11. 10. Szabo G, Kamath PS, Shah VH, Thursz M, Mathurin P, Meeting E-AJ. Alcohol-Related Liver Disease: Areas of Consensus, Unmet Needs and Opportunities for Further Study. Hepatology 2019;69:2271-83.

Disclosure:

Gilead Science// Lectures and advisory board fees Intercept // Lectures and advisory board fees

Complications of liver cirrhosis

14:00-16:00 / Hall 6

Patients with difficult to treat ascites and large varices

Guadalupe Garcia-Tsao, United States

Learning Objectives:

- Identify measures to prevent first variceal hemorrhage in patients with large varices, with or without ascites
- Recognize that non-selective beta-blockers, by decreasing portal pressure, reduce the risk of variceal hemorrhage and other complications of cirrhosis
- Identify settings in which NSBB should be used cautiously in patients with ascites

Abstract:

In patients with cirrhosis and large varices, recommendations to prevent first variceal hemorrhage are non-selective beta-blockers (NSBB: propranolol or nadolol) or carvedilol or endoscopic variceal ligation (EVL) (1). However, it has been suggested that NSBB may be deleterious in patients with difficult-to-treat ascites (2). NSBB have been shown to prevent not only first but also recurrent variceal hemorrhage in patients with cirrhosis. Furthermore, a recent meta-analysis showed that patients with cirrhosis, both with and without ascites, who are hemodynamic responders to NSBB (i.e. reduce portal pressure), not only have a reduced risk of relevant events but also have a lower risk of death/transplant (3) . On the other hand, the main pathophysiologic mechanism in patients with cirrhosis and ascites is splanchnic and systemic vasodilatation that leads to activation of neurohumoral systems, sodium and fluid retention, resulting in increased cardiac output, and a hyperdynamic circulatory state. In patients with difficult to treat ascites, these abnormalities are maximal and a relative decrease in cardiac output due to NSBB can lead to a decrease in renal perfusion and to renal dysfunction (4). An observational study including 151 patients first suggested that mortality was higher in patients with refractory ascites on NSBB (5). The most recent meta-analysis including this and several additional observational studies, totaling 1,397 patients, concluded that NSBB have neither a beneficial or a deleterious effect in patients with cirrhosis and ascites (6). Other studies have suggested that the deleterious effect of NSBB in patients with ascites occur with higher doses of NSBB (2, 7) and when NSBB are associated with a significant decrease in arterial blood pressure (2, 8). Given the benefit of NSBB, particularly in the setting of secondary prophylaxis of variceal hemorrhage where patients with ascites have a significantly better survival with combination EVL + NSBB compared to EVL alone (9), guidelines recommend that NSBB should not be avoided in patients with cirrhosis and ascites but that they should be used cautiously, starting at lower doses with dose/reduction or discontinuation if systolic blood pressure decreases to < 90 mmHg, serum sodium < 130 mEq/L or development of acute kidney injury (1, 10).

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Nothing to disclose

Patients with alcoholic cirrhosis presenting with confusion

Debbie Shawcross, United Kingdom

Learning Objectives:
- To consider the wide differential of causes of brain dysfunction in a patient with alcohol-related cirrhosis presenting with confusion
- To focus specifically on the diagnosis and management of acute hepatic encephalopathy
- To consider the prognosis and strategies to prevent re-admission

Abstract:
Patients presenting with confusion in the context of alcohol-related cirrhosis are challenging to manage especially if acute alcohol withdrawal is present. The differential diagnosis is wide and presents a complex conundrum of issues. Hepatic encephalopathy (HE) is a clinical diagnosis of exclusion when other causes of confusion have been excluded which include intracranial haemorrhage, acute alcohol withdrawal, electrolyte disturbance, sepsis and drug intoxication. Assessment for the presence of a precipitating factor such as gastrointestinal bleeding or infection must be sought. Evaluation and simultaneous management of the airway should be performed at the outset. Blood biochemistry, glucose and a septic screen should be undertaken including a diagnostic ascitic tap. An elevated arterial ammonia concentration can help to confirm the diagnosis of HE; however, a normal or mildly elevated blood ammonia does not exclude a diagnosis of HE. Computed tomography of the head is often required to exclude an alternative cause of altered consciousness. The staging of overt HE with the West Haven criteria remains an imprecise art that is often hampered by its fluctuant course. The development of HE is often unpredictable and its management, particularly in a ward environment, remains challenging. Patients frequently require augmented levels of care in a high-dependency or intensive care area. The overarching priority in any patient presenting with HE is to actively seek out and treat any causative factor(s). A low threshold for antibiotic use should be adopted. Reversal of dehydration and correction of hyponatraemia are important. Enemas often rapidly improve conscious level. Evidence for giving lactulose in acute HE is limited; on discharge it should be used with the aim of producing two bowel movements/day. Treatment with rifaximin and lactulose maintains remission from HE more effectively than lactulose alone and significantly reduced the risk of hospitalization with HE.

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Patients with compensated or decompensated cirrhosis: How to assess (mal)nutrition?

Manuela Merli, Italy

Learning Objectives:
- Understand the relevance of nutritional status in chronic liver diseases
- Recognize sarcopenia in patients with liver cirrhosis
- Comprehend the interplay between muscle depletion, frailty and functional disability in advanced liver failure

Abstract:
Nutritional alterations are not only a bystander in chronic liver disease. Both undernutrition or overnutrition may influence the patient's outcome. Malnutrition has been associated with hepatic encephalopathy, bacterial infections, hyponatremia, and recurrent/refractory ascites.
In the early stages of liver disease malnutrition may be underrated. Clinical suspicious and recognition of patients at risk of malnutrition are mandatory. Muscle depletion (sarcopenia) is a relevant component of malnutrition in patients with liver cirrhosis. Sarcopenia can be evaluated through CT-scan, DEXA (dual energy X-ray absorptiometry), BIA or be estimated through simple anthropometry measurements. Survival is worse in sarcopenic patients vs non-sarcopenic patients independently from the severity of liver insufficiency. The concept of muscle mass cannot be separated from that of muscle performance. These two parameters are not always correlated. The estimate of muscle function is possible through the handgrip test and the Physical Performance Battery. A well-preserved muscle performance has been found to be highly predictive of survival while deterioration of muscle function contributes to frailty and daily living disability and is associated with a worse patient's outcome. Nutritional assessment in a patient with liver cirrhosis is a prerequisite to start adequate nutritional counseling and nutritional therapy.

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Patients with compensated or decompensated cirrhosis: How to manage (mal)nutrition?

Mathias Plauth, Germany

Learning Objectives:

- To know recommended energy and protein intake
- To know the beneficial effect of nocturnal feeding oral nutrition supplements (ONS)
- To know the benefit of enteral tube feeding

Abstract:

Many malnourished cirrhotic patients are anorexic and cannot meet their nutrient requirements by oral intake "ad lib". In patients with clinically stable LC, an intake of 1.3 x REE or 30-35kcal kg^{-1} d^{-1} total energy including 1.2g kg^{-1} d^{-1} of protein is recommended for maintaining body composition and protein status. If the patient cannot eat such a quantity of food, then standard ONS offer the opportunity to provide the patient with additional energy and protein. In order to make full use of the protein consumed, patients should be counseled to drink ONS late in the evening and at night because nocturnal feeding has been shown to result in greater accretion of total body protein than daytime ONS. In malnourished LC patients requiring repletion, more protein (1.5g kg^{-1} d^{-1}) should be given. In such patients, enteral feeding of a high density standard formula via nasogastric tube is recommended. Tube feeding has been shown to improve liver function, mental state and survival without an undue risk of variceal bleeding. In these patients, low grade HE (I-II°) is not a contraindication to an adequate protein supply. As hepatic glycogen stores are depleted cirrhotics who can be fed sufficiently either by the oral or enteral route but who have to abstain from food temporarily (including nocturnal fasting!) for more than 12 hours, should be given hypocaloric parenteral nutrition (PN) or i.v. glucose. When this fasting period lasts longer than 72 h total PN is required.
Adequate nutrition per se counteracts HE and even transient protein restriction is not beneficial. In proven protein-intolerant patients, oral supplementation with branched-chain amino acids (BCAA) may be helpful in achieving an adequate nitrogen intake. PN is only indicated, when oral or enteral nutrition are not possible. Patients in coma (HE III-IV°) can safely be given total PN regimens providing 30-35kcal kg^{-1} d^{-1} total energy including 1.0g kg^{-1} d^{-1} using BCAA-enriched amino acid solutions.

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Patients with a new diagnosis of portal vein thrombosis: Anticoagulate? Yes!

Andrea De Gottardi, Switzerland

Learning Objectives:

- To understand the reasons why cirrhotic patients are at risk for the development of portal vein thrombosis
- To know the incidence of portal vein thrombosis and assess the clinical consequences of PVT in this group of patients
- To identify patients in whom anticoagulation may be beneficial and to know what anticoagulants are available

Abstract:

In patients with Child-Pugh A or B cirrhosis, incidence of newly diagnosed PVT after one and five years has been reported to be 4.6% and 10.7%, respectively. PVT is frequently detected in advanced stages, increasing up to 25% in liver transplant candidates and 35% in cirrhotic patients with hepatocellular carcinoma. Prognosis and treatment of PVT depend on the localization, the degree of extension, and the rapidity of development, as well as risk factors for thrombosis and the stage of advanced chronic liver disease. Risk factors for PVT include Child C stage, prior history of resolved PVT, associated pro-thrombotic risk factors, hepatocellular carcinoma, recent abdominal surgical, endoscopic or invasive interventions and portal flow velocity < 15 cm/s. Symptoms may include abdominal pain, loss of appetite, nausea, vomiting, and diarrhea, gastrointestinal hemorrhage, fever, ascites. The main goal of treatment is control and/or prevention of complications of PVT, which are accomplished by restoring the portal blood flow and prevention of thrombus extension. Anticoagulation should be considered in potential liver transplant candidates with thrombosis of the main portal trunk or progressive PVT, with the goal to facilitate liver transplantation and reduce post-transplant morbidity and mortality. Heparin, low molecular weight heparins, as well as vitamin K antagonists are safe in this population. Current data on the use of direct oral anticoagulants suggest that these drugs are safe in Child A and B patients, while they should not be used in Child C patients or in case of renal failure. Their efficacy remains to be demonstrated in future studies.

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Patients with a new diagnosis of portal vein thrombosis: Anticoagulate? No!

Virginia Hernández-Gea, Spain

Learning Objectives:

- The natural history of portal vein thrombosis (PVT) is not well known, including whether PVT is the cause or the consequence of liver disease progression and it may resolve spontaneously
- PVT increases complexity of liver transplant and it is associated with worse outcome. Therefore achieving recanalization may be a mayor goal in liver transplant candidates, however in non-liver transplant candidates the benefit is not well established
- No randomized controlled clinical trial has compare anticoagulation versus placebo to accurately assess safety and adverse events

Abstract:

The natural history of PVT is not well known including whether PVT is the cause or the consequence of liver disease progression. Prevalence in highly variable among studies ranging from 2-23% and the annual incidence of PVT in patients with advanced cirrhosis may be 10%-15%. Anticoagulation is the considered first line therapy to achieve portal vein recanalization. However PVT may resolve spontaneously in approximately 40%-70% of cases, mainly when not occlusive and no predictive factors of spontaneous recanalization have been found so far. Importantly prospective controlled trials evaluating the effect of anticoagulation and properly considering spontaneous recanalization rate are lacking and available recanalization rates may be biased. Moreover the contribution of PVT to hepatic decompensation and overall mortality in cirrhosis is not well defined and the impact of recanalyzing PVT in development of portal hypertension related complications is not so clear. In addition no randomized controlled clinical trial has compare anticoagulation versus placebo to accurately assess whether bleeding risk is increased Based on all this data, more evidence generated by high quality studies is needed before recommending anticoagulation to all patients with acute PVT. Although still considered as a second line therapy due to the lack of robust evidence, TIPS is very effective achieving recanalization even in patients with fully occlusive PVT with recanalization rates ranging from 57% to 95% and maintain during follow up in more than 90% of the cases and should be also consider.
PVT increases complexity of liver transplantation surgery and even im-

pairs physiological reconstruction of the portal venous axis, increasing complications rates and decreasing post-liver transplantation survival. Achieving portal vein recanalization to allow end-to-end portal vein anastomosis during liver transplant is recommended in liver transplant candidates as it improves outcome.

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Disclosure:

Nothing to disclose

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