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M Birnie et al., J Gastrointest Dig Syst 2017, 7:6(Suppl)
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A 35-year-old patient with abdominal pain, mesenteric lymphadenopathy and Sweet's syndrome-an atypical presentation of Crohn's disease

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Crohn's disease is a form of inflammatory bowel disease that typically presents with abdominal pain, weight loss and bloody diarrhoea. Sweet's syndrome is a neutrophilic dermatosis which presents with erythematous papules and is associated with Crohn's disease amongst other conditions. We present an unusual case of Crohn's disease where abdominal pain, extensive mesenteric lymphadenopathy and Sweet's syndrome were the initial presenting features. We describe how diagnostic uncertainty led to extensive investigations and recurrent admissions. Initially the patient was treated as having Yersinia infection due to suggestive histology although serology was negative. Yersinia infection, gastrointestinal tuberculosis and Crohn's disease may have similar radiological and histological appearances so differentiation can be difficult. We discuss the impact of delayed diagnosis (seventeen months) and share some interesting points on the difficulties of diagnosing Crohn's disease on mesenteric lymph node histology alone, especially when macroscopic examination of the bowel wall is normal. Treatment for Crohn's disease resulted in improvement of her symptoms and return to work.

Biography

Birnie is currently a core medical trainee working for the North West deanery in Stockport, England. She studied medicine at the University of Lancaster, qualifying in 2015. Her current interest is acute medicine, in which she is hoping to specialise in this area.

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Dysregulation of KRAS signaling in pancreatic cancer is not associated with *KRAS* mutations and outcome

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Background: Pancreatic ductal adenocarcinoma (PDAC) is known as cancer with very poor prognosis. *KRAS* oncogene is a major driver of PDAC tumorigenesis but its role as prognostic or predictive factor is not clear. The aim of the present study was to investigate the prognostic significance of *KRAS* downstream signaling pathway genes expression and association with clinical characteristics in PDAC patients undergoing radical surgery.

Methods: Tumors and adjacent non-neoplastic pancreatic tissues were examined in 45 patients with histologically verified PDAC. *KRAS, BRAF* and *PIK3CA* gene mutation analysis was performed using the *KRAS/BRAF/PIK3CA* array. The transcript profile of 52 *KRAS* downstream signaling pathway genes was assessed using quantitative real-time polymerase chain reaction.

Results: *KRAS* mutation was detected in 80% of cases but the mutation status do not influence PDAC patient's prognoses. The genes of four signaling pathways downstream of *KRAS* including the PI3K/PDK1/AKT, RAL guanine nucleotide exchange factor, RIN1/ABL, and RAF/MAPK pathways exhibited differential expression in PDAC compared to the adjacent normal tissue. However, no significant differences in expression were evident between patients with KRAS mutated and wild type tumors. Moreover, expression patterns of *KRAS* downstream signaling pathway do not associate with overall survival of patients.

Conclusions: *KRAS* mutation is present in most cases of PDAC, but is not associated with changes in expression of *KRAS* downstream signaling pathways and clinical outcome.

Biography

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Final results of multi-center, prospective, controlled trial of the duodeno-jejunal bypass liner for the treatment of type 2 Diabetes mellitus in obese patients: Efficacy and factors predicting a suboptimal effect

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Introduction: The global increase in obesity incidence results in an increase of type 2 diabetes mellitus (T2DM). Surgical treatment has proven to be effective; however it carries a high risk of complications. The duodenal-jejunal bypass liner (EndoBarrier®, GI Dynamics, EB) is an endoscopic implant that mimics the intestinal bypass portion of the Roux-en-Y gastric bypass. It results in weight loss and improvements in glucose control in obese patients with T2 diabetes mellitus (T2DM).

Aims & Methods: This is a final report of a prospective, controlled, multicentre study aimed to determine the effectiveness of EB and to identify factors associated with a sub-optimal outcome of EB.

Results: 70 subjects (45 with an implant, 25 controls) were included in the study. The groups were comparable with respect to age, gender, BMI (mean 41.7 vs. 39.5 kg/m2), T2DM duration (7.8 vs. 8.3 years), HbA1c level (88 vs. 86 mmol/mol) and T2DM treatment. In the EB group, all devices were successfully implanted. Only six devices had to be explanted prior to the end of the 10 months study period (bleeding, dislocation and need for ERCP because of choledocholithiasis). The mean procedure time was 17 minutes for an implantation and 16 minutes for an explantation. At 10 months, there was significantly greater weight loss and %EWL (19% vs. 7% and 43 vs. 12) and significantly improved long term compensation of T2DM marker HbA1c (decreased by 25 vs. 10 mmol/mol) in the EB group. T2DM medicinal treatment could be reduced in more device subjects than controls. There was no serious adverse event. Mild abdominal pain and nausea after implantation were experienced by 60% of patients during first 14 days after implantation, 30% of patients during the first month and 10% of patients after one month. Lower initial BMI and lower body height were identified as negative prognostic factors for pain, but positive for efficacy of EB.

Conclusion: The EB is safe when implanted for 10 months, and results in significant weight loss and HbA1c reduction. This suggests that this novel device is a candidate for the primary therapy of morbid obesity and T2DM. Lower initial BMI and lower body height could be negative prognostic factor for pain, but positive for efficacy.

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Periodontal and inflammatory bowel diseases: Is there evidence for complex pathogenic interactions?

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Periodontal disease and inflammatory bowel disease (IBD) are both chronic inflammatory diseases and their pathogenesis is mediated by a complex interplay between a dysbiotic microbiota and the host immune-inflammatory response, influenced by genetic and environmental factors. This review aimed to provide an overview of the evidence dealing with a possible pathogenic interaction between periodontal disease and inflammatory bowel disease. The prevalence of periodontal disease seems to be increased in patients with IBD when compared to healthy controls, probably due to a changed oral microbiota and a higher inflammatory response. Moreover, the induction of periodontitis seems to result in gut dysbiosis and altered gut epithelial cell barrier function, which might contribute to the IBD pathogenesis. Considering the complexity of periodontal and inflammatory bowel diseases, and the coexistence of both, it is very challenging to comprehend the possible pathways involved in both diseases. In conclusion, this review points out to a complex pathogenic interaction between periodontal and inflammatory bowel diseases, in which one disease might alter the composition of the microbiota and increase the inflammatory response related to the other. However, we still need more data derived from human studies to confirm results from murine models. Thus, mechanistic studies are warranted to clarify this possible bidirectional association.

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Dichotomous effects of glucose versus fructose on hepatic lipogenesis, mitochondrial function and insulin signaling

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Non-alcoholic fatty liver disease (NAFLD) is a liver manifestation of obesity and insulin resistance. Overconsumption of high-fat diet and sugar-sweetened beverages are risk-factors for development of NAFLD. The relative effects of different monosaccharides on pathogenesis of NAFLD are not clearly established. We studied metabolic outcomes of mice fed chow or high-fat diet (HFD) consuming either regular water or water sweetened with 30% fructose or glucose, monosaccharide components of table sugar. Mice on HFD supplemented with fructose developed more significant obesity, glucose intolerance and hepatomegaly, as compared to glucose supplemented mice. Fructose supplemented mice had higher levels of SREBP1c and de novo lipogenesis, while glucose enhanced ChREBP transcription factor and fatty acid oxidation. Liver metabolomics profile confirmed that fructose enriched endogenously synthesized fatty acid pools, while RNA-sequence analysis identified lipogenesis and insulin signaling pathways as uniquely regulated by fructose versus glucose metabolism. The most striking difference was observed in mitochondrial structure and function. On HFD, fructose intake was associated with larger number, but smaller mitochondrial size, while ATP and NADH levels were decreased with acute fructose injection. Ketohexokinase C (KHK), the first enzyme of fructose metabolism, was increased in mice with fructose supplementation and in obese adolescents with nonalcoholic steatohepatitis. Knockdown of fructose metabolism specifically in the liver resulted in decreased hepatic steatosis, enhanced mitochondrial function and improved glucose tolerance. Our data show that fructose confers poor metabolic outcomes associated with sugar intake, while glucose metabolism may even be protective.

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Endoscopic range of Austrian surgical units – data of an anonymized survey among the 115 Austrian surgical units

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Introduction: The synergy of endoscopy and surgery becomes more and more important due to multimodal therapeutic approaches and the knowledge about the absolute necessity of a stage dependent tumor treatment. On the other hand, it becomes increasingly difficult to offer both endoscopy and surgery in one single department because of lacking personal and temporal resources.

Methods: A single-sided questionnaire is sent to all Austrian surgical units (n=115). In total seven questions must be answered. Four issues deal particularly with endosonography. After delivery an electronically mailed reminder asks for participation in the survey. The completed questionnaires are sent back to our study group by a prepaid envelope.

Results: The rate of return added up to 63.5% (n=73) and splitted in 67% standard hospitals, 25% specialized hospitals and 8% central hospitals. 94.5% of the Austrian surgical units offer gastroscopies and colonoscopies, but only 8.2% surgical departments (n=6) perform endosonographical examinations of the upper GI-tract. Furthermore, the survey showed that 89% (n=65) respectively 52% (n=38) of the Austrian surgical units do gastric and pancreatic oncological resections.

Conclusion: The results of our survey make clear that a rather high proportion of Austrian surgical departments run endoscopic units. On the other hand, specialized endoscopic techniques like endosonography are very uncommon in the surgical range of services. Because of multiple intersections between endoscopy and surgery as well in diagnostics as in treatment we support an intensified involvement of the Austrian Surgical Society concerning the implementation of specialized endoscopic procedures. The divergence of operative range and endosonographic possibilities is striking. For ensuring a tumor treatment adequate to its stage endosonography is obligatory and must be strongly demanded in the pretherapeutic diagnostic setting.

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Proton pump inhibitors for preventing non-steroidal anti-inflammatory drug induced gastrointestinal toxicity: A systematic review

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Objective: Proton pump inhibitors (PPIs) are recommended for preventing gastrointestinal lesions induced by non-steroidal anti-inflammatory drugs (NSAIDs). We performed this study: (1) to evaluate the effectiveness and safety of PPIs, (2) to explore the association between effectiveness and potential influential factors, and (3) to investigate the comparative effect of different PPIs.

Methods: MEDLINE, EMBASE, and the Cochrane Library were searched to identify randomized controlled trials comparing different classes of PPIs, or comparing PPIs with placebo, H2 receptor antagonists or misoprostol in NSAIDs users. Both pairwise meta-analysis and Bayesian network meta-analysis were performed.

Results: Analyses were based on 12,532 participants from 31 trials. PPIs were significantly more effective than placebo in reducing ulcer complications (relative risk [RR] ¼ 0.29; 95% confidence interval [CI], 0.20 to 0.42) and endoscopic peptic ulcers (RR ¼ 0.27; 95% CI, 0.22 to 0.33), with no subgroup differences according to class of NSAIDs, ulcer risk, history of previous ulcer disease, Helicobacter pylori infection, or age. To prevent one ulcer complication, 10 high risk patients and 268 moderate risk patients need PPI therapy. Network meta-analysis indicated that the effectiveness of different PPIs in reducing ulcer complications and endoscopic peptic ulcers is generally similar. PPIs significantly reduced gastrointestinal adverse events and the related withdrawals compared to placebo; there is no difference in safety between different PPIs.

Conclusions: PPIs are effective and safe in preventing peptic ulcers and complications in a wide spectrum of patients requiring NSAID therapy. There is no major difference in the comparative effectiveness and safety between different PPIs.

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Comparison of oral and intestinal human microbiota and association of *Fusobacterium nucleatum* infection in patients with colorectal cancer: A pilot study

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The study used next-generation sequencing (NGS) to analyze and compare human microbiota from three different environments, saliva, feces, and cancer tissue (CT), of a selected cohort of 10 Italian patients with colorectal cancer (CRC) vs. 10 healthy controls (saliva and feces). Furthermore, the *Fusobacterium nucleatum* (*F. nucleatum*) abundance in the same districts was investigated trough quantitative polymerase chain reaction (RT-qPCR) to assess the association with CRC. The difference of bacterial taxonomic composition, *F. nucleatum* abundance between CRC and healthy controls and the relationship of *F. nucleatum* presence with clinical variables were evaluated. Taxonomic analysis revealed the presence of three main bacterial phyla, which comprises ca. 80% of reads: Firmicutes (39.18%), Bacteroidetes (30.36%), and Proteobacteria (10.65%). The three examined environments showed different bacterial assemblages; in particular, we observed the enrichment of members of Bacteroidetes within fecal samples of CRC patients, while Firmicutes were over-represented in the fecal samples of healthy controls. The CT samples show the highest alpha diversity values. *F. nucleatum* in patients was shown to be more abundant in saliva samples than in feces samples and, notably related to the presence of metastases. These results highlight a different taxonomic composition of feces from CRC compared to healthy controls and that the *F. nucleatum* presence is positively associated with the clinical course of CRC patients (metastasis). So, our results could be useful to promote the development of novel bacteria-related diagnostic tools and therapeutic interventions in CRC patients.

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The effect of time interval between endoscopic retrograde cholangiopancreatography and laparoscopic cholecystectomy

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Aim: The appropriate time for laparoscopic cholecystectomy (LC) following endoscopic retrograde cholangiopancreatography (ERCP) in patients with obstructive choledolithiasis is controversial. We aim to compare early versus delayed LC after ERCP in patients with calcular obstructive jaundice as regards conversion rate, postoperative morbidity and hospital stay.

Methods: This study was conducted on 124 patients who underwent LC after ERCP due to calcular obstructive jaundice. Patients were randomly classified to two groups; in the first group (early group, n=62) LC was performed within 72 hours after ERCP, while in the second group (delayed group, n=62) LC was performed after 6 weeks.

Results: Conversion to open cholecystectomy was significantly more incident when LC was delayed for more than 6 weeks after ERCP (22.6% in delayed group versus 6.5% in early group). The duration of surgery and the postoperative hospital stay in the early group was significantly shorter than that of the delayed group (42.3±10.6 minutes versus 72.2±16.8 minutes and 1.1±1.9 day versus 3.5±1.2 days respectively). No statistically significant difference was found between both groups as regarding the postoperative morbidity.

Conclusion: Performing LC as early as possible (within 72 hours after ERCP) lowers the conversion rate to open cholecystectomy thus decreasing the anticipated postoperative morbidity and prolonged hospital stay.

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Sonographic correlation between portal vein diameter and spleen size (craniocaudal) in Pakistani population

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Objective: Splenic caudocranial length in centimeters (cm) is equal to portal vein diameter in millimeters (mm) in Pakistani population.

Material & Methods: A correlational study was conducted with a sample size of 100 individuals selected conveniently at Gilani Ultrasound Center Lahore. All the normal and abnormal individuals were voluntarily enrolled in this study with signed informed consent form. Approval was taken from the review board and ethical committee. Nemio and Xario (Toshiba) with convex transducer were used for this study. Sonographic caudocranial length of the spleen was taken in the longitudinal plane while the patient in supine or right posterior oblique position with suspended respiration. Portal vein diameter was measured at porta hepatis in longitudinal section in supine or left posterior oblique position in quiet respiration. The diameter was taken by putting the two cursors in the lumen of the portal vein; the wall of the portal vein was excluded. Correlation between the spleen length in centimeters (cm) and portal vein diameter in millimeters (mm) was evaluated with Pearson's correlation in IBM SPSS version 21.

Results: 100 patients; 34% females and 64% males with age's range of 4 to 79 years were included in the study. The mean spleen length was 10.29±1.89 cm and the mean portal vein diameter was 10.27±1.78 mm. Statistical study shows a significant correlation between the spleen size in cm and portal vein diameter in mm; the "r-value" remain 0.01.

Conclusion: A statistical significant correlation was found between the splenic length in centimeter and portal vein diameter in millimeter.

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Serum biomarker panel (GastroPanel®) and slow-release L-cysteine (Acetium® Capsule): Rational for the primary prevention of gastric cancer

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Statement of the Problem: The major risk factors of gastric cancer (GC) are Helicobacter pylori (HP) infection and atrophic gastritis (AG). It is possible to diagnose HP-infection and AG by serological testing with panel of biomarkers (GastroPanel®, Biohit Oyj, Finland): pepsinogen I (PGI), pepsinogen II (PGII), gastrin-17 (G-17) and HP-antibodies. Severe AG leads to acid-free stomach colonized by HP and other bacteria, producing acetaldehyde (Group I human carcinogen; IARC). Together with other conditions leading to acid-free stomach, or those exposing the subjects to increased concentrations of acetaldehyde, these subjects are at high-risk for gastric and esophageal cancer.

Methodology & Theoretical Orientation: GastroPanel® is the first non-invasive diagnostic tool for dyspeptic symptoms, and for screening of asymptomatic subjects for the risks of GC. A novel formulation (Acetium® Capsule, Biohit) based on slow-release L-cysteine, designed to protect the stomach in these high-risk subjects by its capacity to eliminate carcinogenic acetaldehyde.

Findings: The test results test are interpreted by a specially designed software (GastroSoft®) identifying eight diagnostic marker profiles. Of those, four represent purely functional disorders, while three others specify structural abnormalities, and one is typical to HP-infection. Its superb clinical performance was validated in two recent meta-analysis, and the test has excellent longitudinal predictive values for incident GC. Acetium® Capsule is a unique medical device designed to elimination of carcinogenic acetaldehyde in the stomach among the high-risk subjects: AG associated with HP infection; autoimmune AG; cigarette smokers; alcohol consumers; chronic users of PPI medication, and those 500 million people in Asia with mutated ALDH2 enzyme exposed to higher local concentrations of acetaldehyde.

Conclusion & Significance: With a rational use of these two medical devices, one can diagnose the gastric high-risk conditions and subsequently protect the stomach against acetaldehyde exposure.

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Duodenal resurfacing procedure: A novel approach for type 2 diabetes management

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Bariatric surgery has emerged as an effective intervention to treat obesity and its related co morbidities. For multitude of factors, access, insurance, patient fears, referrals and the procedures risks, only 1% of the eligible undergoes bariatric surgery. Considerable needs for effective nonsurgical treatment modalities are mandated. The minimally invasive novel endoscopic therapies with less morbidity could be the answer for many morbidly obese patients. Researches advocate the important role of the foregut in the regulation of glucose homeostasis and diabetes. A novel purely endoscopic catheter-based procedure that targets the duodenal mucosa had been developed by Fractyl Laboratories targeting the abnormal hypertrophy and hyperplasia and the alterations in the enteroendocrine cells of the foregut usually seen in patients with diabetes. This minimally invasive Duodenal Mucosal Resurfacing System DMR is known as Revita. Revita involves two main steps: First, creation of a protective barrier by lifting the sub mucosal space of the duodenum with endoscopic injection of saline and second, hydrothermal ablation (recirculation of hot water within a balloon tipped catheter) of the circumferential duodenal mucosa. This rejuvenation of the lining of the duodenum will change gut signaling in patients with metabolic diseases caused by insulin resistance. The early results with Revita DMR are quite encouraging, with well tolerated procedure, concerning safety, three instances of duodenal stenosis were reported, and treated using endoscopic balloon dilation. The first study involving 39 T2 DM who were failing oral medications, at six months, the treatment had improved glycemic control, with significant decrease in FBG, PPG, and HbA1c. The patients receiving DMR on a long segment (average ¼ 9.3 cm, n ¼ 28) compared to short (average 3.4 cm, n ¼ 11) of the duodenum experienced a greater reduction in HbA1c levels at three months and achieved a reduction in HbA1c levels from 8.5% to 7.1% at six months and about five pounds of weight loss. Further studies are necessary to understand the core mechanism, long-term safety, efficacy, durability and how the procedure performs in a randomized clinical trial setting, while also embracing the potential for wider metabolic benefits.

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DNA demethylation in colon cancer

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DNA hypomethylation increases with patient age and correlates with genomic damage in colon cancer (CC). We proposed a wear and tear model linking aging and cancer by the progressive erosion of genomic DNA methylation, inevitably occurring during aging. Two recent examples of DNA demethylation did not fit the wear & tear model, but added new research avenues. A pericentromeric macrosatellite, named SST1/NBL2, is hypomethylated in 22% of colon cancer (CC) with 7% exhibiting a severe hypomethylation (more than 10%) that co-occurred with TP53 mutations in relatively younger patients. Studying the mechanisms underlying the severe demethylation and its impact in genome stability we found that SST1/NBL2 is expressed as a novel long non-coding RNA, the function of which is under study. In a collaborative study of a cohort of near 1,000 CC patients to search for multiple cancer risk biomarkers, we found that low levels of LINE-1 methylation (a surrogate marker of global methylation levels) correlated with the presence of synchronous CC and were predictive of high risk of developing metachronous tumors. Demethylation levels thus serve as prognostic biomarker for improved identification of individuals at high risk for metachronous CC. Among the patients with enhanced demethylation, those with multiple tumors were younger, supporting a role of genetic factors in the increased risk to develop multiple CC. A long-term ongoing prospective cohort study called Genomes for Life (GCAT) at our institution will be useful to further explore the association between epigenetic alterations and the risk for multiple CC. GCAT was designed to explore the role of epidemiologic, environmental, genomic, and epigenomic factors in the development of cancer and other chronic diseases in Catalonia, Spain. GCAT will have recruited 20,000 participants at the end of 2017 with whole genomes sequenced for 1,000 volunteers.

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The influence of all oral antiviral treatment on carcinogenesis in HCV chronic hepatitis

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Introduction: After the initial enthusiasm of an easy cure for HCV chronic hepatitis using all- oral antiviral treatment, new problems have begun to emerge, one of the most important being the remaining risk of hepatocellular carcinoma (HCC). This study analyzes the particularities of HCC diagnosed during or after treatment with paritaprevir/ombitasvir/ritonavir, dasabuvir with or without ribavirin.

Methods: 173 patients with HCV cirrhosis and 105 patients with F3 degree of fibrosis were included in the study. We followed the patients between January 2016 and March 2017. Alpha-fetoprotein and abdominal ultrasonography were routinely performed at the initiation of therapy and every 3 months afterwards. If AFP levels were higher than the normal value or twice the previous value, abdominal CT or contrast- enhanced ultrasonography were performed.

Results: Before therapy, 120 patients with F4 fibrosis and 28 patients with F3 fibrosis had higher levels of AFP. Abdominal imaging did not reveal any HCC nodules. On treatment, five patients presented higher levels of AFP. Abdominal CT revealed single HCC nodule in three patients and multiple nodules in the other two. The patients continued paritaprevir/ombitasvir/ritonavir, dasabuvir. Transarterial chemoembolization was performed in three patients (without portal vein thrombosis), while on antiviral treatment, with good outcome and at the end of treatment two patients presented higher levels of AFP (twice the initial values). As they both had previously undergone CT scan, we performed abdominal MRI which revealed single HCC nodules and the patients refused surgical resection and underwent transarterial chemoembolization, with good outcome. At three months after end of treatment three patients presented increased level of AFP and these patients required MRI evaluation as abdominal CT scan was not able to determine the exact sizes and extension. In all patients, the abdominal MRI showed more lesions and a larger extension that the CT had anticipated. These patients also underwent chemoembolization; one month and three months follow-up showed no tumor progression and AFP levels decreased. Notably, all patients acquired sustained virologic response.

Conclusion: The screening for hepatocellular carcinoma needs to be continuous, even after virologic cure. Patients who develop HCC after antiviral treatment need to be evaluated by MRI in order to detect the extension of the disease as these tumors are more often infiltrative.

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Acute-on-chronic liver failure: An update

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Acute-on-chronic liver failure (ACLF) is an increasingly recognized distinct disease entity encompassing an acute deterioration of liver function in patients with chronic liver disease. Although there are no widely accepted diagnostic criteria for ACLF, the Asian–Pacific Association for the Study of the Liver (APASL) and the American Association for the Study of Liver Disease and the European Association for the Study of the Liver (AASLD/EASL) consensus definitions are commonly used. It is obvious that the APASL and the AASLD/EASL definitions are based on fundamentally different features. Two different definitions in two different parts of the world hamper the comparability of studies. Recently, the EASL-Chronic Liver Failure Consortium proposed new diagnostic criteria for ACLF based on analyses of patients with organ failure. There are areas of uncertainty in defining ACLF, such as heterogeneity of ACLF, ambiguity in qualifying underlying liver disease, argument for infection or sepsis as a precipitating event, etc. The two ACLF definitions result in differences in mortality and patient characteristics among ACLF patients. Although the exact pathogenesis of ACLF remains to be elucidated, alteration of host response to injury, infection, and unregulated inflammation play important roles. The predisposition, infection/inflammation, response, organ failure (PIRO) concept used for sepsis might be useful in describing the pathophysiology and clinical categories for ACLF. The mechanisms of ACLF were described recently. In 1/3 of cases of ACLF, inflammation develops in response to bacterial infection. However, a significant number of cases are not related to obvious bacterial infection. In these cases, the translocation of PAMPs without viable bacteria or the releases of DAMPs by dying cells are likely mechanisms of the 'sterile inflammation'. Finally, a decrease in the tolerance to inflammation could be involved. Treatment strategies are limited to organ support, but better understanding of the pathophysiology is likely to lead to discovery of novel biomarkers and therapeutic strategies in the future.

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13th International Conference on Clinical Gastroenterology & Hepatology
&
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December 07-08, 2017 Madrid, Spain

Intestinal lymphoma in young male with a hidden diffuse nodular lymphoid hyperplasia and selective Ig A diffecincy

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This is a case of young male patient presented by chronic diarrhea which was attributed at first to intestinal lymphoma, but persistence of the symptoms pushed us for further evaluation to diagnose a rare hidden disease (DNLH) with selective IgA deficiency diffuse nodular lymphoid hyperplasia (DNLH) is a benign rare condition of unknown etiology characterized microscopically by diffuse hyperplasia of the lymphoid follicles (<0.5 cm in diameter) of the gastrointestinal tract mucosa. It can involve any part of the GIT, mainly the small intestine, but it may also involve the colon and rarely the stomach. The disease is usually associated with immunodeficiency syndromes such as common variable immunodeficiency or selective IgA deficiency syndrome. Its prognosis is usually benign but it carries the risk of malignant transformation characteristically to lymphoma.

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Association of 4874 A/G (rs4969170) polymorphism in SOCS3 gene promoter region and RNA expression with liver fibrosis progression in patients with chronic hepatitis C

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Context: Chronic hepatitis C virus infection (CHC) is one of the most important risk factor of hepatocellular carcinoma (HCC). However, the pathogenesis of Insulin Resistance (IR) in hepatitis C infection is a very intriguing problem. In fact, the HCV is now recognized responsible for direct interference with the insulin signaling pathway. In addition, HCV-related IR has been shown to have a remarkable clinical impact on the progression of hepatic fibrosis and development of HCC.

Objective: The present study aims to evaluate the association of 4874 A/G (rs4969170) polymorphism in SOCS3 gene promoter region and RNA expression with liver fibrosis progression in chronic hepatitis C infected patients.

Materiel & Methods: In this study 226 Moroccan patients chronically infected with HCV (95 patients with mild fibrosis and 131 patients with advanced fibrosis) were genotyped for 4874 A/G (rs4969170) variant using the real time PCR. SOCS3 mRNA expression analysis was performed by using Sybr Green. Logistic regression was used to assess the association between polymorphism and progression of HCV infection.

Results: A significant difference in genotypes distribution of rs4969170 was detected between mild and advanced fibrosis group. The AA genotype was significantly overrepresented in Ad-LD patients compared to m-LD, the AA genotype was associated with a 5-fold increase of AdLD risk when compared to mild chronic hepatitis C (OR = 5.14; 95% CI, 2.29 - 11.54; P=0.00004). A similar situation was observed with the dominance model (OR = 4.18; 95% CI, 2.19 - 7.97; P=6.374e-06). The relative expression of SOCS3 to GAPDH mRNA was increased by 2 fold in Ad-LD as compared to m-LD group with AA genotype.

Conclusion: Our results suggest that polymorphism in SOCS3 gene promoter modulates the progression of chronic hepatitis C infection toward advanced liver disease by affecting its mRNA expression.

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The impact of toll-like receptor 9 polymorphisms on hepatitis B virus clearance

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Statement of the Problem: Hepatitis B infection remains a serious public health problem in the world. In connection with poorly defined defects affecting their immune competence, patients chronically infected with hepatitis B virus (HBV) cannot clear the virus. The outcome of infection depends primarily on the interaction between the virus and selected effectors of host immunity. Toll-like receptor 9 (TLR9) plays a crucial role in innate immunity against viral infections through detection of intra-cytoplasmic dsDNA. Defects in this system may result, therefore, in attenuated responses against HBV. Recent research has focused on the possibility of targeting the defects in TLR9 pathway as a novel approach for anti-HBV treatment. Our study aimed to assess the impact of both TLR9 rs5743836 and rs187084 polymorphisms on spontaneous HBV clearance in Moroccan patients.

Material/Methods In this study, 239 chronic HBV (CHB) patients and 134 spontaneously resolved HBV (SRB) individuals were recruited and genotyped using a Taqman allelic discrimination assay.

Results: Remarkably, we observed dosage effect of both SNPs on viral loads. At rs5743836, AA, AG and GG genotypes were significantly associated with a progressive increase of circulating HBV DNA whereas the inverse phenomenon was noticed with AA, AG and GG at rs187084. By contrast, there was no consistent association between TLR9 polymorphisms and spontaneous clearance or persistence of HBV.

Conclusion: To conclude, of Moroccan patients, no significant association of rs5743836 and rs187084 TLR9 polymorphisms was observed with HBV natural clearance. Further studies on larger populations should shed light on the modulating effect of TLR9 polymorphisms on HBV loads that remain a viral factor of paramount importance to predict HCC development.

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Sugar sensor genes in the murine gastrointestinal tract display a cephalocaudal axis of expression and a diurnal rhythm

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Issue: The mechanisms responsible for regulating the expression of the intestinal macronutrient sensors are unknown. Many physiological processes (e.g. intestinal sugar transport) display a diurnal rhythm that is controlled by clock genes. We therefore hypothesised the GI tracts nutrient sensors also display a diurnal rhythm that is entrained by clock genes.

Aims: In rodents, determine evidence of a diurnal rhythmicity in sugar sensor (T1r2/3, SGLT3) and sugar transporter (SGLT1, GLUT2, GLUT5) and gut peptide expression levels along the length of the GI tract.

Methods: Sixteen C57BL/6J mice were fed ad libitum at the standard 12 h light/dark cycle. After six weeks the animals were sacrificed at 7 am (n=8) and 7 pm (n=8). Tongue, stomach, duodenum, jejunum & ileum were prepared for RT-qPCR. Expression levels for each gene were relatively quantified against three reference genes using the 2-ΔΔCT method.

Results: Sweet taste receptor (tas1r2/tas1r3/gnat3/gnat1) sugar transporter (slc5a1, slc2a2, slc2a5) and putative sugar sensor (slc5a4a and slc5a4b) gene expression levels were highest in tongue, proximal and distal small intestine, respectively. Clock gene (cry2/arntl) activity was detected in all regions studied. Slc5a4a and slc5a4b gene expression showed clear diurnal rhythmicity in the small intestine and stomach, respectively, although no rhythmicity was detected in SGLT3 protein expression. Tas1r2 and tas1r3, gnat3 and gcg displayed a limited rhythm in gene expression in proximal small intestine. Microarray analysis revealed a diurnal rhythm in gut peptide gene expression in tongue (7 am vs. 7 pm) and in silico promoter analysis indicated intestinal sugar sensors and transporters possessed the canonical E box elements necessary for clock gene control over gene transcription.

Conclusion: Sugar sensors, transporters and gut peptides, but not αGustducin/transducin, exhibit a diurnal pattern of gene expression in specific regions of the GI tract. Disruption in clock control of intestinal nutrient sensing may contribute to disturbances in metabolism.

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A 24-year old female with indeterminate hyperacute liver failure: A case report

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Background: Acute liver failure (ALF) in young age is rare, yielding limited known data in its pathophysiology and management. ALF refers to sudden massive hepatic necrosis with encephalopathy and impaired synthetic function without pre-existing cirrhosis.

Case Description: A previously healthy 24 year old female with a history of lacrimal gland tumor on chronic oral prednisone (40 mg) for a year was admitted for acute decreased sensorium, generalized jaundice, tea-colored urine, anorexia and undocumented fever. Interval between jaundice and encephalopathy was hyperacute (<7 days).

Results: Laboratory findings showed hyperbilirubinemia, transaminitis, elevated alkaline phosphatase, impaired coagulation hyperammonemia and normal platelets. Extensive work-up including hepatitis panel, paracetamol, methamphetamine, cannabinoids, benzodiazepine, barbiturates, cocaine, opiates, phencyclidine, cytomegalovirus IgM, EBV, HSV1, HSV2, C3, anti-Sm and anti-mitochondrial antibody, LKM1, ceruloplasmin, strepA throat screen test, malarial smear and leptospiral IgM were all unremarkable. Medical and supportive treatments were promptly provided and orthotopic liver transplantation (OLT) was contemplated, however, cerebral edema and hemorrhage ensued on day 5 leading to demise.

Discussion: Etiology varies widely among toxic, viral, metabolic and vascular insults. There are rare reports of ALF with repeated steroid administration. Management consisting of intensive care should be initiated depending on the etiology and chronicity of ALF. OLT has emerged as the only therapeutic intervention with proven benefit for patients with advanced ALF.

Conclusion: We report a case of indeterminate hyperacute liver failure in a healthy female. Despite extensive work-up and prompt intensive medical management, rapid clinical deterioration ensued. History of chronic steroid use might be a precipitant, as supported by few case reports.

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