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Genetics of inflammatory bowel disease

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Crohn's disease (CD) and ulcerative colitis are inflammatory conditions, collectively referred to as inflammatory bowel disease (IBD), which results from defects in the regulation of mucosal immune responses to enteric bacteria in genetically susceptible individuals. Multiple lines of evidence suggest a genetic contribution to the pathogenesis of IBD, which include racial and ethnic differences in disease prevalence, familial aggregation and link to other genetic syndromes. Recent genome-wide association studies (GWAS) have identified >200 genetic variants associated with IBD risk, some of which have functions in biological pathways of pathogen recognition, internalization and autophagy. However, GWAS-identified loci have explained less than a quarter of the heritability estimated for IBD and many are confined to noncoding regions, requiring further studies to understand their role in disease pathogenesis. Recently, next generation sequencing efforts, most successful in isolated populations and individuals with early age of onset and/or significant family history of IBD, identified rare coding variants associated with IBD risk that are more amenable to functional studies than GWAS loci. Also, a number of genetic variants have been linked to adverse events resulting from IBD therapies, particularly thiopurine exposure, including bone marrow toxicity and pancreatitis. Yet, despite substantial progress in the field of genetics and genomics of IBD, reliable tools to identify individuals at risk, determine disease progression and predict response to therapies are still lacking. More comprehensive approaches that incorporate clinical, genetic, epigenetic, metabolomic, and microbiome data need to be developed to allow for an early diagnosis and personalized treatment for IBD.

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Evaluation of the impact of pre & post-transplant metabolic derangements on the neurological complications following liver transplantation

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Neurologic complications after liver transplantation are a major source of morbidity and mortality and proper prediction for those at risk may help in improving the outcome. The results of our study showed that severity of end stage liver failure prior to transplantation might be the most common risk factor for the development of post-transplant neurological complications and careful evaluation of other risk factors may be required for those patients in order to decrease the incidence of complications. Still the use of Tacrolimus is associated with risk of neurological complications and reduction or discontinuation of Tacrolimus lead to improvement of neurological complications. According to our study, electrolytes and metabolic derangements are not risk factors for development of neurological complications. Although the risk of neurological complications in our series is high but there was no impact on the survival.

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Chronic diarrhea: More than just bugs and drugs

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Amyloidois is the term for a group of protein folding disorders characterized by the extracellular deposition of insoluble polymeric protein fibrils in tissues and organs. Amyloidosis is commonly systemic, occasionally organ-limited, and rarely a solitary localized mass. This latter presentation is commonly referred to as tumoral amyloidosis. Although reports exists of these often called "amyloidomas" showing up in almost every tissue/organ, the GI tract has a prevalence that is not well document making it an outstanding diagnostic challenge. A 66 year-old male with a history of IV drug abuse, comes to our hospital to be evaluated due to diarrhea that started 2 months ago; 3-5 depositions a day, watery in consistency no blood or mucus, associated with epigastric abdominal pain described as "burning" in nature, 7/10 intensity without radiation and a 50 pound weight loss. Denied fever, chills, shortness of breath, nausea or vomiting; symptoms were non consistently worsened with food ingestion and did not improve with OTCmedication for diarrhea. PMH was significant for HCV diagnosed 1 month ago. On physical exam the abdomen showed a prominent liver edge 5cm below the costal margin non tender, non-distended without ascites. Laboratory work-up upon admission showed metabolic acidosis, acute kidney injury, no electrolyte disturbances and an elevated TSH. Esophagogastroduodenoscopy (EGD) done the day after admission showed an esophagus that was normal and a friable mass in the antrum of the stomach that bled on contact, cold forceps biopsy was taken. A colonoscopy was also performed, with unremarkable findings; random biopsies taken. Pathology reports a tissue that on red Congo stain has apple-green birefringence indicative of amyloid fibrils in both colonic and gastric samples. The deposition of amyloid fibrils in other organs were sought out with negative results; thus giving the impression of single system involvement. Gastrointestinal amyloidosis causes severe malabsorption due to the deposition of the protein fibrils, explaining the patient's chronic diarrhea and significant weight loss. Since patient's malabsorption caused wasting and malnourishment, total parenteral nutrition was indicated while the patient received chemotherapy for the treatment of amyloidosis. This case illustrates that there is an important risk of misunderstanding and diagnosis delay of patients that present with malabsorption. Even if the clinical symptoms are not obvious upon initial presentation, the hypothesis of gastrointestinal amyloidosis should be considered among the possible diagnosis of patients with chronic diarrhea and weight loss. In doing so, quality of life as well as morbidity improvement should be evident

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PDZK1 targets PTEN to inhibit AKT signaling and malignant phenotypes in gastric cancer

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PI3K/AKT pathway, which is frequently altered in gastric carcinoma, can be negatively regulated via dephosphorylation of PIP3 to PIP2 by (Phosphatase and Tensin Homolog) PTEN. In the present study, PDZK1 was identified as a novel binding protein of PTEN, in which the interaction was mediated by the PDZ2 and PDZ3 domain of PDZK1 with the last four amino acids (ITKV) in the carboxyl terminus of PTEN (PTEN-CT). Our data from PDZK1 over-expression and siRNA-mediated knock-down experiments further demonstrated that by associating with PTEN, PDZK1 inhibits the phosphorylation of PTEN. In addition, over-expression of PDZK1 down-regulated AKT and ERK signals. Consistent with these results, PDZK1 suppressed gastric cancer cell proliferation, impeded the formation of anchorage independent colonies in soft agar and retarded the growth of xenografts in nude mice. Furthermore, PDZK1 was significantly downregulated in gastric cancer tissues in comparison to that in normal gastric tissues. Collectively, this study shows that down-regulation of PDZK1 expression enhances the PTEN inactivation, which may contribute to the carcinogenesis of gastric cancer.

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"The Hidden Evil"- GI bleed and small bowel obstruction caused by carcinoid tumor found during exploratory laparotomy

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Intestinal carcinoid tumors are uncommon malignancies which grow slowly, and rarely cause any symptoms. Small bowel tumors can, at times, cause bowel obstruction and rarely bleeding. We present to you a 52 year old male who presented to the hospital for evaluation of melena. His endoscopy and colonoscopy came back negative and patient unfortunately, failed to get capsule endoscopy as an outpatient. One year later, patient presented with excruciating abdominal pain and was found to have small bowel obstruction with multiple transition points. Patient was found to have 4 nodular lesions in the small intestine which were found to be carcinoid tumor. Surgical resection definitely improved his outcome and patient did not need adjuvant therapy post-surgery. This patient was a diagnostic challenge due unusual presentation and negative CT scan imaging during both presentations. Carcinoid tumors are highly infiltrating tumors hence, high degree of suspicion should be kept for earlier detection and better outcome.

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Role of vitamin D deficiency, C- reactive protein and adhesion molecules in severity of ulcerative colitis

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Ulcerative colitis (UC) is a chronic inflammatory bowel disease (IBD). The hallmark of UC lesions is infiltration of the intestine by mononuclear cells, predominantly lymphocytes. There is growing epidemiological evidence that suggest implication of vitamin D deficiency in the development of IBD and also its influence on disease severity. C-reactive protein (CRP) levels are often used in the follow-up of patients with IBD. Adhesion molecules such as intercellular adhesion molecule (ICAM) and E-selectin are cell surface-expressed glycoproteins that play a prominent role in leukocyte recruitment and proliferation in the inflamed colon. The aim of the present descriptive study is to investigate the role of vitamin D, CRP and the adhesion molecules ICAM and E-selectin in prediction of severity of UC. Samples of blood were taken from 24 diagnosed cases of UC for measurement of serum levels of vitamin D, CRP, ICAM and E-Selectin by enzyme-linked immunosorbent assay (ELISA). Severity index for UC cases and its correlation with the aforementioned measures was determined. Comparing to control groups formed by individuals without clinical and/or laboratory signs of UC, UC patients showed significant increased levels ($p<0.001$) of sICAM-1, E-selectin and CRP in serum samples. On the contrary, vitamin D levels were significantly decreased in UC patients. Strong correlation was statically determined between vitamin D deficiency, CRP, adhesion molecules and UCEIS. In conclusion, the present work confirmed the role of vitamin D deficiency, adhesion molecules notably ICAM and E-selectin and the acute phase biomarker CRP in pathogenesis of UC.

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Role of serotonin in detection of esophageal and fundal varices

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Portal hypertension is a major complication of liver cirrhosis and can be a direct cause of variceal hemorrhage and of bleeding related death. Oesophageal variceal bleeding is one of the most dreaded complications of liver cirrhosis because of its high mortality Prevalence of varices in patients with cirrhosis is 60-80% with incidence increasing 5% per year. The American Association of the Study of Liver Disease and the Baveno V Consensus Conference on portal hypertension recommended that all cirrhotic patients should be screened for the presence of O.V when liver cirrhosis is diagnosed Identification of non-invasive predictors of O.V and portal gastropathy will able us to carry out UGE in selected group patients thus avoiding unnecessary intervention and at the same time not missing the patients at risk of bleeding. Serotonin (5-hydroxytryptamine, 5HT) has been the subject of intense biological research since its synthesis in 1951.Erspamer and Asero.originally isolated a potent vasoconstrictor substance from the intestine, which they called enteramine. About 95% of serotonin in the body is found in the GI tract, of which 90% is in enterochromaffin cells (ECs) and 10% in enteric neurons. The remaining of serotonin (5%) is found in the brain. As serotonin cannot cross the blood-brain barrier, the brain must synthesize its own serotonin. Virtually all of the serotonin in the blood is derived from the GI tract With respect to the liver, it was found that serotonin has the ability to regulate hepatic blood flow at both the portal and sinusoidal levels Serotonin is able to induce the contraction of fenestrae which is achieved via a rapid influx of extracellular Ca2+ leading to activation of the myosin light chain In these cells serotonin also inhibits cAMP production, and activates phospholipase A2, causing the release of arachidonic acid The exact significance of these findings has not been fully qualified, it is, however, well established that SEC fenestrae play an important role in the exchange of fluid, solutes and particles between the parenchyma and the blood. Serotonin may therefore play a role in regulating the exchange of various fluids, solutes and particles across the space of Disse. Serotonin in these cells may also exert complex control over various aspects of inflammation and immunity since arachidonic acid is a precursor of various prostaglandins, prostacyclin, and thromboxane.

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