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Risk factors associated with the severity of acute hepatitis A by phase

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Introduction: Acute hepatitis A (AH-A) is one of the most common forms of viral hepatitis, and distinct clinical features are associated with the prodromal, icteric and recovery phases. This study was designed to investigate the correlations of various clinical parameters with severity in AH-A patients in each of these three phases.

Methods: The medical records of 455 patients diagnosed with AH-A were retrospectively reviewed. The prodromal, icteric and recovery phases were defined by the patterns of changes observed after admission in aspartate aminotransferase (AST), alanine aminotransferase (ALT) and bilirubin levels. Clinical parameters, including phosphate levels, were analyzed to identify their associations with the peak levels of AST, ALT and bilirubin.

Results: Of the patients, 129 (28.4%) were admitted in the prodromal phase, 187 (41.1%) in the icteric phase and 139 (30.5%) in the recovery phase. Phosphate levels showed an inverse relationship with the peak AST and ALT levels in the prodromal phase (P=0.011 and P=0.005, respectively). Prothrombin time (PT, %) showed a negative relationship with peak AST levels throughout the prodromal, icteric and recovery phases (P=0.039, P=0.028 and P=0.001, respectively), the peak ALT level in the prodromal phases (P=0.038) and the peak bilirubin level in the icteric phase (P=0.029).

Conclusion: In conclusion, the baseline phosphate, AST, and ALT levels, as well as PT and the platelet count, were correlated with the peak levels of AST, ALT and bilirubin in patients with AH-A.

Biography

Sangheun Lee has completed his MD and PhD from Catholic Kwandong University. He is a Doctor in St. International Mary's Hospital, Catholic Kwnadong University in Korea. He has published more than 20 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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Accepted Abstracts

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Acute brainstem syndrome secondary to malnutrition from functional dyspepsia

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Brain stem syndrome is a condition commonly characterized by limb weakness, ophthalmoplegia and gait disturbances. The common causes of brain stem syndrome are ischemia, neoplasia, demyelination, infective and hamartomous lesions in the brain. Imaging ideally with a MRI scan is usually diagnostic in most cases and possibly following other investigations to identify systemic abnormality or CSF changes before appropriate therapy can be introduced. A 42 year old Caucasian lady presented with non-specific symptoms like lethargy, malaise, was off food for a couple of months and had lost a considerable amount of weight (4 stones=25.4 kg). She was admitted to the hospital with nausea and vomiting for a few weeks and complained of a lump in her throat. Gastroscopy was unremarkable. She also complained of sudden onset of double vision for the last few days and examination showed vertical Nystagmus. She also complained of unusual sensation in her feet and soreness in the bottom of her feet when she stood up. There was no obvious limb ataxia, absent lower limb tendon jerks but flex or plantars and intact objective peripheral senses. Gait was unsteady while walking with eyes open but was better with eyes closed. The patient was lucid the whole time. During the course of the stay in the hospital the patient developed Oscillopsia. She underwent a MRI scan which was unremarkable. She was investigated for autoimmune cause including GQ1b for Miller Fischer syndrome and paraneoplastic screen to investigate the weight loss. Lumbar puncture which was performed which showed protein of 0.69 and rest of the values are normal. She was transferred to a tertiary neurology centre. Based on her clinical examination finding and MRI report she was diagnosed with brain stem syndrome secondary to malnutrition due to functional dyspepsia. She was seen by the dieticians and NG feed was started.

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When life hands you lemons: A case of primary biliary cirrhosis characterized by pica

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Pica disorder is the ingestion of non-nutritive items often implicated in Iron-Deficiency Anemia (IDA). We present a 48-year-old female presented with worsening fatigue and shortness of breath that concurrently started eating one bag of unpeeled lemons daily for two months. The patient had normal vital signs and mild jaundice. Labs showed hemoglobin 3.3 g/dl, micro-cytosis and elevated liver enzymes. Computed tomography of the abdomen showed hepatomegaly, mild nodularity of the liver, splenomegaly and was negative for ascites. Further workup showed IDA and anti-mitochondrial antibodies >1:40. Esophagogastroduodenoscopy showed multiple esophageal varices, without active bleeding. The patient declined liver biopsy. Her craving for lemons, shortness of breath resolved after blood transfusion and she was started on ursodeoxycholic acid. Five months after discharge, she continues to do well. IDA is a common finding in PBC, 85% of PBC patients experience fatigue during the course of the disease. We present this case because of the subtle, non-classical presentation of pica which is implicated in IDA and was initially missed in the outpatient clinic. It is our understanding that only one case of pica presenting with craving for unpeeled lemons has been reported. Iron deficiency anemia can occur in patients who appear to have early stage PBC. Screening for GI bleeding is indicated, even in the absence of overt bleeding, since affected patients are at risk of severe portal hypertension despite normal bilirubin levels and absence of cirrhosis on liver biopsy. Therapy begins with iron replacement therapy. In cases of refractory pica, selective serotonin reuptake inhibitors and a formal mental health referral may be warranted.

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Large diaphragmatic hernia induced reversible heart failure

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Diaphragmatic hernias are typically congenital and happen during the fetal development stages. Diaphragmatic hernias can be classified into two types: Bochdalek and Morgagni. Bochdalek type is seen in this case and is defined where the stomach and small intestine contents are found in the thoracic cavity. Below we see a rare case in which a diaphragmatic hernia leads to a reversible cause of heart failure. This is a 50 year female with PMH of hypertension and diabetes presenting with worsening shortness of breath, on and off for the last three weeks. Patient denied any fever, chills, chest pain or wheezing. Shortness of breath with rest and exertion is associated. Physical exam showed mild JVD elevation and bilateral lower extremity trace edema. On admission, CXR showed right middle lobe pneumonia and large left sided diaphragmatic hernia. CT chest then confirmed the above and showed the diaphragmatic hernia penetrating posteriorly into the thoracic cavity. ECHO showed both grade three diastolic dysfunctions without concentric hypertrophy and reduced ejection fraction of 40%. BNP level was 748. Hemoglobin A1c was 6.8, on Metformin and Glipizide. Because of ECHO results, patient underwent a Lexiscan stress test in which it showed a high risk of CAD with possible inferior ischemia. Patient then underwent cardiac catheterization which confirmed an ejection fraction of 40% and only mild stenosis of 25% of the LAD. Patient started on Carvedilol and Lasix in the addition of Lisinopril that she was already on for hypertension. Patient was then referred to surgery for correction of the diaphragmatic hernia. Repeat ECHO three months post operatively showed that ejection fraction improved to 50% and diastolic dysfunction improved as well now only being Grade 1 diastolic dysfunction. Diaphragmatic hernia has been associated with respiratory complications but typically not cardiac. In this case, the patient was found to have both systolic and diastolic dysfunction which resolved with correction of the diaphragmatic hernia. Initially patient was thought to have an ischemic cardiomyopathy, but once cardiac catheterization was unremarkable this pointed more towards the diaphragmatic hernia. Additional concern was patient's history of diabetes with this potentially contributing to heart failure. Given that patient's hemoglobin A1c was 6.8, again this pointed towards the diaphragmatic hernia contributing to these findings. Given the increase in intra-thoracic pressure in the setting of the large diaphragmatic hernia, diastolic function is affected. Left ventricular filling is compromised during this stage of the cardiac cycle. Hypertension was of concern as well, but again, given the fact that the ECHO did not demonstrate any concentric hypertrophy and the reversal after corrected surgery, we can conclude that this was secondary to the diaphragmatic hernia.

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Novel therapeutic targets for acute pancreatitis

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Acute pancreatitis is a common clinical condition. Excessive Systemic Inflammatory Response Syndrome (SIRS) in acute pancreatitis leads to distant organ damage and Multiple Organ Dysfunction Syndrome (MODS), which is the primary cause of morbidity and mortality in this condition. Development of in vivo experimental models of acute pancreatitis and associated systemic organ damage has enabled us to study the role played by inflammatory mediators in the pathogenesis of acute pancreatitis and associated systemic organ damage. Using these models, recent studies have established the critical role played by inflammatory mediators in acute pancreatitis and the resultant MODS. Hydrogen sulfide (H2S) plays an important role in cardiovascular, central nervous and gastrointestinal systems and has been shown to act as a vasodilator. We have also shown that H2S acts as a mediator of inflammation. Substance P is an 11 amino acid neuropeptide that is released from nerve endings in many tissues. Subsequent to its release, substance P binds to neurokinin-1 (NK-1) receptors on the surface of effector cells. Using experimental models, recent studies in our laboratory have established the critical role played by H2S and substance P in acute pancreatitis. Furthermore, early results point to the clinical relevance of this research. Studies with experimental animal models of disease will therefore help define the role of these mediators in the pathogenesis of acute pancreatitis and can lead to the development of novel therapeutic approaches for this condition.

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The role of vitamin D/VDR signaling within key mechanisms of ulcerative colitis

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Background & Aim: Epidemiological evidence has shown an associative relationship between low serum vitamin D levels (<35 ng/mL) and disease activity in ulcerative colitis patients. The biological activity of vitamin D is observed to occur through ligand bonding with the VDR, which is highly expression within intestinal epithelial cells. Due to the observed immune-modulatory effect of vitamin D/VDR signaling and the high expression of the VDR within the intestinal track, this review aims to elucidate what role vitamin D/VDR signaling may play in key mechanisms of colitis. To include: Intestinal barrier dysfunction, IEC apoptosis, macrophage inflammation and reduced and penetrable mucus layer. Furthermore, due to the associative link between low serum vitamin D and disease, this review aims to critically appraise the efficacy and safety of vitamin D supplementation in UC patients.

Methods: A systematic and replicable search strategy was employed within this review. PubMed was systematically search from 2005 to 2016 using the terms: Ulcerative colitis or colitis or inflammatory bowel disease or IBD, followed by key search terms pertinent to the mechanism under investigation. Of the 1140 papers returned, 80 papers were accepted within this review.

Results: Within this review, TNF-a was observed to promote intestinal permeability, macrophage inflammation and apoptosis in a NF-kB dependent mechanism. TNF-a signaling was observed to up-regulate the expression of the NF-kB protein p65, which was observed to: Promote intestinal permeability through the up-regulation and phosphorylation of myosin light chain kinase, promote intestinal epithelial cell apoptosis through the up-regulation of p53 Up-regulated Modulator of Apoptosis (PUMA) and promote excessive and prolonged macrophage inflammation through the inhibition of Suppressor of Cytokine Signaling 1 (SOCS1). Conversely, vitamin D/VDR signaling emerged as a key inhibitor of P65 associated transcriptions, being observed to physically bind with the p65 protein and attenuate its' transcriptional activity. The VDR was observed to be significantly down-regulated in the active lesions of ulcerative colitis patients and significantly associated with an exacerbation of colitis symptoms in murine models. TNF-a was observed to actively down-regulate the expression of the VDR in a microRNA-346 dependent mechanism, whereas, 1, 25(OH) 2D3 supplementation was observed to promote VDR expression. An associative relationship was proposed within this review between vitamin D/VDR signaling, cathelicidin expression and the promotion of mucus production. However, there was paucity in studies investigating this relationship explicitly and so the association remains speculative at this time. 1, 25(OH) 2D3 supplementation emerged as a safe and effective way to increase serum vitamin D levels in ulcerative colitis patient, with 2000 IU/day being observed as an efficacious, safe dose associated with increased serum vitamin D levels.

Conclusion: Increasing mechanistic evidence suggests a role for vitamin D/VDR signaling within key mechanisms of colitis. Further investigation is required to ascertain whether VDR down-regulation in the active lesions of UC patients is an associative factor in colitis severity and progression. The therapeutic potential of vitamin D supplementation in ulcerative colitis patients' warrants further investigation in long term randomized controlled trials.

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