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Genetics of inflammatory bowel disease

Inga Peter
Icahn School of Medicine at Mount Sinai, USA

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.

inga.peter@mssm.edu

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

Mohamed Mohamed Bassam Faek Mohamed Hashem
Cairo University, Egypt

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.

mohamedhashem50@hotmail.com