WHATS NEW IN INFLAMMATORY BOWEL DISEASE

Sandeep Shivananda Siddhi (Gastroenterology Registrar)
Correspondence to: Sandeep Shivananda Siddhi: sandeepsiddhii@gmail.com

WHATS NEW IN.....

It is exciting times ahead in the field of Inflammatory Bowel disease. Our understanding of the etiology, genetics, pathogenesis, human–microbiota relationship, the finer details of inflammatory cascade has advanced immensely in the recent year. This will profoundly influence the management of this challenging disease.

The last 12 months has seen a tremendous growth in our knowledge of genetic architecture of IBD.[1] By understanding the genetics we can predict the susceptibility and behaviour of the disease, and therefore have the potential to modify its course and ultimately cure it.[1-2] What started of with twin studies in 1980's has metamorphosed through progressive developments and now with Genome wide association studies and Whole genome sequencing about 71 susceptibility loci for Crohn's and 47 for Ulcerative colitis have been identified.[1-4] This number keeps growing day by day and very shortly we will be using gene sequencing as routine diagnostic workup of patients, although we are not there yet.

IBD is understood to be caused by an imbalance between pro-inflammatory and anti-inflammatory cytokines. These complex intercellular and intracellular cascades have been studied and understood with breathtaking detail. More recently a lot of research has been focused on IL17 secreted by TH17 cells. The role of IL23, IL 12, the cell surface integrins and cell adhesion molecules has been elucidated. The process of apoptosis has been viewed in a new light. Some of the pro-inflammatory molecules have an anti-apoptotic activity, which initiate and maintain pro-inflammatory cells. This is a very good potential target for therapeutics.[2] All these novel ideas have led to the development of new treatment strategies and many new drugs are in various stages of development, especially the monoclonal antibodies targeting multiple pro-inflammatory cytokines.

The approach to treatment has also changed. This is true especially of Crohn's disease. The traditional escalation of treatment from steroids through to potent immunosuppressants has been questioned.[5] Aminosalicylates in Crohn's is falling into disuse. Now the emphasis is on predicting patients who have a high risk of aggressive disease and treating them with monoclonal antibodies at an early stage, the top down approach

The next few years will no doubt see more evidence rolling out and the way IBD is understood and managed will change forever.[6-7]

Siddhi S.S (2012). What's New in Inflammatory Bowel Disease (SUMJ Series). Scottish Universities Medical Journal. EPub 011

References

1.Anderson, c., Boucher, G., & Lees, C. e. (2011). Meta-analysis identifies 20 additional ulcerative colitis risk loci, increasing the number of confirmed asociations to 47. Nature Genetics (43(3)), 246-52.

2.Colombel, J., Rutgeerts, P., Reinisch, W., & al., e. (2010). SONIC : a randomised, double blind, controlled trial comparing infliximab and infliximab plus azathioprine in pateints with Crohn's disease naive to imunomodulators and biologic therapy. New England Journal Medicine (23), 144-9.

3.Franke, A., McGovern, D., Barrett, J., & al, e. (2010). Genome wide meta-analysis increases to 71 the number of confirmed Crohn's disease susceptibility loci. Nature Genetics (42(12)), 1118-1125.

4.Lees, C., & Satsangi, J. (2009). The genetics of Inflammatory Bowel disease: inplications for disease pathogenesis and natural history. Expert review of Gastroenterology and Hepatology (3 (5)), 513-534.

5.Lees, C., Barrett, J., Parkes, M., & J, S. (2011). New IBD genetics : common pathways with other diseases. GUT (60), 1739-53.

6.Podolsky, D. (2002). Inflammatory Bowel Disease . New England Journal of Medicine (347), 417-29.

7.Powrie, F., & Maloy, K. (2003). Regulating the regulators. Science , 299, 1030.