Images of Interest

Gastrointestinal: Mycobacterium avium paratuberculosis and Crohn’s disease

Uncovering the cause of inflammatory bowel disease appears to have become the holy grail of gastroenterology. For Crohn’s disease, the most popular hypothesis is that the disease results from loss of immunological tolerance to bacteria or other microorganisms that are normally present in the bowel lumen. However, several other hypotheses exist including a role for infectious agents such as atypical mycobacteria, Chlamydia species, Listeria monocytogenes, cell-wall deficient Pseudomonas species, Mycoplasma species and a number of viruses including measles virus. Interest in atypical mycobacteria was recently rekindled by observations reported by Dr S Naser and others in an article published in Lancet in 2004. Using a novel method, the group was able to culture Mycobacterium avium subspecies paratuberculosis (MAP) from the peripheral blood in 50% of patients with active Crohn’s disease. This has raised the possibility that MAP may be the cause of Crohn’s disease in humans in a similar way to Johne’s disease in livestock.

Recently, our group has been able to replicate the above findings in a number of patients with active Crohn’s disease. We used Ziehl-Neelsen staining to show what we believe are spheroplast phase forms of MAP (purple spots) within the cytoplasm of a macrophage (Fig. 1) and persisting in modified TB broth after 4 months of culture (Fig. 2). To confirm the identity of these acid-fast organisms, we performed a duplex polymerase chain reaction designed to detect the unique insertion (L1 and L9) sites of the MAP-specific insertion element IS900. The amplified L9 site was sequenced and then compared to a reference DNA of MAP. Alignment of our sequence against the reference sequence showed a perfect match, thereby confirming that the Mycobacterium found in our Crohn’s disease patients was indeed MAP. These observations support the association between MAP and Crohn’s disease. However, further research is required to determine whether MAP is involved in the pathogenesis of Crohn’s disease or whether the detection of MAP in peripheral blood simply reflects translocation of organisms through a leaky epithelial barrier.

Contributed by
RB Gearry,* JM Aitken,1 RL Roberts,1 S Ismail,1 J Keenan1 and ML Barclay*
*Department of Gastroenterology, Christchurch Hospital, 1Southern Community Laboratories, Departments of 2Pathology and 3Surgery, Christchurch School of Medicine and Health Sciences, Christchurch, New Zealand

Figure 1

Figure 2

Contributions to the Images of Interest Section are welcomed and should be submitted to Professor IC Roberts-Thomson, Department of Gastroenterology, The Queen Elizabeth Hospital, Woodville South, South Australia 5011, Australia.
© 2005 Blackwell Publishing Asia Pty Ltd