Autoimmunity and chronic pancreatitis

Autoimmunity was considered important by Jalleh et al (Gut 1993; 34: 1452–7), who proposed an initial immunological hypothesis to explain the development of chronic pancreatitis. This hypothesis was based on the observation that the disease was more common in patients with coeliac disease, which is an autoimmune disorder affecting the small intestine. However, subsequent studies have shown that the prevalence of coeliac disease in patients with chronic pancreatitis is no higher than in the general population, casting doubt on the autoimmune hypothesis.

To address this, several studies have investigated the role of the immune system in the development of chronic pancreatitis. One study found that patients with chronic pancreatitis had a higher prevalence of HLA-B8 and HLA-DR4, suggesting an underlying genetic predisposition. Another study found that patients with chronic pancreatitis had a higher prevalence of autoantibodies against pancreatic tissue, further support for an autoimmune etiology.

Despite these findings, the role of autoimmunity in the development of chronic pancreatitis remains controversial. Some studies have found no evidence of an autoimmune component, while others have found inconsistent results. Nonetheless, continued research in this area is important to further elucidate the potential role of autoimmunity in the development of chronic pancreatitis.

References:


Reply

EDITOR,—Thank you for the opportunity of replying to the letter by Cavallini et al in response to our paper describing the enhanced expression of major histocompatibility complex determinants in chronic pancreatitis. We agree with their suggestion that predisposition of certain subjects to chronic pancreatitis may be congenital in origin. We do not yet have sufficient information, however, to suggest that the abnormal HLA expression we have identified in early chronic pancreatitis may be due, in itself, to a genetic defect. An alternative possibility is that enhanced HLA expression may be an epiphenomenon of the chronic inflammatory process and that the primary aetiology may lie in a genetic defect affecting another, although possibly related, molecule. For example, any of the endogenous molecules participating in the intracellular processing of cellular peptides or proteins (such as the heat-shock proteins) might be affected and hence permit inappropriate targets of immune recognition to develop within pancreatic epithelial cells.

Recently obtained data shortly to be published from our laboratory have clearly shown the enhanced and differential expression of transforming growth factor β1 in human chronic pancreatitis. These findings support the suggestion of Cavallini et al that potent mediators of an evolving inflammatory process probably promote the profound fibrotic characteristic of chronic pancreatitis while they are not aetiological factors. In this respect, we believe that enhanced expression...