Sclerosing cholangitis and hepatic microvascular steatosis in cystic fibrosis and chronic pancreatitis

Liver disease is common in patients with cystic fibrosis, the characteristic histological feature being the presence of inspissated, eosinophilic, periodic acid Schiff positive diastase resistant concretions in small bile ducts and ductules. The precise nature of these concretions is uncertain and the factors responsible for liver disease in only a few patients with cystic fibrosis have not been clearly defined.1 Two recent studies, however, reported that bile duct lesions may be a factor in the development of intrahepatic disease. Gaskin et al found evidence of biliary tract obstruction with stenosis of the distal common bile duct in 36 patients with sclerosing cholangitis in two of 50 patients with cystic fibrosis and hepatic disease; a normal biliary tract was found in 31 control patients with no disease of liver disease. Strandwick et al reported that four of 102 patients with cystic fibrosis had ERCP evidence of sclerosing cholangitis.1 The implications of these findings are that surgical intervention may prevent the onset or progression of the liver disease in cystic fibrosis; the cholangiolar dilatation may be due to bile duct obstruction, and the insulin reductions being a manifestation of the generalised abnormality of exocrine secretion which characterises mucoviscidosis.

Intestinal microsporidiosis in AIDS

We read with interest the letter by Lucas et al regarding the diagnosis of intestinal microsporidiosis in patients with AIDS.2 Although we agree that the organisms appropriately stained microsporidia can be visualised at light microscopy, we are not of the opinion that this can be an absolutely confident diagnosis and feel that electron microscopic examination is essential, particularly in view of the paucity of experience in diagnosing these organisms.

Lucas et al state that, “cases of intestinal microsporidiosis may therefore be confidently identified by light microscopic examination, supplemented by electron microscopy,” but in two of their three cases, which were from dewaxed material reprocessed for electron microscopic examination, only “probable spores” were seen. The fact is that in only one case have they “confidently” diagnosed microsporidia, and this was by electron microscopy, having already suspected the presence of microsporidia by light microscopy. This may seem to be somewhat pedantic but confidence levels among histopathologists are extremely variable.

It is interesting that the microsporidia found by Lucas et al was consistent with Enterocytozoon bieneusi, but this patient was not known to be an AIDS patient, which is in contrast to our case in which both patients were reported to have AIDS. Lucas et al also reported that microsporidiosis may be a marker for impending deterioration of AIDS. We report the occurrence of two patients with AIDS in whom we diagnosed microsporidiosis, one of whom had AIDS-related complex (ARC) and the other had extensive and rapidly progressive Kaposi sarcoma.

Letters to the Editor

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