Editorial

ACUTE PANCREATITIS FOLLOWING DRUG THERAPY

Acute pancreatitis following the use of certain drugs as steroids (Zion et al., 1955), oestrogens, azathioprine, thiazides, ethacrynic acid, frusemide, tetracycline, rifampicin, L-asparaginase, clonidine and calcium has been brought to notice by various workers. The serum amylase level of patients on steroid therapy was found to be higher than their counterparts with the similar primary disease but receiving no steroids. So also the incidence of pancreatitis in these patients was more frequent than the latter (Rifkind 1959). There was no relationship between the duration of therapy and pancreatitis (Openheimer and Boitnott, 1960).

Various animal experiments (Stumpf et al., 1956) give the cause of steroid induced pancreatitis to be inspissation of pancreatic secretions, proliferation of the epithelium of the pancreatic duct resulting in obstruction of the duct, hyperlipidaemia and predisposition to systemic infections. The patient may present with severe abdominal pain accompanied with nausea and vomiting or sudden onset of shock. Prognosis of the condition appears to be bad (Fonkalsrud et al., 1968) possibly due to the underlying lesion.

Oestrogen containing contraceptives have been reported to cause acute pancreatitis especially in women with an existing hypertriglyceridaemia (Bank and Marks, 1970; Zorrilla et al., 1968). The mechanism has been suggested to be embolization of agglutinated serum lipid particles in the pancreatic vessels. This permits pancreatic lipase to act on chylomicrons and release large amounts of fatty acids. These fatty acids damage the capillaries and acini. The patient with oestrogen pancreatitis may have variable degree of abdominal pain, hyperamylasaemia and hyperlipidaemia. The immunosuppressive agent Azathioprine has well established complications as bone marrow suppression, infection and hepato-toxicity. Acute pancreatitis has been reported in patients receiving this drug especially after renal transplantation (Kawanishi et al., 1973; Nogueira and Freedman, 1972). The actual mechanism of azathioprine induced pancreatitis is obscure. It may be activation of a latent infection, production of ischaemia and damage to pancreatic epithelium. Toxic or allergic reaction may play a role (Nogueira and Freedman, 1972). A raised serum amylase level has been found in patients on diuretic therapy (Cornish et al., 1961) especially thiazides. The cause has yet to be determined. Severe abdominal pain associated with nausea, vomiting, fever and shock may be the manifestations. Hyperamylasaemia is found in various degrees.

Hypercalcaemia secondary to hyperparathyroidism, calcium infusion and haemodialysis produces pancreatitis. Acute necrosis of the pancreas due to hypercalcaemia may be the probable cause (Meltzer et al., 1962).

Tetracyclines used over prolonged periods produce pancreatitis. Impaired pancreatic protein synthesis and secretions due to chemotherapy (Tucker and Webster, 1972) has been attributed to be the causative factor. A similar condition is seen with Rifampicin therapy (Mattson 1973).

The various drug therapies leading to acute pancreatitis and the primary disease for which they are being used, may in the course of time produce new grounds for postulating the causative factor of the condition.

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