MANAGEMENT OF ACUTE AND CHRONIC PANCREATITIS

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No specific drug or non-operative procedure has so far been found to be of proven benefit in acute pancreatitis. Results of the clinical trials with numerous therapeutic agents, even on sound physiological principles, have usually been inconclusive and controversial (Soergel, 1978; Warshaw, 1977). Aims of the modern treatment are direct or indirect suppression of exocrine pancreatic secretion with glucagon, nasogastric aspiration, inactivation of circulating and tissue hydrolytic enzymes with Aprotinin and prevention of septic complications with antibiotics.

Supportive measures like maintenance of intravascular volume and relief of pain results in rapid improvement of symptoms in majority of cases (Banks, 1971). Complications like renal failure, local and systemic infection and pulmonary complications should be identified and aggressively treated.

Nasogastric aspiration prevents the release of gastric acid into the duodenum thus causing a reduction in hormonal stimulation of pancrease and its use is rational, simple and safe and almost universally accepted.

Glucagon, given I/M or 1/V, inhibits pancreatic volume and enzyme secretion induced by secretagogues or by pancreatitis. Although recent studies revealed no increased significance of Glucagon compared with asogastric aspiration alone (M.R.C. Working Party, 1977; Olazabal and Fuller, 1978), it reduces mortality if administered at the onset of the disease (Water Worth et al., 1976).

Prophylactic Amicillion produced no benefit in mild acute alcoholic pancreatitis in two prospective clinical trials (Howes et al., 1975; Finch et al., 1976) and their use should be restricted to pancreatic abscess and coexistant obstructive biliary tract disease.

Aprotinin (Trasylol), is a potent inhibitor of hydrolytic enzymes and the kallikrein system. Despite the encouraging reports of clinical success (Trapnell et al., 1974), its value in the treatment of human acute pancreatitis remains uncertain (Soergel, 1978; M.R.C. Working Party, 1977).

Various other drugs and non-surgical procedures like anticholinergics, steroids, heparin, dextran, calcitonin, parenteral hyperalimentation and non operative peritoneal lavage have been proposed but found ineffective. Preliminary results with percutaneous peritoneal dialysis in severe pancreatitis have been encouraging (Ranson et al. ,'1976).

Normally pancreatic enzymes are excreted in great excess and there is a ten-fold reserve for exocrine pancreatic enzyme secretion. In pancreatic insufficiency due to chronic pancreatitis and pancreatic cancer, steatorrhoea and azotorrhoea do not occur until there is 90% reduction in the secretion of pancreatic lipase and tryposin respectively (DiMagno et al., 1973). Lipase insufficiency may occur more rapidly than that of the proteolytic enzymes and hence steatorrhoea is often earlier and more severe problem than azotorrhoea (diMagno et al., 1975). In pancreatic cancer, pancreatic insufficiency occurs only if the distal 60% of the organ is involved and usually coincides with the ductal obstruction in the head of the pancrease (DiMagno et al., 1979). Treatment with oral enzymes is only necessary when there is 90% or more reduction, in the secretion of lipase or trypsin.

Pancreatic enzyme preparations contain either pancreatin or Pancrelipase. Two commercial preparations of Pancreation, Illozyme and Viokase have a maximum amount of lipolytic activity of 3,600 units and 1,646 units per tablet respectively (Graham, 1977) but viokase is cheaper.

Pancrelipase, cotazyme contains 2,014 units of enzyme activity per capsule. The enteric A coated preparation, Pancrease has only been found effective in the treatment of steatorrhoea in children with cystic fibrosis (Khaw et al., 1977) and is less effective than viokase in chronic pancreatitis (Regan et al., 1977).

For efficient lipolysis, the maximum amount of lipase to be delivered into the duodenum post-prandially has been calculated to be at least 8,000 units per hour. Large doses of the enzyme are required also because of acid-peptic inactivation of the ingested enzymes and acid precipitation of bile acids. Both the hourly and the prandial dosage schedules are equally good in abolishing azotorrhoea in majority of the patients and markedly reducing the steatorrhoea (Kaiser et al., 1968). Eight tablets of Viokase with each meal (consisting not more than 25G of fat) are preferred.

Combination of cimetidine with Viokase results in significantly abolishing or reducing steatorrhoea because it maintains the gastric and doudenal pH above 4 (Regan et al., 1977).

Pancrease showed complete abolition of steatorrhoea in two patients who had very high acidic pH in the upper intestine. It is found to be more effective in conditions where post prandial gastric and duodenal pH remains per- sistently acidic such as cystic fibrosis in children (Cox and Isenberg, 1978). Pancrease would not he effective in chronic pancreatine because the initial post prandial increase in pH above 5 would liberate the enzyme with later irreversible inactivation as the pH decreases below 4.

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