The management of pancreatic ascites and pancreaticopleural effusion

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Abstract

Pancreatic ascites and pancreaticopleural effusion result from a complete or partial main pancreatic duct disruption or from a pseudocyst rupture with releasing of pancreatic juice into the surrounding tissues.

The treatment requires to minimize pancreatic secretion, to favor pancreatic juice drainage into the digestive tract and to restore main pancreatic duct disruption. This can be achieved by endoscopic approach in more than 90% of the patients. (Acta gastroenterol. belg., **2000**, 63, **269**).

Key words : pancreatic ascites, pancreaticopleural.

Pancreatic ascites (PA) and pancreaticopleural effusion (PE) share common pathogenesis, diagnosis and treatment. The present discussion excludes fluids collections associated with acute episodes of pancreatitis or peritonitis carcinomatosa.

The pathogenesis results from a complete or partial main pancreatic duct (MPD) disruption or from a pseudocyst rupture with releasing of pancreatic juice into the surrounding tissues (1). PA arise from anterior MPD disruption. Pleural or mediastinal effusion arises from posterior and retroperitoneal MPD disruption.

The etiology of PA is most often alcoholism and very rarely trauma, biliary pancreatitis, fine-needle biopsy,... The pancreatic damage can be acute with severe pancreatitis, necrosis, phlegmon, pancreatic fluid collection and pseudocyst formation or chronic with MPD stenosis or proximal obstructive calculus (1).

High amylase and protein levels (> 3 g/100 ml) in ascitic or pleural fluids sign the diagnosis of PA or PE. Usually serum amylase is elevated and serum albuminemia levels are depressed. ERCP, CT or NMR findings show pancreatic abnormalities in more than 80% of the cases, pseudocyst in more than 80% of the cases, pancreatic calcifications and MPD dilatation (2).

The management and therapy require to minimize pancreatic secretion by low-fat diet or total parenteral nutrition and by the use of somatostatin analogues. The non-interventional management includes paracentesis and thoracocentesis. Diuretics, atropine and some other original attitudes like pancreatic irradiation, peritoneojugular shunt, peritoneal lavage are of little interest. Medical management is reported to be successful in 20 to 50% of patients but PA recurrence reaches 15% (1.2). These results justify a more efficacious treatment.

The interventional therapy should aim to favor pancreatic juice drainage into the digestive tract and to restore partial or complete MPD disruption. This management, endoscopic or surgical, should preserve the pancreatic function (3,4,5) :

- Sphincterotomy provides access to the MPD and reduces the prancreatico-duodenal pressure gradient.
- Drainage of an obstructed MPD is achieved by stenosis dilatation, nasopancreatic tube and MPD stenting.
- Disruption of the MPD needs a pancreatic stent that bridge duct rupture. This is rarely possible in complete duct rupture.
- Pseudocyst are treated by transpapillary, transgastric or transduodenal drainage.

The control of PA or PE can be obtained by endoscopic therapy in more than 90% of the patients. So a surgical approach is considered only in a few cases : failure of endoscopic drainage or pancreatic lesions mainly located in the tail of the gland. Surgical treatment is based on pseudocyst or MPD drainage by pancreaticojejunostomy or pancreatic tail resection with preservation of the spleen.

In conclusion, an interventional management of PA and PE is often required. This can be safely performed by endoscopic approach and a surgical therapy is rarely mandatory.

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