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Case reports

Non-traumatic rupture of the superior mesenteric vein with hemoretroperitoneum

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Summary. A case of rupture of the superior mesenteric vein with hemoretroperitoneum is described. The autopsy revealed the presence of an acute pancreatitis and thrombosis of the superior mesenteric artery. From the histological findings it appeared that the acute pancreatitis had damaged the venous wall from the outside and that the portal system overload, caused by the superior mesenteric artery thrombosis and the anomalous termination of the inferior mesenteric vein, acted from the inside of the vein causing its rupture. To our knowledge this is the first report of a rupture of the superior mesenteric vein caused by acute pancreatitis and resulting in hemoretroperitoneum.

Key words: Superior mesenteric vein – Rupture – Hemoretroperitoneum – Pancreatitis

Zusammenfassung. Ein Fall einer Ruptur der vena mesenterica superior mit nachfolgendem Retrohämoperitoneum wird beschrieben. Die Autopsie deckte eine akute Pankreatitis auf und eine Thrombose der Arteria mesenterica superior. Aus den histologischen Befunden ist ableitbar, daß die akute Pankreatitis die Wand der Vene von außen geschädigt hatte und daß die Überladung Pfortadersystems, welche durch die arterielle Thrombose und eine Anomalie in der Einmündung der unteren Mesenterialvene von der Innenseite her gewirkt hat, die Ruptur verursachte. Nach unserer Meinung ist dies der erste Bericht einer Ruptur der oberen Mesenterialvene, hervorgerufen durch eine akute Pankreatitis und resultierend in ein Retrohämoperitoneum.

Schlüsselwörter: Obere Mesenterialvene – Ruptur – Retrohämoperitoneum – Pankreatitis

Introduction

Pathological conditions of the superior mesenteric vein (SMV) are relatively rare and are mainly represented by thrombosis [1–3] or traumatic rupture [4–9] sometimes of a homicidial nature [13]. A case of rupture of the SMV with hemoretroperitoneum in an old woman with acute pancreatitis and thrombosis of the superior mesenteric artery (SMA) is described. The rarity of this condition makes the case interesting also for the pathogenetic problems it poses.

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Case report

An 83-year-old cardiopathic woman was admitted to hospital suffering from abdominal colicky pain. Clinical examination showed the presence of ileus. Echography revealed a calculus in the common bile duct. The laboratory examinations revealed high blood glucose levels (8.3 mmol): the amylases were normal. The following day the patient's condition worsened and the onset of increasing hypotension preceded death. The diagnosis was that of "cardiogenic shock". The laboratory examinations carried out shortly before death revealed leukocytosis (15.000 mm^3), a decrease in the hematocrit to 24.7% while hyperglycemia had reached 20.6 mmol.

Postmortem examination

The corpse was in accordance with that of an old woman of about 80 years of age, with no external signs of violence or trauma. The skin was extremely pale. The heart was hypertrophic and showed an old scar in the diaphragm wall of the left ventricle. There was severe atherosclerosis. The abdominal cavity contained approx. 50 ml serosanguineous fluid: the intestinal coils were pale and distended with gas. The retroperitoneal tissue was abundantly filled with blood which diffused into the mesentery. After having checked for a rupture of an abdominal aortic aneurysm, the SMA was opened and was found to contain many partially organized thrombi beginning at the origin of the vessel (Fig. 1). The SMV showed a vertical breach (12 mm × 5 mm) of its posterior wall just above the uncinate process of the pancreas (Fig. 1), which communicated with the retroperitoneal hematoma. The inferior mesenteric vein (IMV) terminated in the SMV, about 4 cm below the rupture. The bile ducts were en-

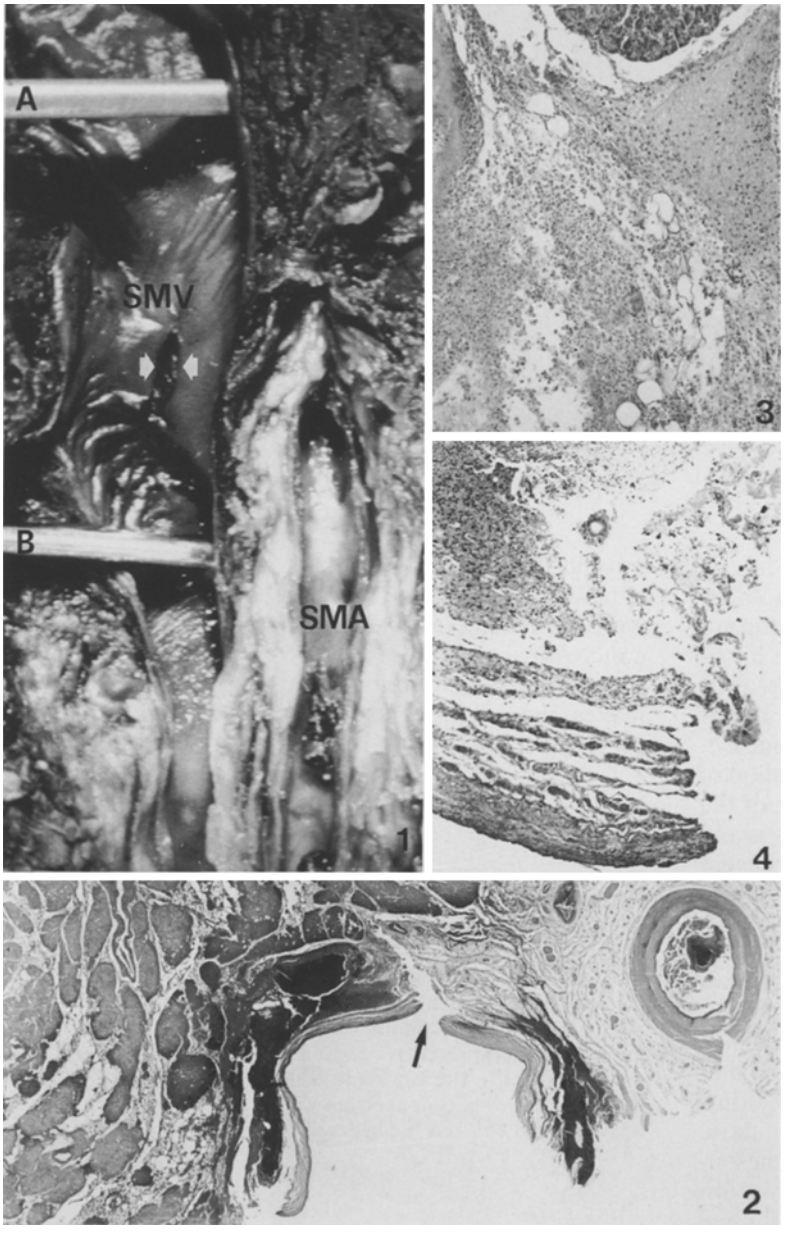

Fig. 1. The opened portal system with the upper probe introduced into the splenic vein *(A)* and the lower into the inferior mesenteric vein *(B)*. The *arrows* indicate the vertical breach in the superior mesenteric vein *(SMV)* wall. The thrombosis of the superior mesenteric artery *(SMA)* is also recognizable

Fig. 2. Transverse section at the level of the SMV rupture *(arrow)*. H-E × 3

Fig. 3. The pancreas shows areas of cytosteatonecrosis and inflammatory lymphogranulocytic infiltration partially masked by the hemorrhage. H-E × 16

Fig. 4. The right edge of the SMV breach appears irregular with slight inflammatory infiltration. The latter, represented by lymphocytes and granulocytes, is marked in the perivenous tissue indicating that the damage to the vascular wall was caused by continuation of the pancreatic inflammatory process. H-E × 16

larged but there were no calculi. The pancreas was increased in size but steatonecrosis of the peritoneal fat was not found. An investigation for calculi in the feces was not performed. Histological examination showed myocardiosclerosis with a postinfarction scar in the left ventricle and slight hepatic cholestasis. The pancreas showed marked autolysis but areas of cytosteatonecrosis and inflammatory lymphogranulocytic infiltrates were recognizable (Fig. 3).

Multiple transverse sections at the level of the rupture of the SMV (Fig. 2) showed a reduction of smooth muscle bundles in the media. The edges of the breach were irregular with moderate inflammatory infiltrates (Fig. 4). The latter was marked in the neighbouring perivenous tissue and was composed predominantly of lymphocytes and granulocytes.

Discussion

The pathology of the mesenteric vessels mainly relates to the SMA, where occlusions of embolic or thrombotic nature occur relatively frequently. The SMV is mainly involved in alterations of the portal circulation. Moreover examples of thrombotic occlusions [1–3] and lacerations of this vessel resulting from penetrating and non-penetrating traumas [4–9] can be found in the literature. The majority of mesenteric lacerations have occurred in car or aircraft accidents and have been attributed to the lap-strap of seat belts [4, 5] or to the rim of a steering wheel [10, 11]. The mechanism of these injuries may be either compression between an external force and the lumbar spine or shearing at the root of the small bowel mesentery, with avulsion of the superior mesenteric vessels [4, 10, 12].

Mesenteric tears with fatal bleeding due to sagittal compression have also been described in homicide victims with high blood alcohol concentrations, supporting the assumption that this kind of lesion is caused by direct rather than indirect trauma [13].

The case observed here shows a rupture of the SMV in a person with no history of trauma. The symptoms were initially indicative of biliary colic and later envolved towards hemorrhagic shock. Besides the hemoretroperitoneum from the rupture of the venous wall, the autopsy revealed thrombosis of the SMA, a myocardial postinfarction scar and signs of cholelithiasis which had resolved itself with the expulsion of the calculus. An obscure aspect of this case is the absence of entero-mesenteric infarction due to the presence of partially organized thrombosis in the SMA.

The histological examination revealed localized signs of acute pancreatitis, even after the onset of autolysis of the gland. The probable retroperitoneal steatonecrosis may have been masked by the extensive hemorrhage.

The most important aethiological factors causing rupture of the SMV are biliary calculosis and acute pancreatitis. The biliary calculosis, with successive expulsion of the calculus into the intestinal lumen, may have caused the pancreatitis by the reflux of bile into the Wirsung's duct.

The SMV was damaged in its tract between the body and uncinate process of the pancreas by continuation of the pancreatic inflammatory process which caused the weakening and successive rupture of the wall with consequent hemoretroperitoneum. The absence of intestinal infarction could have been due to the discharge of blood into the retroperitoneal tissue after rupture of the vein. On the other hand, the portal system overload, linked with the arterial thrombosis which removed the propulsive action of the heart ("vis a tergo"), mechanically favoured the rupture of the already vessel wall.

The rupture of the venous wall was the result of 2 concomitant processes: on the one hand, the acute pancreatitis, which by continuation of the inflammatory process to the vascular wall created the pathological substrate acting from outside the vessel, and on the other hand the portal system overload acting from the inside of the vessel represented the triggering factor.

Moreover in the case in question the IMV ended in the SMV, about 4 cm below the laceration. Such an anatomical variation [14] could have aggravated the effects of a venous overload at the level of the altered vascular wall.

In addition to the possibility of the reflux of bile causing the pancreatitis the thrombosis at the origin of the SMA blocked the flow of blood into its first collateral branch, the inferior pancreaticoduodenal artery. The consequent tissue ischemia is at present considered to be one of the main aethiological factors in acute pancreatitis [15]. To our knowledge this is the first report of rupture of the SMV due to acute pancreatitis and causing hemoretroperitoneum.

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