Vascular Complications – Poor Prognostic Factors in Severe Acute Pancreatitis

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Background: Various complications occur after the onset of acute pancreatitis. Approximately 9–11% of the patients with severe acute pancreatitis may develop vascular complications such as haemorrhage, ischaemic visceral lesions, tromboses on the portal venous system and formation of aneurysms.

Aim: To determine the incidence of these complication and to analyze the therapeutic approach for these patients.

Method: We realized a retrospective study on 279 patients with severe acute pancreatitis (SAP), defined by the Atlanta criteria. In this group we annalized the incidence of vascular complications, the therapeutic methods for their solving and the risk for developing complications and mortality.

Results: The incidence of vascular events was 14.33%. Hemorrhagic complications had a higher incidence and were present in 32 patients (80%). Only 8 patients (20%) presented major ischaemic events. The most commonly involved organ in ischaemic lesions was the colon.

Conclusions: Vascular complications, although rare (leading to an increased morbidity and mortality), may jeopardize the patients’ life. Their diagnosis and treatement becomes possible only by the clinician vigilance on their potential occurence using modern investigitive methods

Keywords: severe acute pancreatitis, colic ischaemia, thrombosis, haemorrhagia

Introduction

Severe acute pancreatitis (SAP) is recognized as an aggressive form of disease characterized by the emergence of local and general complications. Involving both local – (peri-pancreatic necrosis and pancreatic enzymes overflow) and systemic manifestations (hypovolemia, ischemia, bleeding disorders and impaired microcirculation), it may lead to major vascular complications in the digestive tract. The incidence of this type of complications is not well documented yet, reports suggest an incidence ranging from 9-11% and it is associated with an increased mortality [1]. These lesions can be classified as: bleeding (sever upper gastrointestinal bleeding, intracystic bleeding or vascular erosion in areas of necrosis) and ischemic-thrombotic arterial or venous involvement with (thrombosis, ischemia) [2,3]. If the overall mortality in SAP ranges between 15–25%, it reaches 20–50% for ischemic vascular complications [4,5].

Material and method

We realized a retrospective study on 1159 patients with acute pancreatitis (AP) hospitalized between 1995 and 2009 in the 1st Surgical Clinic of Mures Emergency County Hospital. From this group, we selected a subgroup of 279 patients with SAP, defined by the Atlanta criteria: Ranson score > 3 pts, APACHE II> 8 pts, the presence of organ failure or local complications. In this group we analized the incidence of vascular complications, the therapeutic methods for their cure and the risk for developing complications and mortality. Data been scarce, we did not adventure into any sophisticated statistics.

Results

In 40 patients from 279 patients with PAS were encounted vascular complications, which corresponds to an incidence of 14.33%. Hemorrhagic complications had a higher incidence and were present in 32 patients (80%). Bleeding represented the indication of surgical treatment for SAP in 9 cases (22.52%); 6 of them were upper gastrointestinal bleeding and 3 coresponded to severe haemorrhage due to the erosion of a major pancreatic or peripancreatic blood vessel into the pancreatic pseudocyst or into an infected necrosis area. These accidents occurred in the early phase of SAP in 3 cases with a mortality of 33.3% or late stage in 6 cases with a mortality of only 16.66%. Haemorrhage consecutive to surgical intervention was recorded on 23 patients (57.5%), 16 of them underwent early surgical procedures ( in the first three weeks after the onset of SAP with a mortality of 40% (six deaths), compared to a lower mortality – 28.57% – recorded in 7 patients with delayed surgical treatment. The overall mortality in haemorrhagic complications subgroup was 18.75%. Surgical treatment consisted in extensive necrosectomy, identifying the bleeding source followed by haemostasis. In two cases splenectomy was associated. Recurrence of bleeding was recorded in four cases, but with low intensity and did not require reinvention. Ischemia was the main mechanism in only 8 patients (25% of patients with major vascular implications). The site most commonly involved was the colon, in 4 cases with extensive necrosis on the transverse and left colon, in 3 of them subtotal colectomy was performed and extended left hemicolectomy (one case). Death occurred in 2 of these patients. Other organs affected were: the
duodenum in 3 cases (one death) and the common bile duct in two cases (parcelling necrosis in association with coledocholithiasis). Overall mortality recorded in the ischemic complications subgroup was 37.5%.

Discussions
Although rare, vascular complications subsequent to SAP are dangerous and potentially lethal [6]. The survival of these patients with pancreatitis and vascular complications depends on early diagnosis and adequate treatment. The pathogenesis of haemorrhagic complications is multifactorial. In the our study frequency of severe bleeding was 11.46% while the reported incidence varies from 1.2% to 14.5% in literature, being correlated with the severity of the disease [6,7,8,9]. The pathogenic mechanisms incriminated could be the direct injury to the vascular wall and the local process, the proteolytic and lipolytic enzyme action [10], the aggressive surgical maneuvers, particularly for the patients with early surgical debridement in the inflammatory phase of SAP [9]. Our study provided similar results (Figure 1).

Postoperative haemorrhagic complications could be caused by vascular denudation. Long term contact of these structures with the drains or gauzes used for drainage or packing can lead to injury of the vascular wall, even to rupture [10,11]. Late bleedings occurred at 2-3 weeks of the onset of AP and they could be caused by the rupture of an pseudoaneurysm into pancreatic pseudocyst, into the peritoneal cavity or into a hollow organ (stomach, jejunum, colon, the duct of Wirsung) [10,12]. The frequency of these injuries ranges from 3.5 to 10% [11]. In decreasing frequency, the arteries involved were: the splenic artery (40%), gastroduodenal (20%), pancreaticoduodenal (20%), gastric coronary artery (5%), hepatic artery (2%). The size of the pseudoaneurysm is not determinant for risk of rupture [10,12,3,13]. Gastro-intestinal bleedings (haematemesis and/or melena) could be caused by the association of peptic ulcer, portal hypertension and esophageal varices. Clinical diagnosis is marked by the signs of hemorrhagic shock, haematemesis, melena, or bleeding on drains. Flati et al. report that massive or cataclysmical bleeding was noticed in 7.5% of the patients with SAP [10]. Immediate surgery is considered the „gold standard” for haemodynamic unstable patient. Surgical procedures consist in vascular ligation/suture proximally and distally associated with necrosectomy [3,6,13]. Patients who are haemodynamically stable and who have angiographic evidence of bleeding can be treated with transcathether embolisation [12]. The ischemic complications of adjacent viscera are caused by the thrombosis of the large vessels such as the colic arteries and the superior mesenteric artery or by a marked disruption of the microcirculation caused by sepsis, systemic inflammatory response, disorders of coagulation and compartment syndrome [7,14]. Ischemic lesions were observed in 2.8% of patients with PAS (in the literature 1–3%) [9,13]. The colon is particulary involved and the mortality recorded is 50% higher than overall mortality group with PAS (28%) and the subgroup with bleeding complications (25%). The severity of this complication is augmented by the occurrence of infection by bacterial translocation, necrosis and intestinal leakage. Partial bowel resection is the only solution, three out of four patients with colic necrosis underwent either subtotal colectomy or left enlarged hemicolecotomy (Figure 2).

Conclusions
Vascular complications, although rare, (leading to an increased morbidity and mortality), may jeopardize the patients’ life. Our study suggests that the diagnosis and treatment becomes possible only by the clinician vigilance on their potential occurrence using modern investigative methods (CT, MRI, angiography selective Doppler ultrasonography) to document these complications and even solving them. Early diagnosis in hemodynamic stable patients and clear indication of the source of bleeding may benefit from transcathether embolization and percutaneous injection of thrombin. In our opinion the gold-standard remains surgical, resumed to simple homeostatic suturing or pancreatic resection for bleeding, more or less for extensive ischemic lesions.

References

![Fig. 1. Vascular complications after early vs delay surgery](image1)

![Fig. 2. Incidence and mortality of vascular complications in SAP](image2)
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