Introduction

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In 80% of cases, acute pancreatitis occurs in a mild form; only 20% of patients develop the severe (necrotizing) form with organ complications.

Typical systemic complications of necrotizing pancreatitis are pulmonary, circulatory and kidney failure, and in 30–60% of cases, an infection in the form of a superinfection of necrosis occurs. Necrosis of the pancreas and neighboring tissue as well as necrotic infection are the two main risk factors of the disease [1].

Therapy of complications of acute pancreatitis focuses on symptomatic measures as a causal approach is not yet available.

For the past 10 years, therapy of necrotizing pancreatitis has mainly concentrated on a conservative/intensive-care-medicine-based approach [1]. Today, most patients presenting with the severe form can be treated noninvasively; this is especially true for patients with sterile organ necrosis. Infected pancreas necrosis is the only form where operation is indicated, an intervention whose urgency and necessity is internationally accepted.

New laparoscopic procedures in necrosectomy have recently been developed [2], which may be used to supplement standard open laparotomy in the future. The role of interventionally (radiological/endoscopic) controlled drainages of infected necrosis, however, is still controversial.

The prevention of an infection of necrosis has been ascertained in extensive randomized studies, even though

a substantial risk of multiresistant germs and fungal infections has to be feared when antibiotics are administered indiscriminately [3].

The future of the treatment of organ failure (with potential mortality) in severe acute pancreatitis lies in the precise analysis of triggering factors and their early regulation in the course of the disease. Medicinal prevention/reduction of pancreatic parenchymal necrosis will play a major role in this approach. Analysis of genetic determinants of hyperacute versus low reactions in the patient organism seems also to be of some interest. It is a well-known clinical fact that patients with a similar extent of pancreatic necrosis react systemically very differently.

Today, as in the past, complications after pancreas surgery are frequent and dreadful. They originate mainly from the pancreatic anastomosis, which is very difficult to perform, especially if the remaining portion of the pancreas is soft. The perioperative use of somatostatin or its analogues to inhibit exocrine pancreas secretion has been shown in controlled trials to be effective regarding the reduction of complications of the pancreatic anastomosis, especially fistulas [4]; however, this step is not sufficient on its own.

In the future, the most effective way to reduce complications and perioperative mortality is certainly to concentrate pancreas surgery in specialized centers with an adequate case load [5, 6].

The publication at hand summarizes, in an exemplary fashion, the complications occurring in acute pancreatitis and after pancreas surgery. Renowned authors have written down their experiences on the occasion of the ISDS/EDS Postgraduate Course which took place during the 9th United European Gastroenterology Week, Amsterdam,

October 2001. The editor highly recommends the controversial accounts of the contemporary therapies of both disease forms, which are for the most part not standardized. The future will witness the necessary standardization in surgery by means of evidence-based findings.

References

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