Eur Spine J (2000) 9 : 171–173

CASE REPORT

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Acute pancreatitis complicating anterior lumbar interbody fusion

Received: 17 May 1999
Revised: 6 December 1999
Accepted: 16 December 1999

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Abstract Postoperative pancreatitis may occur following surgery in regions remote from the pancreas and the biliary tree. Though uncommon, it carries a high mortality rate. Pancreatitis complicating spinal surgery is extremely rare. This report describes a case of acute pancreatitis following an anterior lumbar interbody fusion and discusses the possible mechanisms of pancreatic cellular injury.

Key words Anterior interbody fusion · Lumbar vertebrae · Pancreatitis · Spinal fusion

Introduction

Acute pancreatitis can evolve into a life-threatening systemic disease with multisystem organ failure. Postoperative pancreatitis carries an alarmingly high mortality rate of 10–45% [3, 14]. Fortunately, in contrast to alcoholic and biliary etiologies, postoperative pancreatitis accounts for less than 1% of all cases of pancreatitis [17]. Postoperative pancreatitis has been observed following a variety of different operations [15], but only a few instances have been reported following spinal surgery [4, 6, 8]. No such complication has been reported following anterior lumbar interbody fusion (ALIF). This report illustrates such a case and discusses the possible mechanisms of pancreatic cellular injury.

Case report

A 60-year-old woman presented with a history of chronic low back pain and a 6-month history of exacerbation of her back pain and left lower extremity pain radiating in the L5 dermatomal distribution. Her symptoms did not improve with conservative measures that included weight loss, physical therapy, and multiple lumbar epidural steroid injections. She had no complaints of weakness, numbness, or bowel or bladder dysfunction. She smoked half a pack of cigarettes per day and did not consume alcohol. Her past medical history was significant for rheumatic fever, mitral valve disease, and peripheral vascular disease. Her medications included enalapril, furosemide, and a potassium supplement.

Clinical examination revealed normal motor, sensory, and reflex functions with a negative straight leg raising test bilaterally. She had a stooped posture with restricted bending in all directions. Plain film X-rays of the lumbar spine revealed grade I spondylolisthesis at the L4-L5 level that measured 11 mm in flexion that reduced to 6 mm in extension. Magnetic resonance imaging (MRI) scans of the lumbar spine demonstrated mild lumbar spinal stenosis from L3 to L5 and degenerative disc disease, especially pronounced at L4-L5. A diagnosis of degenerative disc disease with mechanical instability at the L4-L5 level was made. Preoperative laboratory examinations were all within normal limits. Preoperative cardiac evaluation demonstrated well-compensated mild mitral regurgitation without congestive heart failure. The patient did not have any arrhythmia or evidence of coronary artery disease.

Operative findings

In view of the minimal posterior element disease and insignificant neural compression, an anterior fusion procedure was considered

optimal. The patient underwent an ALIF at the L4-L5 level through a left lateral retroperitoneal approach as described previously [10]. The exposure of the interspace and discectomy was uneventful. Two threaded bone dowel allografts, packed with autologous cancellous bone from the iliac crest, inserted straight anteroposteriorly, served to keep the interspace distracted. The procedure was uneventful and lasted less than 3 h with an estimated blood loss of 300 ml. The patient remained hemodynamically stable throughout the procedure.

Postoperative course

In the immediate postoperative period, the patient was awake with no new neurological deficits. Postoperative pain control was achieved with patient-controlled, on-demand morphine infusion for the first 2 days and with non-narcotic analgesics from the 3rd postoperative day. Other medications used in the perioperative period included her preoperative cardiac drugs, cephalosporin antibiotics, prochloperazine for nausea, and stool softeners. Steroids were not administered at any time. The patient developed nausea, vomiting, and abdominal distension on the 3rd postoperative day that was initially ascribed to postoperative ileus. Persistence of these symptoms led to a detailed hepatobiliary work-up, including hepatic, pancreatic, and cardiac function tests as well as a CT scan of the abdomen. The serum amylase level increased to four times the normal range (peak 364 IU, normal range 0–88 IU) and the serum lipase peaked at 1270 IU, more than six times the normal (range 30–190 IU). There were mild elevations in the lactate dehydrogenase, aspartate aminotransferase, alkaline phosphatase, and serum glucose, with normal bilirubin, alanine aminotransferase, calcium, blood urea nitrogen, and creatinine levels (3 of Ranson's criteria at diagnosis and none at 48 h). The patient did not suffer hypoxia or hypocalcemia. A CT scan of the abdomen (Fig. 1) revealed swelling of the tail of the pancreas and a small collection of free fluid in the pelvis, in addition to the expected changes at the operated site (Balthazar grade B). Further, the bile ducts were normal and there was no evidence of chronic pancreatitis. A diagnosis of mild acute pancreatitis [1] was made and the patient was treated with nasogastric suction, intravenous fluid therapy, and supportive measures. Her preexisting cardiac condition complicated fluid management, necessitating placement of a pulmonary artery catheter and intensive care monitoring for several days. She improved gradually over the next 10 days and all her laboratory values returned to normal levels (amylase 54 IU, lipase 142 IU) by the 3rd postoperative week. She has been followed for 1 year and has not had any recurrence of abdominal symptoms. More importantly, she underwent mitral valve replacement surgery in the interim, without any complications. She has had an excellent result with regard to her fusion operation, with complete resolution of her preoperative symptoms and radiographic fusion.

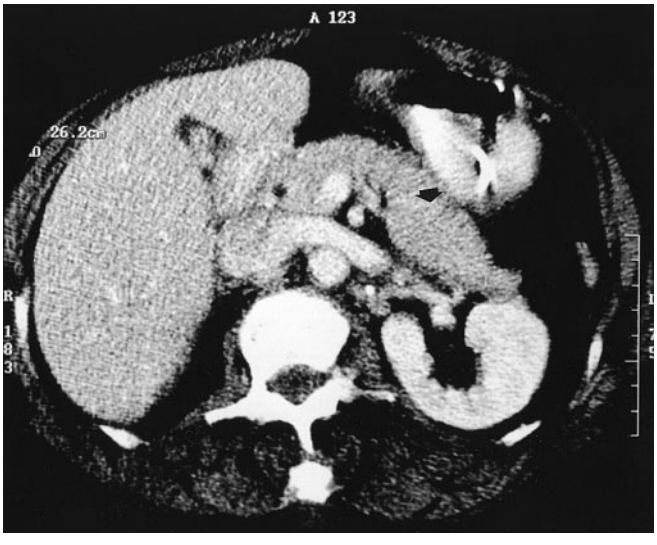

Fig. 1 Intravenous contrast-enhanced CT scan of the abdomen showing a swollen tail of the pancreas (*arrow*)

Discussion

Acute pancreatitis as a complication of an ALIF has not been previously reported. This is our first and only case of such a complication in a series of more than 100 ALIF operations performed over a 3-year period. Among the different abdominal and general surgical complications after an ALIF procedure, vascular injury and sympathetic nerve dysfunction account for the majority [10]. Postoperative ileus after retroperitoneal exposure usually lasts 2–3 days [13]. Prolonged ileus beyond 4 days was seen in only 5% of cases in a recent series of patients undergoing ALIF [10]. In addition to the more common causes of prolonged ileus, such as electrolyte imbalance and retroperitoneal hematoma, the patient presented here demonstrates that pancreatitis should be considered in the differential diagnosis.

Although postoperative pancreatitis has long been recognized following gastric and hepatobiliary surgery, it is being increasingly reported following surgery at sites remote from the pancreas. White et al. [15] reported 70 cases of acute postoperative pancreatitis, representing 9.5% of all cases of pancreatitis diagnosed in their surgical unit over the same time period. Seventeen of these patients had undergone procedures at sites remote from the pancreas, including lower abdominal surgery in ten, transurethral prostatic resections in three, and mastectomy, parathyroidectomy, hip prosthesis, and laminectomy in one patient each. More recently, pancreatitis has been reported following cardiopulmonary bypass surgery and spinal surgery [3, 4, 6].

In many of these postoperative cases of pancreatitis, the exact cause is not clear. Reporting a case of pancreatitis following posterior lumbosacral fusion surgery, Curtin et al. [4], felt that suboptimal positioning of the patient might have caused trauma to the pancreas. Korovessis et al. [6] were unable to decipher a definite cause in their case but felt that prolonged surgical time might have been a contributory factor. Another factor that could have played a role in their case was the use of hypotensive anesthesia, as the pancreas is known to be susceptible to ischemic injury [11]. Leichtner et al. [8] found that intraoperative blood loss was significantly higher in patients who developed postoperative pancreatitis following scoliosis surgery but there was no demonstrable relationship to hypotension.

A variety of causes have been proposed to explain the occurrence of postoperative pancreatitis [14, 15]. Drugs have long been regarded as an important cause of pancre-

atitis. However, this traditional view has been questioned [12] and the true incidence of drug induced pancreatitis is estimated to be between 1 and 2% [7, 16]. The responsible drugs have been categorized based on definite, probable, and possible causality. A careful review of all drugs received by our patient during her hospitalization revealed three incriminating medications: enalapril, nonsteroidal anti-inflammatory drugs (NSAIDs), and furosemide, the former two in the probable and the latter in the possible causality category [16]. However, unlike all reported cases of enalapril-related pancreatitis, our patient had been continually on this medication for several years [16]. Most reported cases of NSAID-related acute pancreatitis followed intoxication. The mechanism of diuretic-induced pancreatitis includes induction of hypercalcemia and hyperparathyroidism [12], neither of which was apparent in our patient. Others have pointed out the role of anesthetic [9] and metabolic [14] factors. Our patient continued taking her regular medications and was exposed to an identical slew of anesthetic drugs during her cardiac surgery without any complications.

Direct surgical trauma to the pancreas probably plays a significant role in abdominal operations in and around the pancreas and the biliary tree [15]. Among operations remote from the region, perioperative hypotension and decreased cardiac output were among the important risk factors for the postoperative development of pancreatitis in one study [3]. The exact cause of pancreatitis in our case is not clear. Possibly, suboptimal placement of the self-retaining retractor could have caused direct traumatic injury to the pancreas.

Postoperative pancreatitis, especially when it is caused by direct trauma to the organ, is associated with an increased incidence and severity of complications [2]. Furthermore, the difficulty of diagnosing pancreatitis in the postoperative period may be a factor in the delay in diagnosis and its effect on outcome [5, 14]. These observations from the literature taken together with the significant blood loss in some spinal procedures, use of hypotensive anesthesia, and increasing popularity of anterior spinal approaches, should serve to warn all spinal surgeons to be vigilant to the possibility of postoperative pancreatitis in every case of prolonged postoperative ileus. Postoperative pancreatitis can assume life-threatening consequences in a patient with other systemic disease.

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