Management of persistent pancreatico-peritoneal fistulae by endoscopic transpapillary stenting

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Introduction

Management of persistent pancreatico-peritoneal fistula due to pancreatic duct disruption is a challenging clinical problem. Ductal disruption may occur as a sequel to acute severe pancreatitis, chronic pancreatitis or following trauma to the pancreas [1]. Resultant fluid collection in the lesser sac may leak into the pleura or to the peritoneal cavity causing pleural effusions or ascites. Many such fistulae heal spontaneously [2]. Persistent fistula implies unlikely spontaneous closure and significant disruption of the pancreatic duct. Restoration of continuity of the duct in such patients is essential to heal the fistula and prevent the occurrence of two disconnected segments – “dislocated duct syndrome” [3].

Endoscopic transpapillary pancreatic stent placement traversing the site of ductal disruption is a versatile, minimally invasive technique in the treatment of such fistulae. We report two cases managed successfully with transpapillary stenting.

Patient 1

A 23-year-old youth underwent an emergency laparotomy for acute abdomen. Free intra-peritoneal fluid, omental fat necrosis and oedematous pancreas suggested a diagnosis of acute pancreatitis. His condition continued to deteriorate and a second laparotomy was done 5 days after the first. An inflammatory mass suggestive of pancreatic phlegmon was found involving pancreas, stomach and transverse colon. An abscess found in the lesser sac was drained. Two tube drains were placed in the lesser sac. Because of slow clinical improvement with persistently heavy output from the drains, he was transferred for specialised treatment. Investigations revealed a drain fluid amylase level of 1396 units/l and drain output more than 200 ml on admission. Culture of the drain fluid yielded a heavy growth of coliform bacteria. Arterial blood gas analysis showed metabolic acidosis. He was managed conservatively. A week later, the drains fell leaving peritoneo-cutaneous fistulae.

On the third week of his illness, he complained of sudden severe central chest pain associated with breathlessness. Chest xray confirmed the presence of bilateral pleural effusions. Pulmonary embolism was excluded with a ventilation-perfusion scan. High amylase levels were noted in pleural aspirate.

Endoscopic retrograde cholangio-pancreatogram (ERCP) was performed to rule out pancreatic ductal disruption. Cannulating the oedematous deformed ampulla revealed a disrupted mid-pancreatic duct with a contrast leak. A 7 F, 7cm plastic stent (Microvasive – Boston Scientific) was positioned to bridge the gap. Complete cessation of the fistulous output was observed with a marked improvement of the general condition of the patient by the fifth day of stenting. The stent was retrieved endoscopically at 8 weeks and the repeat pancreatogram showed ductal continuity. Patient remains well 3 years after the procedure.

Patient 2

A 17-year-old schoolgirl was admitted with a 3-day history of generalised severe abdominal pain. Her serum amylase on admission was 1344 units/l. Ultrasound and the CT scans did not reveal gallstones. She was treated conservatively. Her condition deteriorated and repeat CT scan demonstrated a sub-hepatic fluid collection which was drained under ultrasound guidance. In spite of two aspirations, she continued to accumulate fluid and a drain was placed percutaneously. Her general condition continued to deteriorate. Pancreatic ductal disruption, as a consequence of acute severe pancreatitis, was suspected. ERCP confirmed a mid-pancreatic duct damage with a contrast leak. Disrupted main pancreatic duct was bridged by placing a 7 F, 7cm plastic stent (Microvasive – Boston Scientific), following which her condition improved dramatically. The stent was extracted at 12 weeks. Pancreatogram demonstrated patency of the duct. She remains well after one year.

Discussion

Pancreatic ascites is rare. It may occur following rupture of a pancreatic pseudocyst or disruption of the main pancreatic duct due to increased pressure within. Initial management is conservative with elemental diet, parenteral nutrition, paracentesis or percutaneous drainage...
and somatostatin analogues [4]. Many respond to conservative therapy with spontaneous closure of the pancreatico-peritoneal fistula. However, several unexpected deaths have been reported in patients during conservative management [5].

Stenting of the pancreatic duct is an option when ERCP confirms a ductal damage. The stent is deployed endoscopically via the transpapillary route and is best positioned by extending beyond the site of the contrast leak. The stent decompresses the pancreatic ductal system and relieves downstream obstruction. There is evidence that lowering the ductal pressure by stenting the pancreatic duct sphincter without reaching the leaking point may be sufficient for healing [6]. Mid-pancreatic duct is the commonest site of disruption as seen in these 2 patients. Successful stent placement achieves excellent long term results in this subgroup of patients with a potentially complex management problem.

References

A middle-aged man with monoclonal gammopathy and osteoporosis
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Introduction
Osteoporosis is a rare disorder with generalised increase in bone density, and impairment of osteoclast mediated bone resorption [1], with a reported prevalence of 1 in 100,000 – 500,000 adults for the mild (benign) osteoporosis and 1 in 200,000 – 500,000 for the infantile (malignant) osteoporosis. Mild osteoporosis may cause no symptoms. Serious forms can result in stunted growth, deformity, fractures, and anaemia, and nerve compression by extra bone causing blindness, facial palsy and deafness. There is no known cure. Monoclonal gammopathy is not a recognised association of osteoporosis, and a Medline search did not show previous reports.

Case report
A 53-year old man presented with sudden onset loss of vision of his right eye. He had noticed gradual impairment of vision in the left eye also over a 6-month period. He also had dyspnoea on moderate exertion for 2 months. There was no history of bone pain, recurrent infections, bleeding manifestations or fractures. He was pale, with bilateral axillary and inguinal lymphadenopathy and moderate hepatosplenomegaly. Both optic fundi had Roth spots and deep retinal haemorrhages with normal vessel calibre and a macular haemorrhage was seen in the right eye.

His haemoglobin was 5.2 g/dl, and the platelet count 35×10^9/L. Blood picture was leucoerythroblastic with marked rouleaux formation. ESR was 155 mm in the first hour. Renal function, liver function and serum calcium were normal. Urine Bence-Jones proteins and serum cryoglobulin were negative. Plasma electrophoresis showed a monoclonal gammopathy of 80 g/l.

A skeletal survey showed generalised bone sclerosis (figure 1). Right axillary lymph node biopsy was compatible with extensive extramedullary haemopoiesis (figure 2). A bone biopsy from the right tibia confirmed histological evidence of osteoporosis.

His anaemia and low platelets were corrected by blood and platelet transfusions, and vision in the right eye gradually improved with the regression of macular haemorrhage. He defaulted and died subsequently after emergency hospital admission from heart failure complicating severe anaemia.

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