Assessment of pancreatic duct damage following trauma: is endoscopic retrograde cholangiopancreatography the gold standard?

N Doctor, JS Dooley, BR Davidson

Summary

A 12-year-old girl was admitted as an emergency with blunt pancreatic trauma. Computed tomography (CT) showed an intact pancreas. She failed conservative treatment. Endoscopic retrograde cholangiopancreatography (ERCP) demonstrated division of the pancreatic duct at the neck of pancreas. At laparotomy, however, there was complete necrosis of the pancreatic head. She recovered well following enteric drainage of the body of pancreas. CT and ERCP are considered the optimal imaging for pancreatic trauma but the findings may be misleading.

Keywords: pancreatic trauma, pancreatic duct, endoscopic retrograde cholangiopancreatography

Introduction

Severe pancreatic trauma is associated with a mortality of 10–20%, although this figure may be reduced by careful assessment of the injury and appropriate surgery.¹ Endoscopic retrograde cholangiopancreatography (ERCP) has been suggested as the definitive method of diagnosing pancreatic duct damage and planning surgery.² We report a case in which ERCP findings did not correlate with those at surgery.

Case report

A 12-year-old girl was transferred to our unit 30 h after being kicked in the epigastrium by a horse. Initial assessment at the referring hospital revealed upper abdominal bruising and tenderness. An abdominal computed tomography (CT) scan showed an oedematous pancreas and she was referred for further management. Following transfer she complained of continuing severe upper abdominal pain. She was haemodynamically stable with localised upper abdominal signs of tenderness and mild guarding. Blood investigations revealed haemoglobin 12.3 g/dl (11.5–15.5), white cell count $20.5 \times 10^9/l$ (4.5–11.0) and platelets $321 \times 10^9/l$ (140–400) (normal ranges). Her amylase was 90 IU/l (70–220); urea and electrolytes, liver function tests and clotting screen were normal.

She was treated conservatively with nil by

mouth, intravenous fluids and an octreotide (Sandoz, UK) infusion (50 µg tid intravenously). A CT scan with intravenous bolus contrast enhancement was repeated after 72 h which again showed an oedematous pancreas with no focal necrosis. Her condition did not improve with further conservative treatment and an ERCP was therefore performed. This revealed an intact duct in the head of the pancreas with a leak of contrast at the neck suggestive of a neck transection (figure 1).

At laparotomy the head of pancreas was completely necrotic and liquified. However, the duodenum and the body and tail of the pancreas were intact and appeared well perfused, non-oedematous and healthy. After removing the necrotic material from the region of the head of pancreas the duct in the body of the pancreas was identified and anastomosed to a Roux loop of jejunum. The patient made a complete clinical recovery and was discharged from hospital three weeks post operatively.

Discussion

Blunt trauma to the pancreas is an uncommon injury and therefore experience of its assessment and management is limited even in the major centres.³ Rapid intravenous bolus enhanced CT scanning has proven effective in the assessment of pancreatic necrosis following

Figure 1 Endoscopic retrograde cholangiopancreatography demonstrating a normal pancreatic duct within the head of pancreas and a leak from the neck of pancreas.

Hepatobiliary and Liver Transplant Unit, Royal Free Hospital and School of Medicine, Pond St, London NW3 2QG, UK
N Doctor
JS Dooley
BR Davidson

Correspondence to
Mr BR Davidson, University
Dept of Surgery, Royal Free
Hospital and Medical School,
Pond St, London NW3 2QG,
UK

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Learning points
<ul style="list-style-type: none">• ERCP is of value in the diagnosis of duct transection• Complete pancreatic necrosis may occur leaving the duct intact

acute pancreatitis.⁴ However Sivit and colleagues suggested that this method was unreliable for assessing blunt pancreatic trauma in children as was our experience in the present case.⁵ This discrepancy may be due to the evolution of the pancreatic necrosis in relation to the timing of the imaging and subsequent surgery.

One of the major clinical questions in panc-

reatic trauma is whether the injury has produced duct transection and some authors have suggested that decisions on the necessity for surgery and its nature can be guided by ERCP findings.² This case has clearly shown that ERCP findings may also be misleading and that an area of complete pancreatic necrosis may occur whilst the duct crossing that segment of pancreas appears intact. Since the pancreatic duct is prone to division in severe pancreatic trauma,⁶ this situation may be due to the pancreatic duct's resilience to digestion by activated pancreatic enzymes.⁷

We would conclude that ERCP is of value in the diagnosis of duct transection but that an intact duct at ERCP does not relate to pancreatic viability or the surgical procedure necessary in treatment.

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Endocarditis due to high-level gentamicin-resistant *Enterococcus faecalis*

PYC Lee, SS Das

Summary
We report a case of aortic valve endocarditis caused by *Enterococcus faecalis* highly resistant to gentamicin, which failed to respond to conventional antibiotic combination therapy. Extensive *in vitro* testing was required to determine an appropriate antimicrobial regimen. Despite bacteriological resolution and cardiac surgery the patient died from complications of infective endocarditis.

Keywords: *Enterococcus faecalis*, endocarditis

Introduction

Enterococci are intrinsically relatively less susceptible to penicillin and other cell-wall-active agents, including vancomycin and teicoplanin.¹ They are also intrinsically relatively resistant to aminoglycosides. However, the combination of penicillin (or vancomycin) with an aminoglycoside achieves a synergistic bactericidal effect, and is the established antibiotic regimen for the successful treatment of enterococcal endocarditis. The first case of endocarditis caused by a high-level

gentamicin-resistant (HLGR) enterococcus was reported in 1984 from the US.² Since then, nine such cases, including two in the UK, have been reported and recently reviewed by Moellering.³ This is only the third reported case of endocarditis caused by a HLGR *Enterococcus faecalis* in the UK.

Case report

A 40-year-old man was admitted with a two-month history of fever, night sweats, rigors and a painful leg. On examination, he had a fever of 38.6°C, signs of aortic regurgitation, finger clubbing, anaemia, two scrotal abscesses and deep vein thromboses in both calves clinically. Echocardiography confirmed aortic regurgitation and detected vegetations on the aortic valve. The clinical diagnosis of infective endocarditis was confirmed with the isolation of *E faecalis* in five sets of blood cultures. Treatment was initiated with intravenous benzylpenicillin (2 mega-units every four hours) and gentamicin (dosage determined by serum levels). Ciprofloxacin (400 mg twice daily) intravenously was substituted for gentamicin when HLGR was detected. Pus from the scrotal abscesses grew the same organism. Despite initial resolution of fever with negative

Public Health Laboratory, St George's Hospital, London SW17 0QT, UK
PYC Lee
SS Das

Correspondence to
Dr PYC Lee, Public Health Laboratory, St George's Hospital, Blackshaw Road, London SW17 0QT, UK

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