Perforation occurring at barium enema demonstrates that below the levators perirectal and ischiorectal fat are contaminated, whereas above the levators but below the peritoneal reflection, gas and barium track into the retroperitoneal space[4,7].

Extensive damage presenting more than 6 hours after injury requires faecal diversion with presacral drainage via the perineum and distal bowel washout. Metronidazole, gentamicin and tetanus toxoid should be given.

Less extensive injuries presenting early and in those with a prepared bowel can be treated expectantly, some authors advocating per-anal rectal repair[8].

Diagnosis of a pelvic abscess is made clinically - local pain and systemic toxicity being marked and is confirmed by ultrasound and CT scanning. Pelvic abscesses are intimately related to bladder and bowel and we avoided formal drainage preferring to rely on high dose antibiotics with spontaneous discharge into the rectum. Against this, intraperitoneal rupture sometimes occurs and is dangerous.

Spontaneous resolution of a pelvic abscess occurring at barium enema and treated only with metronidazole has previously been reported[9].

Ultrasound and CT scanning now appear to provide the answer with guided catheter drainage. Percutaneously via the anterior abdominal wall this is difficult and dangerous; posteriorly, performed via the lower portion of the greater sciatic foramen, thereby avoiding the neurovascular structures, is complicated by pain and kinking of the catheter[10]. Catheter placement under digital pelvic real time ultrasound control for abscess drainage was first described in 1985. More recently drainage has been undertaken transrectally under ultrasound control using a 20G catheter applied to the index finger and transvaginally via the posterior fornix, both with excellent results[11,12].

*Acknowledgment:* I thank Mr J M Beaugié for allowing me to report this case.

**References**

1 Howell HS, Bartizal JF, Freeark RJ. Blunt trauma involving the colon and rectum. *J Trauma* 1976;16:624-32
2 Grasberger RC, Hirsch EF. Rectal trauma. A retrospective analysis and guidelines for therapy. *Am J Surg* 1983;145:795-9
3 Strate RG, Grieco JG. Blunt injury to the colon and rectum. *J Trauma* 1983;23:384-8
4 Terranova O, Meneghello A, Battocchio F, Martella B, Celi D, Nistri R. Perforations of the extraperitoneal rectum during barium enema. *Int Surg* 1989;74:13-16
5 Thomas PR. Ano-rectal injury causing extraperitoneal and subcutaneous emphysema. *Injury* 1987;18:426-7
6 Christou NV. Infectious problems in pelvic trauma. *Can J Surg* 1988;31:90-2
7 Vinsant GO, Buntain WL. Blunt anorectal injuries in children. *J Tenn Med Assoc* 1985;78:158-60
8 Marti MC, Morel P, Rohner A. Traumatic lesions of the rectum. *Int J Color Dis* 1986;1:152-4
9 Giron JA, Ozaktay S. Bacteroides fragilis pelvic abscesses: resolution with metronidazole therapy alone. *South Med J* 1984;77:232-4
10 Butch RJ, Mueller PR, Ferrucci JT Jnr, *et al.* Drainage of pelvic abscesses through the greater sciatic foramen. *Radiology* 1986;158:487-91
11 Mauro MA, Jaques PF, Mandell VS, Mandel SR. Pelvic abscess drainage by the transrectal catheter approach in men. *AJR* 1985;144:477-9
12 Loy RA, Gallup DG, Hill JA, Holzman GM, Geist D. Pelvic abscess: examination and transvaginal drainage guided by real-time ultrasonography. *South Med J* 1989;82:788-90

*(Accepted 21 June 1990)*

# Oesophago-atrial fistula: a side effect of tetracycline?

**A R C Cummin** DM MRCP[1]
**J R W Hangartner** MBBS MRCPath[2]
*Departments of [1]Medicine and [2]Histopathology, St George's Hospital Medical School, Cranmer Terrace, London SW17 0RE*

*Keywords:* oesophago-atrial fistula; tetracycline

Although the left atrium is normally separated from the oesophagus only by the pericardium, oesophago-atrial fistulae are rare, especially in benign disease. Only six cases have been reported[1]. Here we describe a fatal case in a patient who had been taking tetracycline.

*Case report*

A 73-year-old woman presented following a fall. She felt that her hips had given way. There was no loss of consciousness but she had been unable to get up again for 20 min. She had been feeling tired and unwell for a few days and the day before she had been started on tetracycline hydrochloride by her general practitioner. She denied any indigestion.

Three years earlier her legs had become swollen and she was found to have mainly right-sided heart failure with signs of tricuspid regurgitation. She was started on frusemide, amiloride and isosorbide mononitrate and improved but within 6 months she developed complete heart block. Cardiac catheterization showed proximal occlusion of a dominant right coronary artery. A dual chamber (DDD) pacing system was inserted via the left subclavian vein.

On examination following the fall she was unsteady when standing and unable to walk but no other new abnormalities were found. She was admitted to hospital. The next day she became pyrexial and developed cellulitis on the lower legs. A urine specimen showed frank pus and a coliform was cultured. Penicillin, flucloxacillin and gentamicin were given intravenously and there was a rapid clinical improvement.

Two weeks later her left arm became oedematous and dilated veins appeared over the left shoulder. Venography showed complete obstruction of the left subclavian vein from the axilla to the superior vena cava. Intravenous heparin was commenced but 2 days later she passed some 200 ml of fresh blood per rectum. The anticoagulant was stopped but within 2 hours she vomited fresh blood and 2 min later lost consciousness and became pulseless. Attempts at resuscitation were hampered by blood flowing into the mouth and the patient died.

At necropsy fresh blood was present in the mouth and oesophagus. Just below the level of the tracheal bifurcation was a 50×30 mm ulcer in the anterior oesophageal wall with abundant overlying blood clot. The edges were flat and undermined with a clean ulcer base. There were dense adhesions between the oesophagus, left atrium and lung. A small round hole was present in the ulcer base through which a probe passed easily into the left atrium. The stomach, duodenum and large bowel contained fresh and altered blood. Histology showed a benign active chronic oesophageal ulcer. The adjacent mucosa

0141-0768/90/110745-02/$02.00/0

was extensively autolysed precluding comment on its type.

*Discussion*

Oesophago-atrial fistulae may present, as in this case, with a massive haematemesis or with neurological features resulting from food emboli which are thought to enter the left atrium during regurgitation[2]. In the absence of cardiac failure, pressure within the oesophagus may not be very different from that within left atrium and small haematemeses may precede the fatal one. A haematemesis in combination with evidence of cerebral emboli might suggest the diagnosis but, being so rare, the condition is unlikely ever to be considered. A long history of oesophagitis is usual and some consider repeated insults to be a prerequisite for fistula formation as this will cause the oesophagus to adhere to the pericardium and left atrium[2]. In our patient there was no history of oesophagitis.

Sumithran *et al.*[3] described a patient with mitral valve disease who developed an atrio-oesophageal fistula and suggested that this may have resulted from Slow K being obstructed in its passage down the oesophagus by an enlarged left atrium. The only predisposing factor for oesophageal ulceration in our patient was that she had received tetracycline on the day before admission[4] and we wonder whether a similar mechanism might have occurred.

**References**

1 Zeiderman MR, O'Grady JT. Fatal haematemesis arising from benign oesophagoatrial fistula. *Thorax* 1988;43:733-4
2 Mott LJM, Austin GE. Cerebral embolization resulting from esophago-atrial fistula. *Arch Intern Med* 1976;136:718-20
3 Sumithran E, Lim KH, Chiam HL. Atrio-oesophageal fistula complicating mitral valve disease. *BMJ* 1979;ii:1552-3
4 Crowson TD, Head LH, Ferrante WA. Esophageal ulcers associated with tetracycline therapy. *JAMA* 1976;235: 2747-8

*(Accepted 24 January 1990)*

## Primary carcinoma of the cystic duct causing obstructive jaundice

**A Baraka** FRCS[1] **N Y Al Mokhtar** MD[2]
**J P Madda** MBChB MRCPath[3] **M Amirrad** MBBS[1]
**S Asfar** MD FRCS[1] *Departments of [1]Surgery, [2]Diagnostic Radiology and [3]Pathology, Amiri Teaching Hospital, Kuwait*

*Keywords:* obstructive jaundice; carcinoma; extrahepatic bile ducts; Mirizzi syndrome

Primary carcinoma of the cystic duct is rare. To diagnose a case, the strict criteria put forward by Farrar should be fulfilled: (a) the growth must be restricted to the cystic duct; (b) there must be no neoplastic process in the gall bladder, hepatic or common bile ducts (CBD); (c) a histological examination of the growth must confirm the presence of carcinoma cells[1].

Using these criteria 23 cases have been reported in the literature[2-4]. We report another case with some unique features.

*Case report*

A 55-year-old woman presented with 3 week history of right hypochondrial pain, jaundice, itching and anorexia. Examination revealed a tender gallbladder mass, her haemoglobin and WBC were normal. Serum bilirubin was 138 μmol/l (normal range 3-20) and alkaline phosphatase 1035 U/l (normal range 30-85).

Abdominal ultrasound showed distended gallbladder with no calculi, a 2.5×1.5 cm mass in the cystic duct involving and obstructing the common hepatic duct (CHD), dilated intrahepatics and normal liver (Figure 1). Percutaneous transhepatic cholangiography (PTC) showed dilated intrahepatic biliary tree down to the confluence of both hepatic ducts and a curvilinear compression along the lateral aspect of the CHD, a picture similar to Mirizzi syndrome (Figure 2).

At laparotomy, the gallbladder was distended and a 2×2.5 cm tumour was found at the region of the confluence of cystic and CHD with gross dilatation of both hepatic ducts.

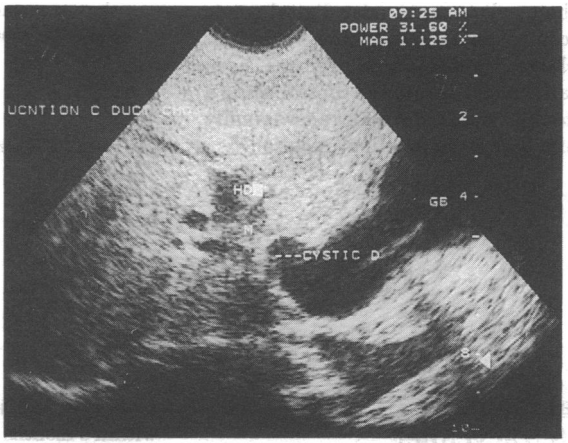


*Figure 1 showing cystic duct tumour (M), distended gallbladder, dilated hepatic ducts and intrahepatic biliary radicles*

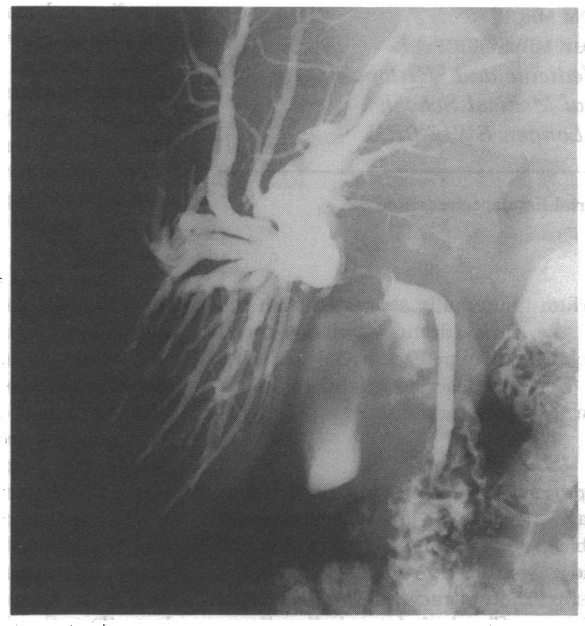

*Figure 2. Percutaneous transhepatic cholangiogram showing dilated intrahepatic biliary tree down to the confluence of both hepatics and curvi-linear compression of CHD simulating Mirizzi syndrome*

0141-0768/90/
110746-02/$02.00/0