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Late oesophageal perforation after intraoperative transoesophageal echocardiography

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Serious haemodynamic instability occurred during emergency surgery for a perforated duodenal ulcer in a 72-year-old man with acute myocardial infarction. Intraoperative transoesophageal echocardiography was crucial for diagnosis of the location of myocardial infarction in the right ventricle and the subsequent haemodynamic management. Postoperatively, a thrombus in the right coronary artery was removed by coronary angiography. The patient's trachea was extubated on the fourth postoperative day. Another 4 days later a leak in the lower oesophagus was suspected because of pleural empyema, and verified. The patient's trachea had to be re-intubated and an oesophageal stent was inserted. The patient was discharged, fully recovered, 2 months after the operation.

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Transoesophageal echocardiography (TOE) is frequently used in cardiac and transplant surgery. TOE may also have a significant impact on intraoperative patient management in non-cardiac surgery.[1] We describe a late oesophageal perforation occurring after intraoperative TOE in a 72-year-old man with acute myocardial infarction undergoing emergency surgery for a perforated duodenal ulcer.

Case report

A 72-year-old male patient (170 cm; 125 kg; body mass index 43.3) attended our emergency room with upper abdominal pain, severe dyspnoea and circulatory shock (arterial pressure 80/60 mm Hg; heart rate 120 beats min^{-1}). He had a history of long-term use of non-steroidal anti-inflammatory medication. Suspected perforated duodenal ulcer was corroborated by intra-abdominal air on the computer tomogram and diffuse peritonitis. The ECG revealed acute inferior myocardial infarction, and this was confirmed by laboratory findings: troponin I, 45 μg $litre^{-1}$ (normal value: <0.35 μg $litre^{-1}$); creatine kinase CK-MB enzyme activity, 396 U $litre^{-1}$ (normal value: <25 U $litre^{-1}$); CK-MB mass, 313 μg $litre^{-1}$ (normal value: <5.0 μg $litre^{-1}$). The patient had no chest pain.

Before inducing anaesthesia we inserted radial arterial, central venous and pulmonary artery catheters under local anaesthesia. Standard monitoring included ECG (five leads), pulse oximetry and capnometry. Anaesthesia was induced and maintained with midazolam, flunitrazepam, fentanyl and rocuronium. Immediately after induction, the patient's mean arterial pressure dropped to 35 mm Hg. Urgent vasoactive support with bolus injections and continuous infusions of epinephrine (0.16 μg kg^{-1} min^{-1}) and norepinephrine (0.12 μg kg^{-1} min^{-1}) was necessary to maintain arterial pressure. His heart rate increased to 140 beats min^{-1}, and cardiac output (8–10 litre min^{-1}), central venous pressure (16–20 mm Hg) and pulmonary artery pressure (45–65 mm Hg) were all above the upper normal limit. We decided to insert a TOE probe (multiplane 5 MHz probe, HP Sonos 1500; Hewlett-Packard, Andover, MA, USA) which was achieved without incident. The patient had a highly hypokinetic and dilated right ventricle, but good contractility of the left ventricle and a small endsystolic left ventricular volume. With on-line ultrasonographic control of right and left ventricular function, we undertook careful volume replacement using Ringer's lactate and a gelatine solution, and gradually reduced the rate of the catecholamine infusions. The patient's heart rate decreased to 90–100 beats min^{-1} while his mean arterial pressure was maintained at 70–80 mm Hg.

At surgery, the abdominal cavity was found to be full of gastric juice and the peritoneal surface severely inflamed, with several areas of fatty tissue necrosis, which were confined to the vicinity of a perforated pyloric ulcer. The ulcer was excised, and a Heineke–Mikulicz pyloroplasty performed. The TOE probe remained *in situ* for about 2.5 h.

On postoperative day 1 the patient's haemodynamic status deteriorated. Because the right-heart failure was refractory to inotropic support, coronary angiography was performed. This revealed almost complete occlusion of the right coronary artery by a thrombus, which was removed. A temporary intra-aortic balloon pump was placed. In the following days the patient was successfully weaned from catecholamines and mechanical ventilation, and his trachea was extubated on postoperative day 4.

On postoperative day 5 a left-sided pleural effusion was identified on a chest radiograph, and then drained. On day 6, pulmonary function deteriorated rapidly and the patient's trachea had to be re-intubated. From day 5 onwards, neutrophil granulocytes increased significantly. An increase in C-reactive protein on the first two postoperative days was followed by a subsequent decrease, with a renewed increase on postoperative day 8. Pleural empyema was drained through a small thoracotomy. An oesophageal perforation was suspected when what appeared to be gastroenteral nutrition fluid was seen to issue from the thoracic drains. The leak in the lower oesophagus was verified by an upper gastrointestinal radiological contrast series (Fig. 1), which excluded other oesophageal lesions such as oesophageal stricture. We decided against a surgical repair of the leak, and inserted an oesophageal stent instead. The leakage then subsided, and the further postoperative course was uneventful. The patient was discharged from the ICU 8 weeks after surgery and was transferred to a rehabilitation centre 2 weeks later. He was discharged fully recovered 2 months later.

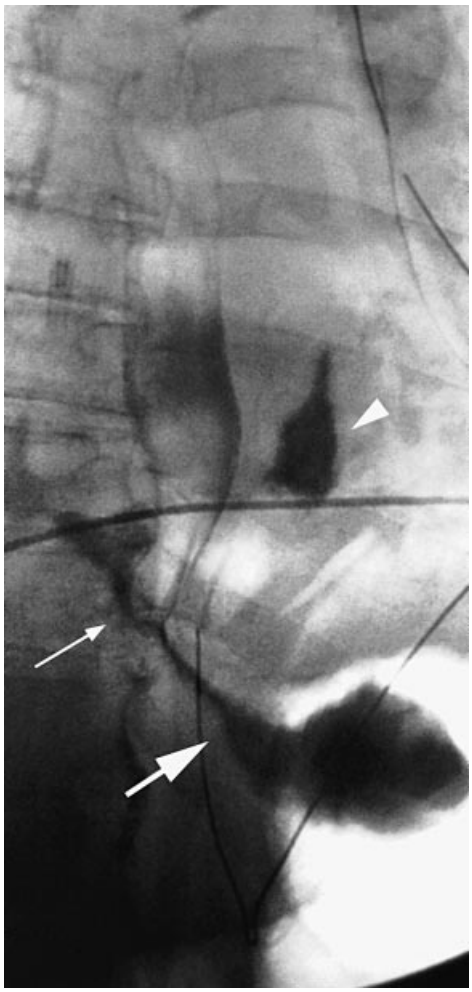

Fig 1 Perforation of the distal oesophagus identified by the extravasation of water-soluble contrast agent (thin arrow). Arrowhead: extraluminal contrast agent in the dorsal mediastinum; large arrow: cardia.

Discussion

While serious oesophageal lesions associated with the use of TOE are rare, reports have been increasing.[2–7] To date, eight cases of serious oesophageal perforation occurring during TOE have been reported. Five of these eight perforations occurred during insertion of the TOE probe, and the lesion was located in the hypopharynx or the proximal oesophagus.[2 5–7] Only a single case of lower oesophageal perforation had been described, which was not caused during insertion of the probe, but occurred intraoperatively while the probe remained *in situ*.[8]

The aetiology in our patient may be different. TOE probes can exert pressures of up to 60 mm Hg, with the potential to cause mucosal ischaemia.[9] In addition, perfusion of the oesophageal mucosa in our patient may have been impaired by prolonged cardiovascular instability with low arterial pressure, which may have significantly contributed to the oesophageal lesion.

In contrast to Kharasch and colleagues[8] we observed a significantly longer interval between the lesion induced by the TOE probe and the first symptoms of perforation (6 days *vs* a few hours). The reason for the delay in clinical symptoms in our pateint is not clear, but may be related to delayed enteral nutrition. Because of the late presentation of the oesophageal perforation, we refrained from surgical repair.[10 11] The mortality rate from iatrogenic oesophageal perforation is high, and early diagnosis and treatment significantly improve the prognosis. Three of the five patients with oesophageal TOE perforation died.[2 5–8] The overall mortality rate for oesophageal perforation varies from 10% to 25%.[3]

It is highly unlikely that the oesophageal lesion had already been present, and was not caused by the TOE probe. The incidence of oesophageal ulcers or perforations in the absence of Boerhaave's syndrome or a carcinoma is extremely low, and the concurrence of such an oesophageal lesion with a duodenal ulcer is even lower.[12]

The use of TOE in patients with known gastro-oesophageal diseases such as strictures, burns, cancer or diverticuli, or in patients undergoing irradiation therapy or gastroduodenal ulcer therapy is not recommended.[8 13] Oesophageal varices are only a relative contraindication, because TOE is often used during liver transplantation in patients with end-stage liver disease and oesophageal varices. In our case we used TOE because the information provided by the Swan–Ganz catheter with continuous measurement of cardiac output and mixed oxygen saturation was inconclusive. TOE revealed the location of the myocardial infarction in the right ventricle, the low left ventricular preload and good left ventricular contractility.

In conclusion, TOE may provide crucial information in patients with relative contraindications and thus may be used in life-threatening situations. However, impaired mucosal perfusion because of cardiovascular instability appears to be an important risk factor for late oesophageal TOE perforation.[8]

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