Mediastinitis after oesophagoscopy: A case report

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ABSTRACT
A 50-year-old male presented with signs and symptoms of oesophageal perforation after a biopsy. Suggestive symptoms and signs were pain in the neck radiating to the back, a rise in temperature and pulse, emphysema in the neck and widening of the mediastinum or a pneumothorax revealed by a chest X-ray. He survived with medical management.

Introduction

Oesophageal perforations carry a serious prognosis because the early diagnosis is often missed. The condition is rare and may be confused with myocardial infarction, aortic dissection, pneumothorax, and perforation of a peptic ulcer.[1] The most common cause is manipulation of the oesophagus by instruments. It occurs more commonly with rigid endoscopes than flexible fibrescopes. Perforation is more common in the upper oesophagus just above the upper sphincter. The next most common site is the lower oesophagus as it narrows to pass through the hiatus. Perforation of the middle third of the oesophagus is uncommon unless benign or malignant strictures have necessitated dilatation.[2] The consequences of perforation are attributable to contamination of the mediastinal tissues and pleural cavities through digestive juices and bacteria that give rise to cellulitis and suppuration.[3] We here report a case in which oesophagoscopy led to a small perforation in the thoracic part.

Case report

A 50-year-old male with a diagnosis of carcinoma oesophagus in the middle third was posted for an oesophagoscopy. He had experienced dysphagia for solids for the previous two months. A pre-anaesthetic examination revealed an eight-year history of bronchial asthma, for which he was taking theophylline and salbutamol. Pulmonary function tests revealed a peak expiratory flow rate (PEFR) of 124 lt/mt, FEV_1 0.7 lt, FVC - 1 lt and a ratio of FEV_1/FVC of 70%. The patient was optimised with steam inhalation, nebulisation, intravenous bronchodilators and antibiotics. A chest examination revealed bilateral decreased air entry and occasional crepts. The chest X-ray was normal. Preoperative dyselectrolaemia (K+ > 6 meq) was corrected. The patient was accepted with high-risk consent and premedicated with alprazolam 0.25 mg in the morning.

Induction was done with intravenous propofol 120 mg and vecuronium bromide 4 mg. Intubation was achieved with a 6.5 mm cuffed tracheal tube. The intraoperative course was uneventful. After two to three minutes of extubation, the patient started desaturating, although his breaths coincided with the movements of bag, therefore ruling out laryngospasm. An injection of hydrocortisone 100 mg was given. His SpO_2 did not pick up beyond 90%, even after 100% of oxygen therapy. Auscultation revealed bilateral decreased air entry. He was moved to a respiratory intensive care unit (RICU) for oxygen therapy at FiO_2 of 0.6. A blood gas analyser at this time showed pH-7.42, pO_2-83 mmHg, pCO_2-56.8 mmHg, and HCO_3-36.2 mmol/litre. Four hours later his blood pressure dipped to 80 mmHg systolic, for which dopamine was started at a rate of 5 µg/kg/mt. He also had tachycardia with a pulse rate of 120/min.

On the second postoperative day his blood pressure was still maintained on dopamine. A chest X-ray revealed broad mediastinal widening. Due to low blood pressure and associated mediastinal widening, a contrast-enhanced CT scan of the chest was advised. The patient was moved to oxygen therapy with a continuation of the dopamine drip. The CT scan revealed a small tear in the oesophagus proximal to the growth. A diagnosis of oesophageal perforation was made. The patient was kept nil per mouth and given broad-spectrum antibiotics in the form of amoxycillin and clavulinic acid. Gastrostomy feeding was given. The patient remained on dopamine for another three days, which was tapered off gradually. The patient did not require ventilatory support at any time, and central venous pressure (CVP) cannula was also not required. This patient survived, as he had a very small thoracic perforation, and was shifted from the RICU on the seventh postoperative day. A series of barium studies was done to trace his healing. Complete healing of the perforation occurred within three months and the patient was administered oral feeds after three months. Until that time, gastrostomy feeding was kept in situ. The patient was discharged from the hospital after one month.

Discussion

Any manipulation of the oesophagus can lead to perforation as it is a thin, walled structure. An oesophagoscopy with a biopsy or dilatation carries an increased risk. Distal perforation can result when the lumen of the oesophagus is difficult to identify, especially if it is deviated from its usual course.[2] Symptoms of perforation depend on the site of perforation and the extent of the inflammatory reaction. Pain, fever and difficulty in swallowing and flexing the neck are early symptoms of a rupture of the cervical part. Cardiorespiratory embarrassment (pain, tachycardia, hypotension), pneumothorax and surgical emphysema may be features in a thoracic rupture.[4] X-rays of the chest and neck are useful in making a diagnosis, which may show air in mediastinal tissue spaces and widening of the mediastinum.

Oesophageal perforation is usually apparent within several hours of the procedure. Patients in whom it is suspected should be

observed for at least 8–12 hours postoperatively, during which time they are to be kept nil by mouth and treated with intravenous antibiotics. A nasogastric tube should not be used initially as it may cause further injury at the site of perforation. A CVP should be placed for parenteral feeding. The longer the diagnosis and treatment are delayed, the greater the mortality. The mortality rate has been shown to be 40% to 60% after 24 hours.[5] The perforation can be treated surgically or medically. The factors affecting the management of any oesophageal perforation depend on the time of diagnosis,[6] the anatomic location, the cause and size of the rupture, the clinical state of the patient, the underlying oesophageal pathology and the age of the patient.[7] Cervical perforations have the best prognosis, with a survival rate of 90%.[8] In addition, spontaneous ruptures have a poorer outcome than iatrogenic perforations by instrumentation.[3] Although in early cases requiring operation, primary repair of the perforation seems to be the method of choice,[6] the management of delayed perforation with mediastinal sepsis remains controversial. Oesophagectomy is superior to primary repair in the management of delayed oesophageal perforation.[9] Barium studies are helpful in detecting the site of perforation and the underlying pathology. Healing of the perforation is followed by a series of oesophagograms. In our patient the features suggestive of thoracic perforation were hypotension, tachycardia and mediastinal widening, while chest pain and emphysema were absent. Our patient survived with conservative treatment, as the diagnosis was made on the second postoperative day.

Perforation of the cervical part is managed by simple conservative measures and surgical drainage of the retro-oesophageal space if suppuration has developed. Complete healing of the oesophagus may take as long as two months and mobilisation of the patient may be speeded up by gastrostomy feeding. Oral feeding can be resumed when there is no radiological evidence of a leak. Perforation of the thoracic oesophagus is more serious and active treatment is required. Repair of the perforation with drainage of the pleural cavities is needed if diagnosis is made early (within six hours), as there will be minimal soiling of the mediastinum and pleural cavity.

It is suggested that the possibility of perforation should be kept in mind if a patient has cardiorespiratory embarrassment after a simple procedure of the oesophagus, such as a biopsy and dilatation. Early diagnosis is essential, as the mortality rate is 50% or more if treatment is delayed beyond 24 hours of injury. **SAJAA**

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