A 63 yr old man was admitted to the emergency-room suffering from severe respiratory distress. An interview with the patient was impossible. His landlady reported a rapid deterioration of the patient’s physical condition during the last 24 h. For at least one year he had complained about lodging of food, frequently followed by regurgitation. He had lost a considerable amount of weight and had an unsteady gait.

On admission he was found to be dyspnoeic, very lean and dehydrated, with pulse rate 120·min⁻¹, and blood pressure 80/40 mmHg. Communication was not possible. Examination of the chest revealed no abnormalities. Screening laboratory tests showed a white blood cell (WBC) count of 0.8·10⁹·l⁻¹, with 64% neutrophils, 8% band forms, 12% lymphocytes and 8% myelocytes. The haemoglobin concentration was 12.6 g·l⁻¹ with a mean corpuscular volume (MCV) of 93 fl. There were signs of a prerenal azotaemia with normal electrolytes and serum protein. Arterial blood gas analysis revealed metabolic acidosis and severe hypoxaemia with pH 7.32; arterial carbon dioxide tension (Paco₂) 3.8 kPa; arterial oxygen tension (Pao₂) 5.0 kPa; bicarbonate (HCO₃⁻) 15 mmol·l⁻¹. An anteroposterior (AP) chest roentgenogram of the patient was taken in the supine position. Infiltrates in both lower lobes and a symmetric widening of the superior mediastinum were found (fig. 1).

Turn to next page for diagnosis.
Diagnosis

Despite intensive treatment with antibiotics, oxygen and volume suppletion, the patient died within 24 h. At necropsy an extensive bronchopneumonia of both lower lobes was found. Cultures revealed Streptococcus pneumoniae and Haemophilus influenzae. There were no signs of aspiration. At the proximal oesophagus a giant hypopharyngeal (Zenker’s) diverticulum of maximal circumference 18×9 cm was found, containing the remains of food.

Discussion

The main causes of mediastinal widening of oesophageal origin are neoplasms, oesophageal hiatus hernia, megaesophagus (e.g. due to achalasia) and, less frequently, diverticula. Localized lesions in the oesophagus may mimic other mediastinal lesions and occasionally even pulmonary lesions [1, 2]. They are predominantly situated in the middle mediastinal compartment.

Roentgenological widening of the mediastinum is seldom of oesophageal origin. If the diverticulum is large, it may be visible on a PA or lateral roentgenogram of the thorax; frequently showing an air-fluid level [2, 3]. Fluoroscopy and cineradiographic examination may be helpful in the diagnostic work-up. The contours of a diverticulum may change with coughing, and a barium swallow shows an abnormal function of the oesophagus in the area of the mass [4]. A contrast oesophagogram is the prime method of establishing the diverticulum. Surgical treatment includes diverticulectomy or cricopharyngeal myotomy [5-7]. The latter can also be conducted endoscopically, using electrocoagulation or carbon dioxide laser [8].

The frequency of the Zenker’s or hypopharyngeal diverticulum has been estimated to be about 0.1% in all upper gastrointestinal radiographic series [9]. It is generally diagnosed in patients over 50 yrs of age, and occurs twice as often in males as in females. The cause of development of a Zenker’s diverticulum is probably dysfunction of the cricopharyngeal muscle, involving a relative slowing of relaxation of the muscle during the course of swallowing [10].

Clinical symptoms of a diverticulum are: dysphagia, regurgitation of food eaten several hours earlier, aspiration, gurgling sounds in the cervical region, foul breath and weight loss. Aspiration, mainly during sleep, may cause pneumonia, lung abscess and chronic pulmonary disease [11, 12]. Complications such as oesophagotracheal fistula, peptic ulceration or development of squamous cell carcinoma are rare [13].

In the patient described, bronchopneumonia was the primary cause of death. Signs of aspiration were not found. Malnutrition due to the Zenker’s diverticulum was probably a contributing factor in the rapid deterioration of the acute illness.

References