X-ray showed bilateral pleural effusion. The white blood cell count was 6.6 x 10/1 with 89% neutrophils. Thoracentesis yielded 0.5 litres of a yellow fluid, from which S. enteritidis was grown. Blood cultures were sterile. Repeated X-ray films showed no underlying pulmonary involvement. Bilateral thoracostomy tubes were inserted and treatment was ampicillin 2 g/4 hours i.v. for 4 weeks and oral amoxicillin for 2 weeks more, but pleural fluid drainage persisted. A bilateral chemical pleurodesis was performed but a sterile pleural effusion recurred 4 months after finishing antibiotic therapy.

Non-typhoidal salmonellae are known to colonize previously damaged tissues and non-pleural pleural and peritoneal effusions have become infected.1,2,6-8 Extension from a nearby site, bacteraemic spread from a gastrointestinal source or a dormant focus in the reticuloendothelial system are all pathogenic possibilities. In contrast to extraintestinal infections, salmonella pleuropulmonary disease usually has an acute onset with symptoms lasting less than a week before a diagnosis is established,2 and half the patients have a positive blood or stool culture.2

Most other Gram-negative pleuropulmonary infections are nosocomially acquired; salmonella is frequently community acquired. Prior abnormalities of the lungs and pleura are found in about 40% of patients, malignancy being the most common predisposing condition and 36% are immunosuppressed, including SLE and corticosteroid-treated individuals.1,2

Treatment of salmonella empyema resembles that of other bacterial empyema. Relapse is common and mortality may be as high as 15%.2 salmonella empyema should be excluded in febrile lupus patients with pleural effusions. Optimal treatment must include drainage and prolonged parenteral effective therapy with a beta-lactam or fluoroquinolone antibiotic. Mortality in salmonella pleuropulmonary infections may reach 100% when antimicrobial agents not active against salmonella are used.1

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Spontaneous oesophageal haematoma presenting as acute myocardial infarction: implications for thrombolytic therapy

Sir, Retrosternal chest pain of oesophageal origin is well known to mimic ischaemic chest pain.1 The benefits of thrombolytic treatment in acute myocardial infarction are well established and are reported to outweigh the risks.2 Speedy thrombolysis has been recommended3 and electrocardiographic changes are not considered to be mandatory.4 We describe a patient who was a potential candidate for thrombolytic therapy but was subsequently proven to have a spontaneous intramural oesophageal haematoma.

A 66 year old woman with a sudden onset of severe retrosternal chest pain of 1 hour’s duration was referred with a suspected myocardial infarction. The pain woke her up from sleep, radiated to her throat and culminated in a sensation of choking. She denied recent vomiting, retching, dysphagia or dyspepsia. Previously she had been fit and well, and was a teetotaller. Clinical examination was normal. The blood count showed a haemoglobin of 13.4 g/l and platelet count and white cell count were within normal limits. The electrocardiogram and chest X-ray were normal. Serial cardiac enzymes and coagulation studies were later found to be normal.

Thrombolytic treatment was withheld. The following day she complained of dysphagia and pain on swallowing. Esophagography showed pronounced bulging of the posterior oesophageal mucosa extending from the upper oesophagus (approximately 25 cm from the tip of the endoscope) 5 cm distally. No oesophageal tear, hiatus hernia, gastric or oesophageal lesions were seen. The appearance was that of an intramural oesophageal haematoma and repeat esophagography at 10 days showed clear signs of it reducing in size. Follow-up esophagography at 4 weeks revealed almost complete resolution of the haematoma and the oesophageal mucosa looked normal. She was treated conservatively and her recovery was rapid and complete.

Painful dysphagia, haematemesis and retrosternal chest pain are well documented as common presenting symptoms of spontaneous oesophageal haematoma.3 However, the only presenting symptom may be severe retrosternal chest pain. This patient would have fulfilled the criteria for thrombolytic treatment in most institutions where electrocardiographic evidence of myocardial infarction is not essential. The consequences of such
treatment may have been profound. We advise continued caution in the selection of patients for thrombolytic therapy in the absence of electrocardiographic changes.

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