

our patient this treatment was only partly successful. Endoscopic clearance of the diverticulum may solve the problem²; but, failing non-operative treatment, diverticulectomy should be considered. This can be a difficult operation with a high morbidity, especially for diverticula in the juxtapapillary region. Smith³ reported a case very similar to ours. A giant duodenal diverticulum obstructed the third part of the duodenum and had to be resected.

Salmonella osteoarticular infection without predisposing factors

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Acute salmonella osteomyelitis is well known to occur in neonates or adults who have sickle cell disease, who are immunocompromised, or who are chronic salmonella carriers¹. It is rare in the healthy adult.

CASE HISTORY

A man aged 28 reported a constant, dull pain in the right shoulder for the past week. There had been no trauma and there was no history of recent foreign travel or preceding diarrhoeal illness. He described some mild 'flu-like symptoms a fortnight previously, but otherwise had been well.

His temperature was 38 °C and his shoulder was mildly tender and swollen, with reduced range of active movement. He had a mild leucocytosis, with raised erythrocyte sedimentation rate (36mm/h) and C-reactive protein (126 mg/L). Plain radiographs showed a cystic lesion in the greater tuberosity (Figure 1). Aspiration of the shoulder joint yielded thick yellow fluid which showed no organisms on microscopy of a Gram stained specimen. Provisional diagnosis was acute osteomyelitis and secondary septic

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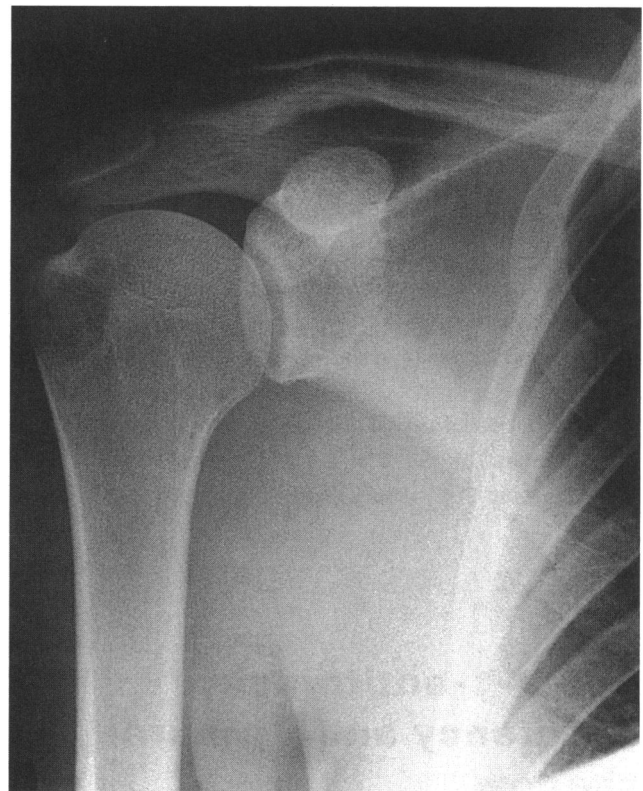


Figure 1 Anteroposterior view of right shoulder showing lytic lesion in greater tuberosity of humerus

arthritis and he was initially treated with intravenous benzylpenicillin and flucloxacillin.

After three days' culture the aspirate from the shoulder grew *Salmonella enteritidis* (sensitive to amoxicillin, cefuroxime and ciprofloxacin). The shoulder was surgically explored. At operation the cystic area surrounded by softened cancellous bone was curetted and lavaged, and the shoulder joint was also irrigated. No free pus was found in either the lesion or the joint. Ciprofloxacin 500 mg twice daily was started postoperatively. Cultures of swabs and scrapings from the cyst and the shoulder joint did not grow *S. enteritidis*.

Further screening of the patient was undertaken in light of the unusual microbiological findings. Bone and

liver biochemistry was normal as were autoimmune rheumatoid profiles and haemoglobin electrophoresis. He was human immunodeficiency virus negative. Stool cultures did not yield salmonella. Throughout his admission he remained systemically well; he became afebrile and his inflammatory markers returned to normal within a week. He was discharged on a six-week course of ciprofloxacin.

COMMENT

In children salmonella osteomyelitis usually develops from haematogenous spread¹. Since the advent of antibiotics the condition has most commonly been seen in patients with sickle-cell anaemia or other haemoglobinopathies, systemic lupus erythematosus, neoplasms, or immunosuppression. About 60% have diarrhoea or positive stool cultures on presentation².

Salmonella osteomyelitis frequently involves the diaphyses of long bones as well as vertebrae, and most cases are caused by non-*typhi* serotypes³. Salmonellosis can be divided into five syndromes—enterocolitis, enteric fever, bacteraemia, local infection and the chronic carrier state. These may overlap or coexist. Salmonella osteoarticular infection in a healthy adult is very unusual. In the few reported cases it has usually been chronic in nature⁴ or been preceded by a

diarrhoeal illness. To our knowledge *S. enteritidis* has never previously been reported as causing osteoarticular infection in a healthy patient without predisposing factors. In patients with secondary salmonella infections who have negative stool cultures, the organism is believed to have been dormant in the reticuloendothelial system or bowel and to have been activated by depression of the host's defences². Our patient showed no signs of immunodepression. Salmonella osteoarticular infection tends to become chronic and can be difficult to eradicate⁴. Extensive and often multiple debridement may be required in addition to lengthy antibiotic treatment.

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Alpha-1-antitrypsin deficiency and a pleural shadow

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Haemothoraces are normally associated with acute thoracic trauma, coagulopathy or rupture of a great vessel¹. In addition, pulmonary embolism and pneumothorax may be complicated by bloody effusions^{2,3}.

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CASE HISTORY

A woman aged 72 was referred with a two-week history of productive cough and fever. She was known to have the homozygous PiZZ phenotype for α -1-antitrypsin deficiency. There was no history of smoking, asbestosis or industrial dust exposure. On examination, she was pyrexial, tachypnoeic and tachycardic. Chest examination was consistent with left-sided consolidation and pleural thickening.

Her haemoglobin (Hb) on admission was 12.4 g/dL with total white cell count $23.2 \times 10^9/L$. By day two her Hb had fallen to 9 g/dL with a white cell count of $37.3 \times 10^9/L$. Clotting studies were unremarkable, with prothrombin time 12 s and activated partial thromboplastin time 34 s. Her urea and electrolytes were normal and remained so.

Her admission chest radiograph (Figure 1) revealed marked pleural thickening around the left lung with evidence of emphysema and consolidation. A spiral computed tomogram (CT) scan of her chest with intravenous contrast confirmed the lobulated pleural appearances and consolidation within the left lung. The CT numbers of this pleural mass were equivalent to the