Bacterial oesophagitis in an immunocompromised patient

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Summary: Bacterial oesophagitis is an uncommon and poorly described entity affecting particularly the immunosuppressed patient. The diagnosis rests on the demonstration of bacterial invasion of the oesophageal wall in the absence of other pathological processes. The causative organisms usually are Gram-positive cocci and there may be associated bacteraemia. The case report describes a leukaemic patient with bacteraemic bacterial oesophagitis.

Introduction

Oesophageal infections in the immunocompromised host are well recognized and usually are secondary to fungal or viral pathogens. Bacterial oesophagitis is a rarely reported entity presenting with dysphagia and odynophagia and hence, is clinically indistinguishable from other opportunistic oesophageal infections. Bacterial oesophagitis may be the source of bacterial sepsis in immunocompromised patients and requires antibiotic treatment.1 The authors describe a patient with relapsing leukaemia whose course was complicated by bacterial oesophagitis and bacteraemia.

Case history

A 57 year old woman developed acute lymphocytic leukaemia which responded to chemotherapy and her disease went into remission. An episode of submandibular cellulitis 2 years later was treated successfully with broad-spectrum antibiotics. Five months later she was retreated with chemotherapy for relapse of leukaemia. She became pancytopenic (haemoglobin 8.6 g/dl, white blood cell count 0.1 x 10^9/l, platelet count 4 x 10^9/l), febrile and complained of odynophagia and heartburn. Blood cultures were positive for coagulase-negative Staphylococcus and Streptococcus viridans, despite appropriate antibiotic coverage.

Upper endoscopy revealed extensive ulcerations at the gastroesophageal junction covered by exudate. Brushings were negative for yeast, cellular inclusions or giant cells. Owing to the profound thrombocytopenia, no biopsies were obtained. Omeprazole 40 mg daily was administered orally. Eight days later the patient developed a sudden and fatal intracerebral haemorrhage.

Complete postmortem examination showed no evidence of a septic focus except for the distal oesophagus where marked submucosal oedema and haemorrhagic necrosis with minimal inflammation were found (Figure 1). There was heavy bacterial infiltration with Gram-positive cocci consistent morphologically with Staphylococcus.

Discussion

Oesophageal infections occur frequently in immunocompromised patients. Both fungal and viral agents are the usual pathogens. Bacterial oesophagitis is a poorly recognized and characterized entity that may develop in the immunosuppressed host. Interpretation of culture results is difficult as oropharyngeal bacterial flora may contaminate the oesophagus. Hence, areas of mucosal disruption secondary to acid reflux, viral or fungal pathogens could become colonized by bacteria acting as secondary invaders.

Walsh et al. established strict histopathological criteria requiring the demonstration of bacterial invasion of oesophageal mucosa or deeper layers with no concomitant fungal, viral or neoplastic involvement and no prior oesophageal surgery.1 A subset of patients has associated bacteraemia in which the organism causing bacterial oesophagitis is morphologically consistent with that cultured from blood in the absence of an extraoesophageal source of bacteraemia. This case report fulfills these criteria as complete postmortem examinations did not reveal any other source of infection and blood cultures grew Staphylococcus.
Although oesophageal infections by Mycobacterium tuberculosis, Actinomyces israelii and Treponema pallidum have been well described there is a paucity of reports of other bacterial pathogens affecting this organ.

With rare exceptions the clinical setting is an immunocompromised and/or neutropaenic patient affected by malignancy or after organ transplantation. Recently, bacterial oesophagitis has been reported in patients infected by the human immunodeficiency virus.

Reviewing their 2 year experience with infectious oesophagitis after bone-marrow transplantation, McDonald et al. found no cases of bacterial oesophagitis after 46 endoscopies, of which almost half demonstrated fungal and/or viral pathogens. However, out of 59 autopsied patients, three had bacterial oesophagitis caused by a single organism and four cases had mixed infections, including two patients with both fungal and bacterial, and two patients with viral and bacterial infections.

A second clinical series of immunocompromised patients demonstrated bacterial oesophagitis in 16% of 123 autopsied cases but only three patients were diagnosed antemortem. The causative organisms were mainly orally derived Gram-positive cocci though mixed bacterial infection and Gram-negative pathogens also were seen. Only four patients had bacteraemic bacterial oesophagitis and Gram-positive bacteria were isolated in all of them.

In contrast to the above two series, a recent prospective endoscopic study reported in abstract form suggests that bacteria more frequently cause infectious oesophagitis in the immunosuppressed host. Of all documented oesophageal infections in 77 patients, 19 (25%) were bacterial with Gram-positive cocci being the most frequently isolated organism.

In contrast to other causes of infectious oesophagitis the treatment of bacterial oesophageal infection includes antibiotics and hence, accurate diagnosis is essential. Although this entity was recognized in only a few patients antemortem the response to antibacterial therapy appears favourable provided the underlying clinical situation is not hopeless as it was in this case report.

The presence of oesophageal symptoms in an immunocompromised patient is a clear indication for oesophagoscopy, brushings and biopsies to determine the underlying cause. Bacterial oesophagitis should be strongly considered in such patients particularly in the presence of unexplained sepsis.

It is obvious that more studies are needed to establish the frequency of true bacterial oesophagitis in these patients.

References