Candida albicans peritonitis in a patient with Felty’s syndrome

D W Thomas, P Munuswamy, K Adu-Poku, C S Holgate, P Hickling, A Lambert, A G Prentice

A 53 year old man with Felty’s syndrome presented with abdominal pain and fever. He underwent a laparotomy after starting broad spectrum antibiotics. An intestinal biopsy showed skip ulcers with fungal hyphae. Peritoneal exudates grew Candida albicans. He was started on intravenous fluconazole and then switched to liposomal amphotericin to which he showed a good clinical response. After one month at home he was readmitted with candidosis and died of a myocardial infarction.

A 53 year old man presented with abdominal pain lasting one week. The pain started in the epigastrium, localising to the right iliac fossa. He had fever and rigors and complained of vomiting and diarrhoea for four days. The oropharynx appeared to be normal.

Rheumatoid arthritis was diagnosed 10 years previously, with uncomplicated Felty’s syndrome for three years. His median neutrophil count during this time ranged from 0.7 to 1.6 × 10^9/litre. He also had asthma and ischaemic heart disease and was taking aspirin, diclofenac, amoxicillin, digoxin, and salbutamol and beclomethasone inhalers.

On presentation his neutrophil count was zero. He was started on ceftazidime and gentamicin. His fever continued to spike and he developed diffuse abdominal tenderness. Computed tomography of the abdomen showed hepatosplenomegaly. The pancreas, kidneys, adrenals, and bowel were all normal.

Because he remained unwell, antibiotics were changed to meropenem and vancomycin and a laparotomy was performed. At surgery, skip lesions were seen in the small bowel. A section of small bowel was removed. Purulent peritoneal fluid was cultured.

Blood cultures were negative, but peritoneal fluid grew Candida albicans, sensitive to fluconazole and amphotericin. Oropharyngeal candidiasis was then noted.

Histopathological examination of the ulcerated intestinal mucosa showed fungal hyphae and spores (fig 1) with Gram positive cocci.

Fluconazole was started for the candida infection, and because the fever did not settle, liposomal amphotericin was added. After a week the fever started to abate. His neutrophil count ranged from 0 to 15.6 × 10^9/litre from 0 to 15.6 × 10^9/litre without granulocyte colony stimulat-

In one of the larger reported series of this complication there were 12 major and 32 minor infections over a three year period in 15 patients. Only two of the minor episodes were superficial C albicans infection. Systemic fungal infections were not seen. Invasive epiglottitis with C glabrata has been described in a patient with Felty’s syndrome.

Phagocytosis by neutrophils is an important part of the host defences against infection with Candida spp, which exist in three forms, namely: yeast/spores, elongated forms without hyphae (pseudohyphae), and true hyphae with septa. The transition from yeast to hyphae is important for fungal virulence. Hyphae appear able to exit from the cells that engulf them and avoid phagocytic death. The mucosae of the mouth, gut, and vagina may be colonised in up to 80% of normal individuals. When host defences are compromised, invasive disease may occur. Corticosteroids, antibiotics, and alterations in gastric pH predispose to invasive disease.

In this case, the clinical presentation and the appearances of the bowel biopsy suggest that the gut was overgrown with C albicans, which invaded the mucosa and penetrated the gut wall directly into the peritoneal cavity. This invasion may have been made possible by mucosal bacterial infection or ulceration from long-term non-steroidal anti-inflammatory drug treatment. Kopelson et al described two patients with intra-abdominal malignancy who developed isolated C albicans peritonitis. They presumed that the tumour facilitated the passage of fungi into the peritoneum. Most cases of candidal peritonitis are documented in patients on chronic ambulatory peritoneal dialysis where there is direct inoculation through the skin.

Cases of spontaneous peritonitis with C albicans are rare. This case report appears to be the first spontaneous C albicans peritonitis in Felty’s syndrome, and illustrates that the diagnosis may not be clear at the initial presentation. The case also demonstrates the difficulty in clearing these infections. Although the immediate cause for his death was ischaemic heart disease, the disseminated candidosis and methicillin resistant Staphylococcus aureus infection made an important contribution to the patient’s morbidity. In view of
the initial poor response to intravenous fluconazole, it is not clear whether continued use of oral fluconazole would have altered the outcome.

**Take home messages**

- This is the first report of a patient with Felty’s syndrome and spontaneous *Candida albicans* peritonitis
- The diagnosis was not clear at the initial presentation and the infection persisted despite treatment with fluconazole
- Although the immediate cause for this patient’s death was ischaemic heart disease, the disseminated candidosis and methicillin resistant *Staphylococcus aureus* infection made an important contribution to his morbidity

**Authors’ affiliations**

D W Thomas, P Munuswamy, K Adu-Poku, C S Holgate, P Hickling, A Lambert, A G Prentice, Departments of Haematology, Histopathology, Rheumatology, and Surgery, Derriford Hospital, Plymouth, PL6 8DH UK

Correspondence to: Dr D W Thomas, Department of Haematology, West Suffolk Hospital, Bury St Edmunds, Suffolk IP33 2GZ, UK; wayne.thomas@wsh.nhs.uk

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**REFERENCES**