Letters to the Editor

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Suppressive treatment he was on for ulcerative colitis was a significant risk factor for deep-seated fungal infection and, in retrospect, the diagnosis could perhaps have been suspected earlier.

Prompt treatment and early diagnosis is critical, but as this case illustrates, it can be very difficult. Apart from oral candidiasis, there was nothing to raise the suspicion of a fungal peritonitis. Without the third laparotomy, the diagnosis would not have been considered until Candida was demonstrated from peripheral sites seven days later.

Serodiagnosis of candidal infection can be difficult. Candida antigen detection is specific, and can help distinguish colonised from systemically infected patients, but sensitivity can be low.4 Confirmation of systemic candidiasis requires isolation from blood cultures or an otherwise sterile site except urine, or histological evidence of yeast or mycelial forms in tissues from an 'at risk' patient,5 which can take time. Amphotericin B is recommended for the treatment of deep Candida infections, with fluconazole being a second-line treatment.6 Conventional amphotericin B is nephrotoxic and should not be administered in a dosage exceeding 1.5 mg/kg/day. Liposomal amphotericin has a significantly shorter infusion time than conventional amphotericin B and is reported as giving a rise to less systemic toxicity and renal impairment,6 enabling the administration of a much higher dosage than with conventional amphotericin. Amphotericin B causes hypokalaemia, and although liposomal amphotericin B has been reported as causing hypokalaemia in up to 18% of cases, our patient tolerated the liposomal amphotericin well, with rapid clinical improvement.

The possibility of candidal peritonitis, although very rare, should be considered in any patient potentially at risk. Empirical antifungal treatment should be considered pending the results of other investigations such as serology and peripheral cultures. We suggest that clinicians consider liposomal amphotericin as a useful alternative to conventional amphotericin B in these patients.

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We would like to thank Mr AJ Knox and Dr TJ Clarke for their permission to report this case.


Follow-up of mycoplasma pneumonia

Sir,

I was interested to read the learning points tabled in Dr Shah’s useful paper1 on adult respiratory distress syndrome due to mycoplasma pneumonia. They did not mention follow-up of this acute condition.

In 1973 my late wife developed respiratory distress shown to be due to mycoplasma pneumonia. It cleared rapidly on treatment and three months later a chest X-ray was normal. A year later another severe respiratory infection led to a further chest X-ray and this showed widespread deposits from primary adenocarcinoma of the lung. This, of course, is anecdotal evidence, but I think you will understand why I personally feel that such a respiratory infection occurring in a previously healthy subject should be followed up very carefully for at least a year or two after the event.

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