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Community-acquired *Acinetobacter* pneumonia

Sir,

Achar and colleagues report a case of community-acquired *Acinetobacter calcoaceticus* var. *anitratus* (now known as *Acinetobacter baumannii*) pneumonia from the Middle East.¹ They summarize cases from the literature but do not include our series of 11 blood culture-positive cases from Darwin, Northern Territory, Australia, with an accompanying review of 34 previously reported cases.²

In our ongoing study of adult bacteraemic community-acquired pneumonia at Royal Darwin Hospital since 1986, *A. baumannii* accounts for 16 of 148 cases (11%). *A. baumannii* is the third commonest cause of death (nine deaths, case fatality rate 56%), behind *Burkholderia* (formerly *Pseudomonas*) *pseudomallei* (16 deaths) and *Streptococcus pneumoniae* (12 deaths). Our cases of *A. baumannii* are characterized by male predominance (14/16), presence of underlying risk factors such as alcoholism or chronic lung disease (16/16), wet season occurrence (14/16), fulminant presentation with unilateral chest X-ray consolidation (15/16) and death within 24 hours in fatal cases (6/9). All our community-acquired isolates have been gentamicin sensitive and cefotaxime/ceftriaxone resistant. Pleural effusions are unusual.

Because *B. pseudomallei* and *A. baumannii* account for 48% of adult mortality from community-acquired pneumonia in our region of tropical Australia, our protocol for empirical therapy of severe pneumonia is to add gentamicin to ceftriaxone in patients with underlying risk factors. If *B. pseudomallei* is isolated or strongly suspected (for example, in diabetics), ceftazidime is substituted.³ If *A. baumannii* is isolated, piperacillin is added to the gentamicin and ceftriaxone ceased, or imipenem monotherapy is substituted.² Despite a high level of suspicion, intensive-care facilities and appropriate antibiotics, mortality from these two organisms remains high. This is consistent with studies from Thailand demonstrating that ceftazidime therapy, while halving mortality from septicemic melioidosis, does not substantially decrease mortality in the first 48 hours.⁴

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Watermelon stomach in the CREST syndrome

Sir,

El Omar and colleagues¹ describe the interesting association between severe bleeding from gastric telangiectasias and the CREST syndrome, and the authors conclude that this association is surprisingly rare with only four previous reports. They also comment that gastric vascular abnormalities can be very extensive giving rise to the watermelon stomach. I have recently dealt with such a problem in a patient with the CREST syndrome and this case illustrates several important points in the management of such patients.

A 46 year old female with CREST syndrome was referred for assessment of chronic iron deficiency. Two years previously gastritis and oesophagitis had been treated with omeprazole, but because of continued bleeding, with negative investigations including angiography, a laparotomy and on-table enteroscopy had been performed. Small bowel telangiectasias were found and the most severely affected segment of ileum was excised. At the time it was known that some small bowel vascular lesions had been left *in situ*. This procedure was initially successful but after 18 months iron deficiency anaemia resistant to oral iron was again apparent; at this stage she was receiving approximately 3 units of blood every 3 weeks.

At upper gastrointestinal endoscopy red streaks radiating out from the pylorus were seen, characteristic of watermelon stomach. Histological examination of endoscopic biopsies confirmed the diagnosis. Selective visceral angiography showed subtle abnormalities with hypervascularity of the antrum but the small bowel vasculature appeared normal. A technetium-99 labelled red cell scan suggested that the blood loss was from the gastroduodenal region rather than the distal small bowel. Her bleeding has been controlled by endoscopic treatment with heater probing and injection of alcohol subsequently because of a technical problem with the heater probe. Following ablation of the endoscopically visible lesions she has remained stable for 13 months, she has required no blood transfusions and there is no further evidence of bleeding.

This report illustrates one further case of symptomatically bleeding gastric telangiectasias with the CREST syndrome in addition to those described by El Omar *et al.* and is unusual amongst these in demonstrating the extensive lesion called watermelon stomach.

It shows that such patients may have multifocal gastrointestinal vascular lesions and recurrence of anaemia following surgery may occur. Careful assessment using endoscopy, biopsy, angiography and radio-