CASE REPORT

A case of necrotising fasciitis caused by Serratia marcescens: extreme age as functional immunosuppression?

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Abstract

We report the case of a 97-year-old woman who had a prolonged hospital admission for the treatment of right-sided heart failure. During her stay she experienced a rapid deterioration, characterised by shortness of breath, cardiovascular compromise and a hot, red, swollen calf. Post-mortem examination demonstrated that this was caused by necrotising fasciitis due to Serratia marcescens as a single pathogen. This is only the second reported case of this condition in the absence of diabetes or immunosuppression, and clinical deterioration was much more rapid. The case underlines the importance of circumspection and regular review in the diagnosis of the elderly patient. It reminds us that these patients should be viewed as functionally immunosuppressed, and that some or all of the haematological markers of infection can be absent even in severe disease.

Keywords: gerontology, immunocompromised patient, fascitis, necrotising, Serratia, older people

Case report

A 97-year-old woman was admitted to our elderly care ward following a fall. Her background included vascular dementia, atrial fibrillation, chronic kidney disease stage 3 (estimated glomerular filtration rate of 35 ml/min) and right-sided heart failure, with preserved left ventricular function. On examination, significant peripheral oedema, bibasal pleural effusions and an elevated jugular venous pressure were noted. Her records revealed that she had gained 24 kg since her admission to residential care 5 months previously. Diuretic therapy was introduced but, despite escalating treatment to combination furosemide and metalozone, only 2 kg of weight loss was achieved over 3 weeks.

At 02:00 on the 21st day of her inpatient stay, the patient was reviewed by the medical team as she was complaining of leg pain. No abnormality was detected on examination, but early abnormalities are likely to have been obscured by marked peripheral oedema. At 10:00 the patient was newly tachypnoeic (respiratory rate 25), hypotensive (BP 81/56) and had oxygen saturations of 81% on air (98% with 3 l/min oxygen delivered through nasal cannulae). Her right leg was noted to be mildly erythematous, and more swollen than her left. A clinical diagnosis was made of deep vein thrombosis with pulmonary embolus and therapeutic-dose low molecular weight heparin was administered. Her D-Dimer was measured to be 1,192 ng/ml, and CT pulmonary angiography was requested. Her C-reactive protein (CRP) was noted to have increased to 157 mg/l, from 31 mg/L the previous day, but her neutrophil count was only 2.7 × 109/l. At the time, this was thought to be in keeping with the clinical diagnosis as CRP is commonly elevated in pulmonary embolism, and some have suggested its use as a biomarker; however, this rise is rarely to levels >85 mg/l [1]. Similarly, elderly individuals are known to have an impaired neutrophil response [2], and infection should still be suspected in this
context. At 16:00 her right calf appeared engorged and dusky, but the extent of discolouration had not changed and the foot was spared. At 20:30 her calf was erythematous, warm, sloughy and blackened. The patient was now unresponsive. Her CRP at this time was 207 mg/l, neutrophils $3.6 \times 10^9$/l, prothrombin time 22 s, venous pH 7.14 and lactate 9.8 mmol/l. The diagnosis was revisited, and intravenous flucloxacillin and amoxicillin were administered. Unfortunately the patient suffered a cardiac arrest and passed away at 22:24.

Post-mortem examination was performed to determine the cause of death. No significant thrombi were identified in the pulmonary arteries or leg veins. Histological examination of the right leg showed acute inflammation with extensive necrosis involving skin and subcutis, including superficial fascia, consistent with necrotising fasciitis (see Figure 1).

Microbiological swabs taken from the affected area resulted in the growth of \textit{Serratia marcescens} as a single pathogen. Owing to the infrequency of this organism as causative in this context, paraffin embedded samples were cut from the fascial layer for 16S ribosomal DNA analysis. This analysis confirmed the presence of a single ribosomal DNA sequence, which demonstrated $>99\%$ homology with \textit{S. marcescens}.

As can be seen in the figure, numerous, monomorphic, Gram-negative bacilli were present throughout the dermis and involving subcutis, but solely scanty, Gram-positive bacilli were seen on the skin surface (not shown). This pattern would be inconsistent with post-mortem contamination.

\section*{Discussion}

\textit{S. marcescens} is a Gram-negative bacterium from the enterobacteriaceae family. It is ubiquitous in the environment, especially in damp conditions, but can be an opportunistic pathogen, most frequently in the respiratory and urinary tracts of hospitalised patients [3]. Soft tissue infection by \textit{S. marcescens} is rare, and typically occurs in immunocompromised individuals with diabetes, malignancy, steroid use or renal failure [4]. It can be responsible for necrotising fasciitis as a single pathogen [5], but only one other case has been reported in the absence of diabetes or immunosuppression [6]. In this case, the evolution from first symptoms to overt necrosisation was much slower, occurring over 4 days. When it does occur, \textit{S. marcescens} soft tissue infection carries a high mortality, in part because of co-morbidity but also because the antibiotics frequently given to treat cellulitis are typically ineffective. The antimicrobial therapy administered in this case was in accordance with local policy, but would have provided poor coverage against those Gram-negative or methicillin-resistant pathogens that can be acquired in hospital, and would certainly have been ineffective against \textit{S. marcescens}.

In unwell, elderly patients the risks of antibiotic-associated...
diarrhoea must be balanced against the benefits of broader spectrum antimicrobial therapy on an individual basis.

We propose that, despite the relatively benign pathogen, disease progression was rapid in this case because of the functional immunosuppression of extreme age. It is often clinically overlooked that there is a decline in the speed and efficacy of immune response with increasing age, which is independent of the level of co-morbidity [7]. These effects are even more pronounced if they are accompanied by a poor nutritional status [8].

This case underlines the importance of circumspection and regular review in the diagnosis of the elderly patient. It reminds us that these patients should be viewed as functionally immunosuppressed. The failure of this woman to mount a neutrophilic response to life-threatening bacterial infection further demonstrates that some or all of the haematological markers of infection can be absent even in severe disease.

Key points

- Extreme age is a form of immunosuppression.
- Haematological markers of infection can be absent in severe disease.
- ‘Benign’ pathogens can present aggressively and atypically in the elderly.

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Conflicts of interest

None declared.

References


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