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Case Report

Cerebral Abscesses and Septic Pulmonary Emboli due to *Serratia marcescens* Infection Arising from a Portacath

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Percutaneous intravenous devices are an essential component of modern health care. Although they are generally associated with a low incidence of vascular access device-related sepsis, the events following a vascular catheter-related sepsis can be clinically significant and difficult to treat. Here we report a case of Portacath-related sepsis with *Serratia marcescens* resulting in cerebral and pulmonary emboli, which in our knowledge, has not been reported before. Definitive identification and prolonged antimicrobial treatment according to culture sensitivities can lead to resolution of septic and pulmonary emboli.

KEYWORDS: catheter-related infection, cerebral abscess, septic emboli, *serratia marcescens*

Introduction

Although percutaneous intravenous (IV) devices are an essential component of modern healthcare for administering fluids, medications and for hemodynamic monitoring, they are also a recognized risk for significant clinical morbidity and mortality due to bloodstream infections.[1] Here we report a case of Portacath-associated septic cerebral and pulmonary emboli caused by *Serratia marcescens* in a lady undergoing chemotherapy, which, to the best of our knowledge, has never been reported before.

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Case Report

A 73-year-old woman who was undergoing chemotherapy with FolFox (5-FU, Leucovorin and Oxaliplatin; 2-weekly) through a Portacath for a resected Dukes C colon cancer, presented to our hospital 3 days post-7th cycle of chemotherapy with nausea, vomiting and diarrhoea.

On admission, she was afebrile and haemodynamically stable, but clinically and biochemically dehydrated with a raised creatinine, urea, sodium and potassium (Table). An electrocardiography showed atrial fibrillation with ventricular rate of 120 beats/min. Chest X-ray, abdomen X-ray and mid-stream urine were all normal. Blood cultures were sent, in view of recent chemotherapy and as per hospital policy. Over the next 36 hours, she became increasingly confused. A computed tomography (CT) scan of her brain revealed no hemorrhage/space occupying lesion or extra axial collection, but age-related generalized cerebral atrophy was present. There was no biochemical abnormality noted at this stage apart from borderline neutropenia (Table) and blood/urine/stool cultures were all negative.

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Table. Haematological and biochemical parameters during clinical course of illness

	Day 1 (admission)	Day 3	Day 20	Day 30	Day 60
White cell count ($\times 10^9$/L)	10.5	2.0	23.6	10.7	7.9
Neutrophils ($\times 10^9$/L)	8.8	1.2	20.5	8.1	5.2
C-Reactive Protein (mg/L)	52.0	–	50.7	135.4	27.9
Urea (mmol/L)	30.9	15.5	4.3	7.2	6.7
Creatinine (µmol/L)	204	96	73	81	80
Sodium (mmol/L)	137	142	139	135	142
Potassium (mmol/L)	5.7	4.3	5.0	4.2	4.1

She was commenced on IV antibiotics with ceftriaxone (1 g twice daily) and gentamicin (240 mg once daily) empirically, as per hospital policy for presumed neutropenic sepsis.

On the 20th day of her hospital admission, the patient became increasingly short of breath with pleuritic type chest pain, and hypoxic on room air; chest X-ray was unchanged from previous on admission. In view of the above, CT-pulmonary angiogram was arranged, which demonstrated multiple thick-walled cavitating lesions in both upper lobes of the lungs located peripherally with a few small cavitating lesions showing "tree in bud" appearance and "ground glass" haze in the right upper lobe, suggestive of septic or metastatic emboli (Figure 1). There was also a small right-sided pleural effusion but no pulmonary embolism. At this stage, she was febrile (body temperature = 38°C), with raised inflammatory markers (i.e. raised C-reactive protein and neutrophilic leukocytosis) on blood test (Table). She was continued with IV ceftriaxone and gentamicin at this stage. An aspirate of the right-sided pleural effusion showed no organisms on microscopy and had no growth on aerobic and anaerobic culture. Biochemistry of pleural effusion was normal and no malignant cells were seen. A bronchoscopy was performed at this stage which showed no endobronchial lesion, but multiple washings were taken and sent for microbiology. In view of the probable septic emboli in the lungs, an magnetic resonance imaging (MRI) brain was undertaken (although the patient did not exhibit any focal neurological signs), which revealed multiple "ring-enhancing" lesions in left parietal, frontobasal region and corona radiata, suggesting septic emboli (Figure 2).

At this point the search for a causative organism and a source began to yield results. Blood cultures from the portacath grew *S. marcescens*, which was resistant to a wide range of antibiotics (i.e. ceftriaxone, ampicillin, gentamicin, cotrimoxazole and tobramycin), but sensitive to meropenam, amikacin and ciprofloxacin. Repeated blood cultures taken from peripheral veins had no growth. Bronchial washings taken during bronchoscopy also had heavy growth of *S. marcescens* (which had the same susceptibilities to the original blood culture isolate) and *Stenotrophomonas maltophilia*, and the latter was sensitive to sulphafurazole. Legionella antibody and Allergic bronchopulmonary aspergillosis/Acid-fast bacilli/Pneumocystis jirovecii screen were negative. In view of the microbiology results the patient's antibiotics were changed to amikacin (500 mg twice daily), meropenam (1 g 3 times daily) and sulphamethaxazole (1 g twice daily) and were administered via a central line (left-internal jugular). The portacath was removed on the 25th day of admission and the tip on culture grew *S. marcescens* (similar susceptibilities to previous isolates from blood culture and bronchial washings). A transthoracic and transoesophageal echocardiogram of heart

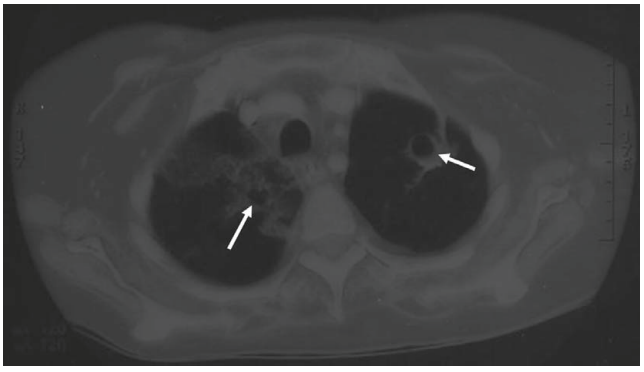

Figure 1. Computed tomography scan of chest showing thick-walled, septic emboli in upper lobes, bi-laterally (arrow), with some associated ground-glass opacity, mostly pronounced in the right-lobe.

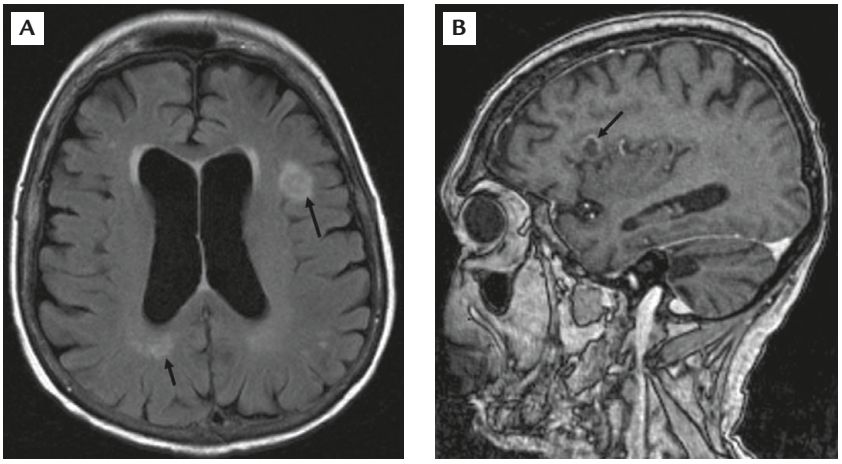

Figure 2. Contrast enhanced T2-weighted magnetic resonance imaging of brain showing septic emboli in subcortical and periventricular areas (arrows); there is minimal perilesional oedema.

showed no vegetations, no valvular/septal abnormality with a normal ejection fraction of > 50%. The patient was afebrile at this stage with evidence of clinical improvement.

With input from clinical microbiologist, the IV antibiotics were continued for a total of 6 weeks, with repeat MRI brain and CT chest every 2 weeks. Repeat imaging of head and lung on week 2 suggested resolving consolidation and cavitating lesions in the lung, but stable "ring-enhancing" lesions in the brain, as seen on the index MRI. The IV antibiotics were stopped at week 6 and central line removed and the patient was commenced on oral ciprofloxacin 750 mg twice daily for a total duration of further 6 months. Progress imaging of lung at week 4 and week 6 showed complete resolution of pneumonic changes including all cavitating lesions, and imaging of brain at week 6 and week 8 showed normal evolutions of septic emboli with gradual but slow regression of abscesses with mild perifocal oedema. The patient was well clinically 10 weeks post-admission, receiving rehabilitation for discharge to home, with completely normal blood counts (Table).

At the time of this report, 15 months since the initial event, the patient remains well, with follow-up MRI brain at 3-, 6- and 9-months showing almost complete resolution of brain lesions. There is also no evidence of metastatic disease at the time of this report based on a CT chest/abdomen/pelvis and bone scan.

Discussion

Serratia marcescens is a Gram-negative enteric bacilli. It rarely colonizes human hosts, being mainly an opportunistic pathogen. It can however grow in many areas in hospitals and healthcare facilities and can occasionally cause sporadic infections.[2] There are an estimated 250,000 cases of nosocomial infections occurring each year in the United States[3] and intravascular catheter-related sepsis due to Serratia species accounts for about 1.4% of these cases.[4,5]

This case highlights a rare but serious complication of *S. marcescens* infection. There are only a few reported cases of cerebral abscesses caused by *S. marcescens*. Two such cases of adults with cerebral infection caused by *S. marcescens* were—a lady who developed *S. marcescens* cerebral abscess secondary to infective endocarditis embolisation[6] and an elderly man with *S. marcescens* meningitis who was found to have cerebral abscesses.[7] The occurrence of cerebral abscesses due to *S. marcescens* is usually restricted to neonates. The usual route of infection that results in cerebral abscess formation tends to be either contiguous (e.g. sinusitis or otitis media), from cranial trauma, or due to hematogenous spread from an infected distant focus.[8]

In one study examining the frequency of nosocomial Serratia infections and the common sites of entry along with the underlying risk factors for infection, it was found that 80% of Serratia infections were nosocomial, with the commonest entry sites being, in descending order, lung, genitourinary tract, unknown, IV line, gastrointestinal tract and skin.[9] More relevant to this case, the same study also found that when they examined the risk factors for such serious infection with *S. marcescens*, the most common underlying disorder was malignancy, followed by renal failure and diabetes mellitus.

In another case report by Johnson et al, as previously mentioned, describing a fatal case of *S. marcescens* meningitis

where the origin of the infection was an indwelling urinary catheter, the patient was an 83-year-old man without malignancy but with a past medical history of left ventricular failure, chronic obstructive airways disease and chronic renal impairment.[7] The authors concluded that the significance placed on the isolation of *S. marcescens* from urine is higher when the patient has multiple comorbidities given the risk for serious complications. The patient in our case report had colorectal cancer which would have been the main risk factor for developing a serious nosocomial infection from *S. marcescens*.

The *S. marcescens* grown from blood cultures in our case was resistant to most β-lactam antibiotics which appear to be consistent with previous case reports.[10] It was however sensitive to meropenem. Carbapenem resistance has, however, been reported with clinical isolates of *S. marcescens*.[11] Aminoglycosides generally have good activity against *S. marcescens*.[12] The combination of meropenem with the aminoglycoside amikacin proved to be successful in this case despite disseminated infection. This particular combination of antibiotics has been recommended in a case report of a premature neonate successfully treated for cerebral abscesses due to *S. marcescens*.[13]

It is important to note that this case describes an unusual complication from a central venous access device. Despite *S. marcescens* being a rare opportunistic infection, it does have the potential to cause severe complications such as those described in this case. The other issue arising from this case is the difficulty of treating these infections, given the unusual antimicrobial resistance of these organisms. Fortunately, despite resistance to most of the β-lactams, the *S. marcescens* isolated in this case remained sensitive to meropenem and amikacin.

In summary, *S. marcescens* is a rare source of serious nosocomial infections and is an unusual cause of cerebral abscesses. Being an opportunistic infective agent, a patient undergoing chemotherapy for colorectal cancer such as in this case would be the type of patient most at risk to develop such a complication. Although the patient remains free of metastatic disease during the time of this report, it remains to be seen if the interruption of systemic chemotherapy as a result of the infection impacted on her predicted overall- and disease-free survival.

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