Case Report

A puzzling cause of persistent Pseudomonas aeruginosa septicaemia in a patient on maintenance haemodialysis

Ying-Kai Chen¹,⁴, Hua-Chang Fang¹,², Kang-Ju Chou¹,², Po-Tsang Lee¹,²,³ and Hsiao-Min Chung¹,²

¹Division of Nephrology, Department of Medicine, Kaohsiung Veterans General Hospital, Kaohsiung, ²National Yang-Ming University, School of Medicine, ³Institute of Clinical Medicine, National Cheng Kung University Medical College, Tainan and ⁴Division of Nephrology, Department of Medicine, Armed Forces Zuo-Ying Hospital, Kaohsiung, Taiwan

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Introduction

Vascular access is the life-line of chronic haemodialysis patients. Its complications play a major role in morbidity and mortality [1]. Vascular access infection remains the leading cause for access failure when cuffed silicone catheters are used for long-term access and is the second most common cause of graft failure when polytetrafluoroethylene (PTFE) grafts are used [2]. Most reports deal with dual-lumen tunneled cuffed catheter related bacteraemia. Arteriovenous (A-V) graft infection with Pseudomonas aeruginosa, as reported here, has been rarely discussed.

Case

A 72-year-old diabetic male had a history of hypertension and cerebral infarction with sequel of right side hemiplegia. An A-V graft was created on the right forearm on 26 June 2003 for diabetic nephropathy. He started maintenance haemodialysis on July 10 using this A-V graft. He was re-admitted on 23 July because of fever and mental disturbances. Physical examination showed grade 2 systolic murmur of the heart over left lower sternal border and gangrene of the 4th right toe. Vital signs were as follows: blood pressure 100/76 mmHg, pulse rate 84 beats per min, respiratory rate 15 beats per min and body temperature 38.2°C. White blood cell count showed 11 300/mm³ with differential count of neutrophils 79%, haemoglobin 8.3 g/dl and platelet count 152 000/mm³. Blood urea nitrogen was 50 mg/dl, plasma creatinine 7.2 mg/dl, glucose 307 mg/dl, sodium 137 mmol/l, potassium 4.1 mmol/l, alanine transaminase 24 U/l and aspartate transaminase 21 U/l. The urine was clear, with protein 100 mg/dl, RBC 2+/high power field and aspartate transaminase 21 U/l. The urine was clear, with protein 100 mg/dl, RBC 2+/high power field and white blood cell count 2–4/high power field. There was no urinary tract infection or graft infection. After admission, P. aeruginosa, which was sensitive to piperacillin and gentamicin, was isolated from two sets of blood culture. Infective endocarditis was suspected, but no vegetation could be found on trans-esophageal echocardiogram. After 4 weeks administration of piperacillin and gentamicin, fever subsided and general condition improved. The patient was discharged on 21 August.

One week later, fever and abdominal pain developed. White blood cell count was 10 970/mm³, with neutrophils 73% and lymphocytes 14%, haemoglobin 7.6 g/dl, platelet count 246 000/mm³ and C-reactive protein was 2.9 mg/dl. Urinalysis revealed white blood cell count 2–4/high power field, RBC count 3–5/high power field. No obvious abdominal lesion was found on CT. Upper GI panendoscopy disclosed gastric ulcer with signs of recent bleeding. Pseudomonas aeruginosa septicaemia was considered not to be entirely healed and piperacillin and gentamicin were administered for another two more weeks. The patient was discharged on 18 September in a relatively stable condition.

The patient was sent to our emergency division on 26 September due to fever and lethargy for 2 days. His blood pressure was 98/57 mmHg, pulse rate 103 beats per min, respiratory rate 24 per min and body temperature 35.4°C. He was drowsy with a coma scale of E3V2M4. Physical examination revealed supple neck, grade 2 systolic murmur over apex, and soft abdomen without focal tenderness. There were no signs of graft infection or embolism. White blood cell count was 23 190/mm³, haemoglobin 12.0 g/dl and platelet count 140 000/mm³. Blood urea nitrogen was 60 mg/dl, plasma creatinine 9.4 mg/dl, glucose 487 mg/dl, sodium 132 mmol/l, potassium 5.4 mmol/l, alanine trans-
aminase 19 U/l, aspartate transaminase 17 U/l and C-reactive protein 49.2 mg/dl. Arterial blood gas analysis (under nasal cannula of O₂ 21/min) showed pH 7.505, PaCO₂ 30 mmHg, PaO₂ 122 mmHg and HCO₃⁻ 23.9 meq/l. No active lung lesion could be found by chest X-ray. *Pseudomonas aeruginosa* was isolated in two of four sets of blood culture but no pathogen was isolated in urine culture. Because no infectious focus could be found, whole body Gallium-67 scintigraphy was performed, with marked uptake on the right forearm, corresponding to A-V graft location. Despite the absence of signs of inflammation, A-V graft infection with sepsis was highly suspected. The A-V graft was removed immediately and piperacillin and gentamicin were prescribed for another 2 weeks. A-V graft tissue put in culture led to growth of *P. aeruginosa*, which was identical to the pathogen isolated from blood. Oral ciprofloxacin was prescribed for two more weeks and a permanent catheter was inserted in the left subclavian vein later. The fever resolved and the patient remained in a relatively stable condition after discharge.

### Discussion

We presented a chronic haemodialysis patient with persistent *P. aeruginosa* septicemia due to A-V graft infection, despite the absence of local signs of infection. The diagnosis was made by gallium scintigraphy and graft tissue culture. The patient rapidly recovered after A-V graft removal and appropriate antibiotic treatment.

The clinical diagnosis of graft infection can be difficult, especially when there are no local signs of graft infection. Nuclear imaging techniques, such as Indium-111 labelled WBC scan and Gallium-67 citrate scintigraphy, have been used to complement the diagnosis. Compared with Indium-111 labelled WBC scan, Gallium-67 citrate scintigraphy is the preferred method because it is less expensive and does not require special preparation. According to previous reports [3–6], Gallium-67 citrate scintigraphy is a good tool to diagnose graft infection, with a sensitivity of 78–100% and a specificity of nearly 100%. The only obstacle to Gallium-67 citrate scintigraphy is the incision uptake of graft, which rapidly decreases after the first week of operation [3,4].

According to Marr *et al.* [7], only 15 (24%) of 62 confirmed episodes of graft-related bacteraemia were caused by Gram-negative bacilli. *Pseudomonas aeruginosa*, which is widespread in nature and has a predilection for moist environments, is a usual pathogen in graft infection. It rarely causes disease in the healthy host. The relative risk for infection is greatly increased, however, when normal cutaneous or mucosal barriers have been breached or bypassed, when the protective function of the normal bacterial flora has been disrupted or when immunologic defense mechanisms have been compromised. In our patient, puncturing vascular access for dialysis, exposure to the hospital environment, diabetes mellitus and end-stage renal disease are all risk factors for infection. No event of burn injury, nosocomial lung infection or urinary tract infection could be traced in the course of hospitalization. Infection due to access puncture during dialysis is the most probable cause of infection.

Local infections of the A-V graft site can often be cured with early aggressive medical therapy, without resorting to graft excision. Once bacteraemia is established, intravenous antibiotics should be given for 3–4 weeks [8]. Considering that patients undergoing long-term haemodialysis have often exhausted all other possible sources of vascular access, it seems preferable to preserve vascular graft as well as possible. However, abscess formation in the immediate proximity to the graft, purulent drainage from infection that dissects onto the graft material, infected aneurysmal dilations of the graft and severe clinical symptoms such as septic shock require surgical excision of the entire graft or a segment of it. This is particularly true when the source of recurrent bacteraemia is a PTFE graft. With recurrent bacteraemia, antibiotic therapy should be continued for another 3–4 weeks.

The initial antibiotic selection for *P. aeruginosa* bacteraemia should take into account antimicrobial susceptibility [9–12], and two antipseudomonal antibiotics should be administered together. Antipseudomonal penicillins plus aminoglycoside are the preferred therapy.

In addition to antibiotic treatment, measures should be taken to prevent infections during access puncture, such as adequate disinfection of the skin, paying more attention to possible contamination of the puncture needle (re-puncturing using the same needle can cause contamination, and should never be undertaken), avoiding scratching of the puncture site due to pruritus and preventing contamination of the puncture site at the time of bathing.

In conclusion, A-V graft infection with *P. aeruginosa* is uncommon in uraemic patients and persistent septicemia may be the unique sign, without evidence of local infection. Gallium-67 citrate scintigraphy is helpful in the evaluation of A-V graft infection. Appropriate antibiotic treatment is necessary and early removal of A-V graft may be unavoidable to cure patients.

**Conflict of interest statement.** None declared.

### References


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