

*The Interesting Case***The patient with a clotted PTFE graft developing fever**

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**Abstract** Haemodialysis access graft infection is easily recognizable when local symptoms (warmth, swelling, pain, or drainage) predominate, and endocarditis is a well established complication of infected grafts. We report a case of bacterial endocarditis complicating silent infection in clotted haemodialysis access graft. It is suggested that, clotted non-functioning grafts may be the harbingers of silent infection, and should be suspected as the source of infection in every haemodialysis patient that presents with fever, even in the absence of clinical signs of graft site infection.

**Key words:** haemodialysis access graft; infection

**Introduction**

Polytetrafluoroethylene (PTFE) grafts are the most commonly used access devices for chronic haemodialysis in the US, and thrombotic complications account for graft failure in most instances [1,2]. While infections are not as common as thrombotic events in causing access graft failure, the inability to recognize an ongoing infection originating from the vascular access, can lead to metastatic seeding of bacteria with potentially ominous complications, with heart, bone and lungs being the most commonly involved sites [3]. Bacterial endocarditis is a well recognized complication of infected Patent haemodialysis access [4]. Recent report by Robinson *et al.* [5], identified 20 cases of bacterial endocarditis in haemodialysis patients, who had an overall mortality of 30%. Of interest, PTFE graft was the access of choice in all patients who developed fatal complications and only in one patient was the PTFE graft removed prior to patients demise. We have recently reported that clotted haemodialysis access graft can harbour silent infection, which can result in metastatic infectious complications [6]. Here we report a case of bacterial endocarditis, resulting

from bacterial seeding from silent infection in clotted haemodialysis access graft.

**Case report**

Forty-four-year-old black male with end-stage renal failure (ESRD) secondary to focal segmental nephrosclerosis, who has been on haemodialysis for 7 years, presented with fever and malaise. At presentation, he appeared acutely ill, his temperature was 39°C, blood pressure 140/90 and respirations 24/min. The patient had a clotted graft in the left arm, which had been non-functional for 2½ years prior. Haemodialysis was performed through a patent Gortex graft in the opposite arm. Of note, physical examination did not suggest any signs of infection (warmth, redness, pain, swelling, or purulent discharge) in any of the graft sites. Lung and heart examinations were normal. The white cell count was 20 000/cu<sup>3</sup>, with a left shift, and the chest radiogram was negative. Blood cultures were positive for *Staphylococcus aureus*, which was sensitive to vancomycin. The patient was placed on vancomycin with resolution of the fever. As part of the work-up, a WBC Indium-111 scan was performed and revealed diffuse increased uptake, which was confined to the clotted graft area. The lungs, heart, bone, as well as the Patent graft area did not show increased uptake. A trans-oesophageal echocardiogram study failed to reveal valvular vegetations. The findings of increased Indium-111 uptake in the clotted graft were dismissed as non-specific, and were attributed to the thrombus in the graft. Intravenous vancomycin therapy was given for 4 weeks. Two weeks after the antibiotic therapy was stopped, the patient presented again with fever and malaise. Physical examination revealed a new murmur consistent with mitral regurgitation. *S. aureus* was again cultured from the blood. In addition to increased uptake in the clotted graft, repeat WBC Indium-111 scan revealed increased uptake in the mitral valve, consistent with endocarditis. A trans-oesophageal echocardiogram showed mitral valve vegetations. Therapy with vancomycin and tobramycin was started, and the clotted graft was surgically removed. Purulent material was found in the clotted graft, and cultures revealed *S. aureus*. After 2 weeks

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Note. This case was one of the 41 patients included in a study which was published by the authors in the *Journal of the American Society of Nephrology* [6].

therapy with vancomycin and tobramycin and four additional weeks of weekly i.v. vancomycin, the patient had complete clinical resolution of infection, and uneventful recovery. Follow-up echocardiogram and WBC Indium-111 scan which were performed 1 month after completion of antibiotic therapy were negative. He had no recurrence of bacteraemia thereafter, and remains alive and doing well on haemodialysis.

## Discussion

The present case illustrates that silent infection originating from clotted graft can lead to recurrent bacteraemia and endocarditis. While it is well established that endocarditis can originate from infections in Patent grafts in haemodialysis patients, and some of which can be clinically silent [4], this is to our knowledge the first report of bacterial endocarditis resulting from silent bacterial infection in clotted access graft. Furthermore, while there is a clear indication for removal of infected Patent grafts in unresolved bacteraemia [7], until our recent report [6], there has been no data in the literature about the incidence of infections in clotted non-functioning haemodialysis grafts and their management. Our results demonstrate that clotted non-functioning grafts are frequent harbingers of infection. They should be suspected as the source of infection in every haemodialysis patient that presents with fever, even in the absence of clinical signs of graft site infection. Furthermore, we find that even in the totally asymptomatic patients these grafts are frequently infected; 13 of 21 asymptomatic controls, who were enrolled in the study for the simple reason of having clotted grafts, had increased Indium-111 uptake in the clotted graft area, which upon surgical removal contained purulent material. Of interest, a large proportion of the patients who had clotted grafts and presented with signs of systemic infection, as well as the portion of asymptomatic controls who were found

to have infection in the clotted graft were diabetic (60% in all). These findings are in agreement with literature reports of higher incidence of graft complications (infection and clotting) in diabetic patients [7,8]. In summary, the practicing nephrologist should be made aware, that clotted PTFE access graft in haemodialysis patients can be the source of infection, which can lead to significant morbidity and mortality. Patients with clotted grafts that present with fever of unknown origin should be studied with an Indium scan. If positive, surgical removal of the graft is indicated. Furthermore, haemodialysis patients with clotted grafts who are undergoing evaluation for transplantation, should have an Indium scan done. If positive, removal of the graft should be performed prior to transplantation and institution of immunosuppression.

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