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Case Report

Central venous catheter-related right atrial thrombus in two kidney transplantation recipients

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Abstract

Right atrial thrombus formation is a known mechanical complication of central venous catheter insertion, with an incidence of between 1.9% and 42%. There is an increased risk of thromboembolism following renal transplantation. However, a right atrial thrombosis is rarely reported in renal transplant recipients. Here, we report two cases of renal transplant recipients in whom a right atrial thrombus developed after kidney transplantation. One of them required surgery, whereas the other patient recovered after being given an anticoagulant therapy with warfarin.

Keywords: anticoagulant therapy; central venous catheter; kidney transplantation; right atrial thrombus; surgery treatment

Introduction

Central venous catheters are used commonly for intravenous medications. Right atrial thrombus formation is a known mechanical complication of central venous catheter insertion, with an incidence of between 1.9% and 42% [1]. There is an increased risk of thromboembolism following renal transplantation. Renovascular thrombosis of the graft, deep venous thrombosis and pulmonary thromboembolism are the most common sites of thrombosis after kidney transplantation [2]. Although recent reports have shown that a right atrial thrombus formation can be visualized 6–8 weeks after catheter insertion, thrombus sites in the right atrium change according to the location of the catheter tip. The optimal location of the catheter is the junction of the superior vena cava and the right atrium [3]. However, a right atrial thrombosis is rarely reported in renal transplant recipients. The only case report concerned a child who received a renal allograft from a deceased donor [4]. Here, we report two cases of renal transplant recipients in whom a right atrial thrombus developed post-operatively. One of them required surgery, whereas the other patient recovered after being given an anticoagulant therapy.

Case 1

A 29-year-old female patient, who had been receiving a continuous ambulatory peritoneal dialysis therapy for ~6 months because of irreversible post-partum renal failure, received a human leucocyte antigen (HLA)-two-mismatched renal allograft from her mother. Before transplantation, her physical examination was normal. An electrocardiogram showed a normal sinus rhythm. Echocardiography, which was routinely performed before transplantation, showed a normal left and right atrial and ventricular configuration. On the day before the transplantation, a temporary jugular three-lumen central venous catheter was inserted for intravenous medications; this was removed 4 days after transplantation.

The transplantation surgery was successful, and we started an immunosuppressive regimen including tacrolimus, mycophenolic acid and prednisolone. No complication was observed during the post-operative period. The serum creatinine level decreased to 0.8 mg/dL at the 10th day of the transplantation. Fifteen days after the transplantation, she suffered from palpitation, and an electrocardiogram revealed sinus tachycardia. Transthoracic echocardiography showed the presence of a mobile, oval mass measuring ~2.5 × 2.0 cm in the right atrium, and the mass was prolapsing *via* the tricuspid valve into the right ventricle at diastole (Figure 1A). Therefore, transesophageal echocardiography was performed, and again, it was seen that a mass image measuring ~1.5 × 2.2 cm was located adjacent to the inferior vena cava end of the right atrium (Figure 1B). The mass was not impairing the tricuspid valve function. Doppler ultrasound evaluation of the lower extremity venous system was normal. She had not been receiving any oral contraceptive drugs. Tests including antinuclear antibody, anti-double-stranded DNA, lupus anticoagulant, antiphospholipid antibodies, antithrombin III activity, protein C and S activity, homocysteine level, activated protein C resistance and prothrombin mutation were performed to evaluate whether there was any hypercoagulable state. The plasma lipid levels and serum fibrinogen level were normal. An activated

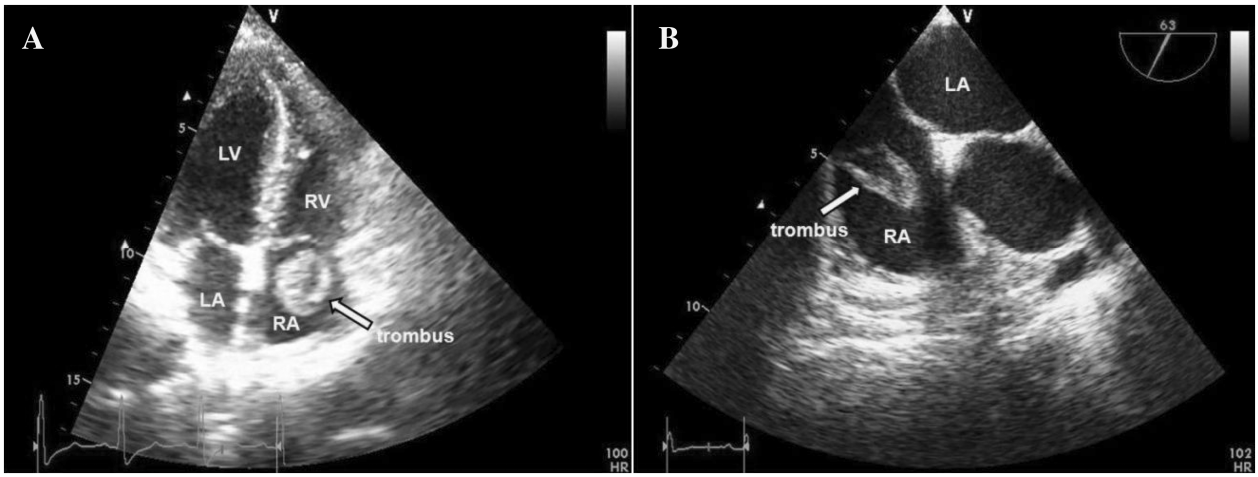

Fig. 1. A. The arrow shows an oval mass measuring ~2.5 × 2.0 cm in the right atrium in transthoracic echocardiography. **B.** The arrow shows a mass measuring ~2.5 × 2.0 cm in the right atrium in transesophageal echocardiography.

protein C resistance due to the factor V Leiden mutation was found. Cardiac surgery was performed, and the mass measuring ~3.0 × 2.0 cm, which was attached to the entrance of the inferior vena cava, was removed (Figure 2). The histological examination of the material revealed that the mass was a thrombus. She was started on an anticoagulant treatment with warfarin. Her graft function and physical examination were completely normal, and she was discharged from the hospital.

Case 2

A 29-year-old female patient, who had been on haemodialysis therapy for ~3.5 years because of end-stage renal disease of unknown origin, received one haplotype-matched renal allograft from her father. She received haemodialysis *via* a permanent tunnelled dialysis catheter because an attempt to fashion arteriovenous fistula failed, and she refused peritoneal dialysis. Echocardiography was performed as a routine procedure before transplantation, and it was found normal except for a grade 1 aortic regurgitation and mitral valve regurgitation. She started the same immunosuppressive regimen as mentioned above. The catheter was withdrawn 17 days after renal transplantation. Transthoracic echocardiography was repeated for a research study, and it revealed a thrombus formation measuring ~3.5 × 0.8 cm within the right atrium (Figure 3A). Also, transesophageal echocardiography showed a mass with irregular margins measuring ~3.5 × 0.9 cm extending from the superior vena cava to the right atrium (Figure 3B). The above-mentioned haematological tests were performed, and no abnormality was found. An evaluation of the lower extremity venous system by Doppler ultrasonography was normal. Similarly, she did not receive oral contraceptive drugs. Because she was clinically asymptomatic, surgery was not performed, and anticoagulant therapy with warfarin was started. In the control echocardiography, it was seen that the size of the thrombus decreased, and she was discharged from the hospital. Twenty days after the anticoagulant therapy, a control transesophageal echocardiography showed a residual thrombus measuring 0.5 × 0.6 cm. The therapy was continued, and 45 days after the therapy, transesophageal echocardiography was repeated, and no thrombus formation was observed.

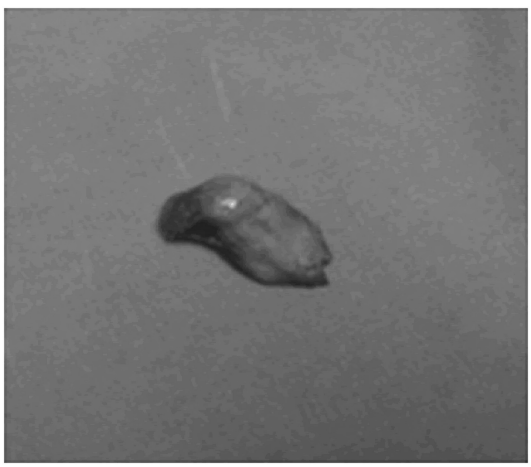

Fig. 2. It is seen that the mass measuring ~3.0 × 2.0 cm was removed by cardiac surgery.

Discussion

Right atrial thrombus is a well-known complication of central venous catheters in haemodialysis patients. Thrombosis sites after kidney transplantation include the renovascular thrombosis involving the graft, deep venous thrombosis (mainly on the side of the transplant) and pulmonary thromboembolism [2]. A right atrial thrombosis following a renal transplant is extremely rare. There has been only one case report about a right atrial thrombus in a renal transplant recipient in the literature. This case with right atrial thrombosis was a paediatric renal transplant recipient [4]. Here, we present two cases of renal transplant recipients who experienced a right atrial thrombus formation. In one of them, a temporary jugular three-lumen catheter was inserted for vascular access in the post-

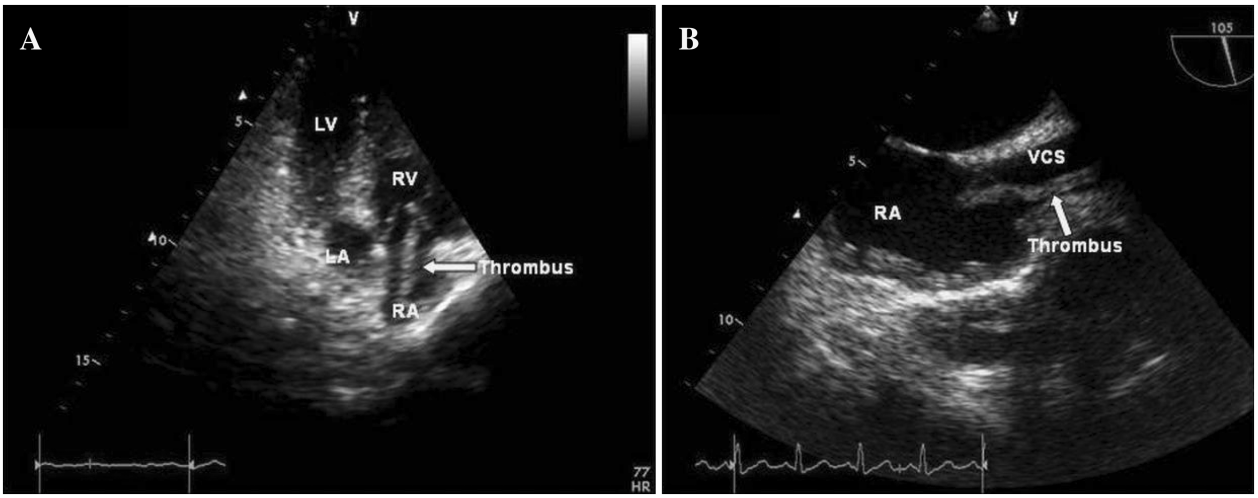

Fig. 3. A. The arrow indicates a thrombus formation measuring ~3.5 × 0.8 cm within the right atrium in transthoracic echocardiography. **B.** The arrow shows a mass with irregular margins measuring ~3.5 × 0.9 cm extending from the superior vena cava to the right atrium in transesophageal echocardiography.

operative period for intravenous medications and was withdrawn 4 days later. In the other patient, a jugular permanent dialysis catheter was placed 3 years earlier for haemodialysis and was removed 17 days after kidney transplantation.

There are two categories of complications associated with central venous catheters: infectious and non-infectious (i.e. mechanical). The thrombus formation is the most common non-infectious complication. Thrombotic complications are usually diagnosed when solutions cannot be infused or blood cannot be withdrawn [3]. Catheter-related thrombus formations are usually asymptomatic, and their true incidence is not known because most of them are explored during autopsy.

It is known that, if there is a separated or stagnant blood flow, blood clot formation is facilitated [5].The other possible mechanism of the thrombus formation is the endothelial damage caused by trauma to the free wall of the atrium by the catheter [1]. If the patient has an underlying hypercoagulable disorder, it can also be a contributing factor for the atrial thrombus formation. We investigated our patients in terms of inherited and acquired thrombotic disorders. In the first patient, we detected an activated protein C resistance due to the factor V Leiden mutation. We believe that the catheter insertion facilitated the atrial thrombus formation because of the thrombotic diathesis of the patient. However, in the second patient, we did not find such a hypercoagulable state.

Cyclosporine A has procoagulant properties, releases von Willebrand factor and P-selectin from the endothelium, and increases F8 levels [2]. Although cyclosporine A is well known for a drug-induced thrombus formation, none of our patients received this immunosuppressive agent.

Permanent catheter tip location is associated with the development of thrombus. According to Hickman *et al.*, the optimal location for the catheter tip is at the junction of the superior vena cava and right atrium, but not within the atrium itself [6]. Gilon *et al.* studied 55 patients by transesophageal echocardiography within 1 and 8 weeks after Hickman catheter implantation. In the study, the tip of the catheter was placed within the right atrium in 13 patients, on the superior vena cava–atrium junction in 8 patients and within the superior vena cava in 27 patients. The thrombus developed in 12.5% of patients within 1 week, and all of the patients with the thrombus were in the group in which the catheter was placed in the right atrium. None of the patients with the other catheter tip locations developed demonstrable thrombus [5]. Similarly, in both of the present cases, the catheter tips were in the right atrium.

The optimal management of patients with catheter-related atrial thrombus is still unclear. Removal of the catheter [5], fibrinolytic therapy [7], very-low-dose warfarin (1 mg daily) [5] and low-molecular-weight heparin [8] are reported treatment options for the catheter-induced thrombosis. Surgery is another choice of treatment and should be considered in the beginning if the thrombus is infected [9]. The size of the thrombus is an important factor to decide the suitable therapy [1]. If it is <2 cm, simply removing the catheter is probably adequate, although some nephrologists suggest a systemic anticoagulation for a few weeks. If the thrombus is >2 cm, an immediate surgical thrombectomy should be considered along with the removal of the catheter [10]. In both of the patients, the catheter was removed before the thrombus was detected. In our first patient, a large mass was successfully removed by a surgical treatment, so we agree that surgery is a good option if the thrombus is >2 cm as previously mentioned. In the second patient, because she was asymptomatic, a surgical treatment was not performed, but anticoagulant therapy was started. The patient's thrombus was completely dissolved without any complication such as an embolism.

In conclusion, we consider that further studies are warranted to determine whether to give anticoagulant therapy to renal transplant patients with a central venous catheter during the post-operative period. Further study is also needed to decide the best therapy for renal transplant recipients with cardiac thrombus.

Conflict of interest statement. None declared.

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