Renal Replacement Therapy

CASE REPORT

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Vascular access in a post-lung transplant patient on maintenance hemodialysis: a case report

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Abstract

Background An arteriovenous fistula (AVF) is the most common type of vascular access for hemodialysis. As it causes volume overload and sometimes increases pulmonary artery pressure, it is unsuitable for some patients. Herein, we describe a patient with acute kidney disease who required maintenance hemodialysis with vascular access other than an AVF owing to post-lung transplant pulmonary hypertension.

Case presentation A 50-year-old man with interstitial pneumonia underwent living-donor lobar lung transplantation at our hospital. Weaning from venoarterial extracorporeal membrane oxygenation was achieved; however, the patient required mechanical ventilation owing to pulmonary hypertension. He developed acute kidney disease and required maintenance hemodialysis with sustainable vascular access. Although echocardiography showed a normal ejection fraction, we expected volume overload after arteriovenous access construction to worsen his pulmonary hypertension because of his inadequate pulmonary vascular bed. Therefore, a tunneled central vein catheter was implanted into the right femoral vein as a bridge, and superficialization of the right brachial artery was performed for long-term vascular access.

Conclusions As this patient had sustained post-transplant pulmonary hypertension and small grafts, we avoided creating arteriovenous access because of concern over the aggravation of pulmonary hypertension. Evaluation of right heart function and pulmonary hypertension is important before arteriovenous access construction.

Keywords Vascular access, Pulmonary hypertension, Hemodialysis, Lung transplantation, Case report, Arteriovenous fistula

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Background

Vascular access plays an important role in hemodialysis, and an arteriovenous fistula (AVF) has been the preferred choice in terms of complication rate [1]. AVFs are also the most popular type of vascular access.

AVFs and arteriovenous grafts cause hemodynamic changes because they are artificially created with left-to-right shunts. Arteriovenous (AV) access leads to volume overload and subsequent cardiac muscle remodeling [2]. In clinical practice, new-onset or worsening heart failure

may occur in some patients. According to the guideline, a tunneled central vein catheter (CVC) should be considered when AV access fails, life expectancy is limited, or special medical reasons exist [3]. According to the Japanese guidelines for vascular access, use of a subcutaneously fixed superficial artery is recommended for patients with reduced ejection fraction as the outflow route or for tunneled CVCs [4]. However, risk assessment for AV access construction is insufficient in these guidelines, particularly regarding its effect on right heart function. The volume overload caused by AV access may result in right heart failure and pulmonary hypertension in some patients [5, 6].

Herein, we describe a patient with end-stage kidney disease (ESKD) who developed pulmonary hypertension after double-lung transplantation and was not a candidate for AV access.

Case presentation

A 50-year-old man was diagnosed with interstitial pneumonia (IP) 7 years ago. His medical history included a cervical spine injury due to a traffic crash. He was admitted to another hospital because of acute IP exacerbation. Despite various immunosuppressive therapies, the patient required mechanical ventilation and venovenous extracorporeal membrane oxygenation (VV-ECMO), and he was transferred to our hospital for lung transplantation. His preoperative serum creatinine level was 0.49 mg/dL (reference range, 0.65–1.07 mg/dL). The patient underwent living-donor lobar lung transplantation on day 5 after admission. He required postoperative venoarterial extracorporeal membrane oxygenation (VA-ECMO) because the lung grafts were small and he developed dyspnea and exacerbated pulmonary hypertension during weaning from ECMO. Owing to the surgical relief of the left pulmonary arterial stenosis and medication, weaning from VA-ECMO was achieved on day 45 after admission. However, his hemodynamics were unstable, and he developed acute kidney disease (AKD). The patient also experienced atelectasis-induced hypoventilation. Continuous renal replacement therapy and VV-ECMO were required. Weaning from VV-ECMO was achieved after 2 months; however, hemodialysis was still required because of prolonged anuria, which caused ESKD. He was referred to our department for sustainable vascular access for maintenance hemodialysis.

The patient was treated with mycophenolate, tacrolimus, and prednisone for post-transplant immunosuppression; however, he was placed on a ventilator. His vital signs and laboratory findings are presented in Table 1. Noradrenaline was used to maintain hemodynamics during dialysis without peripheral circulatory disturbance. Contrast-enhanced computed tomography revealed stenosis of the right internal jugular vein and fluid collection on the surface of the left femoral vein (Fig. 1a–d). Transthoracic echocardiography showed preserved ejection

Table 1 Vital signs and laboratory findings while the patient was on ventilator management and hemodialysis

Vital signs			
Height, 175 cm; weight, 74.8 kg			
State of consciousness, awake and alert			
PR, 99 /min; BP, 71/42 mm Hg; SpO_2, 95%			
(A/C mode, PCV; FiO_2, 30%; Ppeak, 18 cm H_2O; PEEP, 4 cm H_2O; RR, 25/min)			
Arterial blood gas analysis results		Blood chemistry results	
pH	7.302	Total protein, g/dL	5.8
PCO_2, Torr	50.7	Albumin, g/dL	2.9
PO_2, Torr	77.2	Uric acid, mg/dL	6.5
HCO_3^-, mmol/L	24.3	Blood urea nitrogen, mg/dL	109.2
Lactate, mmol/L	0.9	Creatinine, mg/dL	5.39
		Sodium, mmol/L	136
Peripheral blood results		Potassium, mmol/L	4.7
White blood cell count, /μL	4.8×10^3	Chloride, mmol/L	94
Hemoglobin, g/dL	9.4	Calcium, mg/dL	8.9
Platelet, /μL	8.2×10^4	Inorganic phosphorus, mg/dL	6.7
		Serology result	
		C-reactive protein, mg/dL	2.59

Pre-dialysis data from 2 days after the last dialysis are presented.

PR, pulse rate; BP, blood pressure; SpO_2, saturation of percutaneous oxygen; A/C, assist and control; PCV, pressure control ventilation; FiO_2, fraction of inspiratory oxygen; Ppeak, peak pressure; PEEP, positive end-expiratory pressure; RR, respiratory rate; pH, potential hydrogen; PCO_2, partial pressure of carbon dioxide; PO_2, partial pressure of oxygen; HCO_3^-, hydrogen carbonate

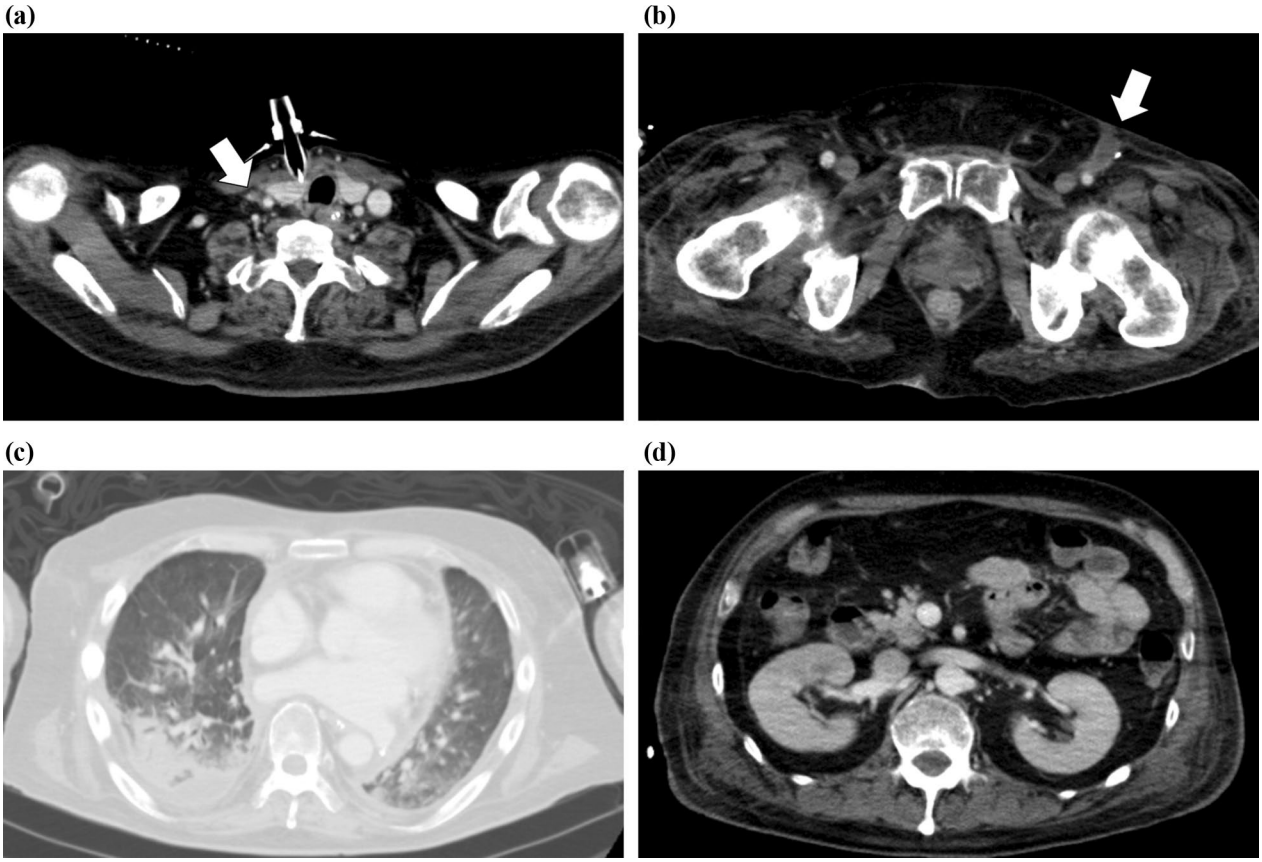

Fig. 1 Computed tomography images. **a** Stenosis of the right internal jugular vein (arrows). **b** Fluid collection on the surface of the left femoral vein (arrows). **c** Consolidation and atelectasis of the lungs. **d** Normal-sized kidneys

fraction of 65%. However, the patient showed an elevated right ventricular systolic pressure of 39–42 mmHg with right ventricular enlargement. Tricuspid annular plane systolic excursion was 11 mm and fractional area change in the right ventricle was 31%. Thus, he developed right heart failure even after volume reduction by hemodialysis.

On day 263 after admission, tunneled CVC implantation was performed in the right femoral vein, which was far from the tracheotomy site. The reasons for this were that placement of the CVC in the right internal jugular vein was difficult owing to vascular stenosis after ECMO insertion and the CVC in the left internal jugular vein was difficult to clear because of tracheotomy.

Superficialization of the right brachial artery was performed on day 340 after admission (Fig. 2a, b). Puncture of the artery was planned after the postoperative edema resolved. For approximately 5 months, the catheter could be used for maintenance hemodialysis without catheter occlusion or infection. The patient was transferred to the hospital for rehabilitation on day 423 after admission.

Discussion and conclusions

The present patient developed severe kidney injury requiring kidney replacement therapy (KRT) approximately 1 month after lung transplantation. He did not recover from this condition and required maintenance dialysis. Although no difference was reported in the survival of patients with ESKD after heart and lung transplantations between hemodialysis and peritoneal dialysis [7], hemodialysis was chosen for this patient owing to prolonged anuria, as well as cholecystitis and subcapsular liver abscess requiring drainage for 3 months. Cautious consideration for long-term vascular access is required due to the unique challenges posed by post-lung transplantation pulmonary hypertension. Because the lung grafts were small, they were thought to be intolerant of the volume overload triggered by AV access construction. Instead of AV access construction, tunneled CVC implantation was performed as a bridge, and a subcutaneously fixed superficial artery was constructed for long-term use. Since hemodynamics were able to be maintained under noradrenaline use during dialysis with

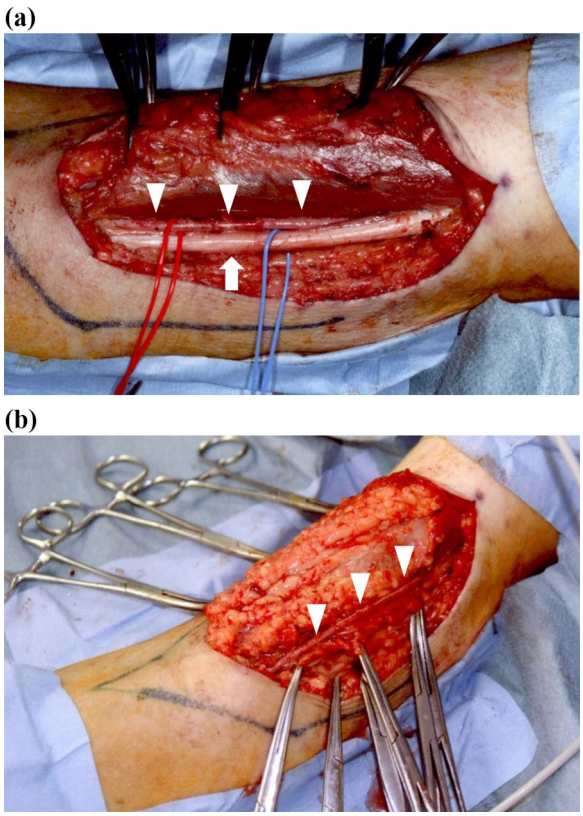

Fig. 2 Superficialization of the right brachial artery. **a** Right brachial artery (arrow heads) and median nerve (arrow) in situ. **b** Right brachial artery (arrow heads) lifted from the subcutaneous tissue

CVC, arterial superficialization provided tolerable vascular access.

Acute kidney injury (AKI) is a common complication after lung transplantation, accounting for 33–69% of cases. Approximately 5–13% of patients require KRT for severe AKI. Patients with severe AKI requiring KRT have a poor prognosis [8, 9]. Moreover, many patients with postoperative AKI transition to chronic kidney disease (CKD) [10]. Many risk factors, including renal hypoperfusion, sepsis, and nephrotoxic agents, contribute to AKI after lung transplantation [8, 11].

Pulmonary hypertension has been defined as the presence of mean pulmonary artery pressure of ≥ 25 mmHg at rest measured by right heart catheterization. Noninvasive evaluation of pulmonary hypertension is often performed using echocardiography to estimate pulmonary artery systolic pressure instead of right heart catheterization. The prevalence of pulmonary hypertension in patients with CKD stage 5 ranges from 9% to 39% [5, 6, 12, 13]. Pulmonary hypertension is an independent predictor of all-cause mortality and cardiovascular events in both patients with CKD and those on hemodialysis [5, 14]. Various factors are associated with the pathophysiology of pulmonary hypertension in patients with CKD. Although the most common cause of pulmonary hypertension is left heart failure, there are many other factors, including anemia, coexisting pulmonary disease, and sleep apnea syndrome. Moreover, previous studies have reported elevated levels of endothelin (a pulmonary artery vasoconstrictor), decreased levels of nitric oxide (a pulmonary artery vasodilator), and calcification and remodeling of the pulmonary artery [15]. As AV access is a left-to-right shunt that bypasses capillary beds, it provokes increased cardiac output and volume overload of the right heart system, which exacerbates pulmonary hypertension [2]. However, the pulmonary vascular bed is so vast that elevated nitric oxide production in response to increased pulmonary blood flow maintains a normal vascular tone [16]. Pulmonary artery pressure is supposed to increase when there is more than two-thirds of loss of pulmonary microcirculation [17]. Therefore, AV access may contribute to one of the factors of pulmonary hypertension with CKD. Many guidelines on vascular access mention this negative effect, i.e., high-output heart failure [3, 4, 18], but further discussion about pulmonary hypertension associated with AV access is needed.

The present patient underwent a living-donor lobar lung transplantation. Pulmonary hypertension was a problem when we attempted to wean the patient off of ECMO support. Owing to surgical relief of the left pulmonary arterial stenosis and treatment with pulmonary vasodilators, weaning from ECMO was achieved; however, pulmonary hypertension persisted. Regarding permanent vascular access, the ejection fraction was within the normal range. Because the estimated pulmonary artery systolic pressure was elevated, the increased preload caused by AVF construction was believed to have worsened his pulmonary artery hypertension, which might have been life-threatening. Therefore, we decided to use other types of vascular access instead of AV access.

An important point regarding his pulmonary hypertension was that the graft size was small for his body size. Although size matching is performed before transplantation, it is not always sufficient for living-donor lobar lung transplantation. The inadequate pulmonary vascular bed was unique to this patient. Previous studies have reported the possibility of a decrease in the pulmonary vascular bed with regard to other pulmonary hypertensions caused by lung disease. This decrease in the pulmonary vascular bed is due to the exclusion and occlusion of small arteries and capillaries due to lung parenchymal damage [19–21]. In such cases, increased blood flow into the pulmonary vascular bed may cause pulmonary hypertension.

Risk factors for progression from AKI to CKD include severity of kidney injury, chronic diseases

(e.g., hypertension, diabetes, and obesity), and non-modifiable risk factors (e.g., genetics, advanced age, and sex) [22]. Lung transplant recipients are at risk for AKI and CKD. Regarding risk of AKI after lung transplantation, a retrospective analysis at a single center reported that women and pulmonary hypertension are potential risk factors for AKI after lung transplantation [23]. Another study reported that patients with cystic fibrosis who have undergone lung transplantation frequently develop AKI, which often progresses to CKD, resulting in worse short- and long-term outcomes [10]. Another single-center retrospective analysis reported that persistent AKI was associated with preoperative pulmonary artery hypertension, severe hypotension, postoperative multiorgan failure, and nephrotoxic agents [24]. Although the cause of AKD and the definite mechanism that led to ESKD in the present case remain unknown, we assume that several factors, including preoperative pulmonary artery hypertension, small graft size, hypotension, postoperative sepsis, and many drugs, contributed to the development of ESKD.

In summary, we described a post-lung transplantation patient on maintenance hemodialysis. As this patient had sustained post-transplant pulmonary hypertension and received small grafts, we avoided creating AV access because of concern over the aggravation of pulmonary hypertension. It is important to evaluate not only the left heart function, but also the right heart function and pulmonary hypertension before AV access construction.

Abbreviations

AVF	Arteriovenous fistula
AV	Arteriovenous
CVC	Central vein catheter
IP	Interstitial pneumonia
VV-ECMO	Venovenous extracorporeal membrane oxygenation
VA-ECMO	Venoarterial extracorporeal membrane oxygenation
AKD	Acute kidney disease
KRT	Kidney replacement therapy
AKI	Acute kidney injury
CKD	Chronic kidney disease
ESKD	End-stage kidney disease

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Author contributions
M.K. and M.N. designed and performed the study and drafted the manuscript. R.M., M.O., Y.K., M.S., D.Y., Y.H., C.K., M.S., and H.K. treated the patient. M.N. supervised the study. The final manuscript has been read and approved by all authors.

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Availability of data and materials
All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval and consent to participate
This study was approved by the Research Ethics Committee of the Faculty of Medicine of the University of Tokyo (approval no.: 2879). Consent to participate was not applicable.

Consent for publication
Informed consent of the patients involved was provided about the publication of this article.

Competing interests
The authors declare that they have no competing interests.

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