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Coronary sinus endocarditis in a hemodialysis patient: A case report and review of literature

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Abstract

BACKGROUND

Infective endocarditis is more common in hemodialysis patients than in the general population and is sometimes difficult to diagnose. Isolated coronary sinus (CS) vegetation is extremely rare and has a good prognosis, but complicated CS vegetation may have a poorer clinical course. We report a case of CS vegetation accidentally found *via* echocardiography in a hemodialysis patient with undifferentiated shock. The CS vegetation may have been caused by endocardial denudation due to tricuspid regurgitant jet and subsequent bacteremia.

CASE SUMMARY

A 91-year-old man with dyspnea and hypotension was transferred from a nursing hospital. He was on regular hemodialysis and had a history of severe grade of tricuspid regurgitation. There was no leukocytosis or fever upon admission. Repetitive and sequential blood cultures revealed absence of microorganism growth. Chest computed tomography showed lung consolidation and a large pleural effusion. A mobile band-like mass on the CS, suggestive of vegetation, was observed on echocardiography. We diagnosed him with infective endocarditis involving the CS, pneumonia, and septic shock based on echocardiographic, radiographic, and clinical findings. Infusion of broad-spectrum antibiotics, fluid resuscitation, inotropic support, and ventilator care were performed. However, the patient died from uncontrolled infection and septic shock.

CONCLUSION

CS vegetation can be fatal in hemodialysis patients with impaired immune systems, especially when it delays the diagnosis.

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Core Tip: Coronary sinus vegetation is rarely observed. It might also be confused with a coronary sinus thrombosis and inappropriately managed. We present a case of coronary sinus vegetation with typical echocardiographic findings and review clinical challenges, including differential diagnoses and therapeutic options, associated with treating hemodialysis patients suspected to have coronary sinus endocarditis.

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INTRODUCTION

Infective endocarditis in hemodialysis patients is more common and has higher morbidity and mortality than that in the general population[1,2]. Although infective endocarditis in hemodialysis patients occurs due to vascular access-related infection during dialysis, it mainly involves left-sided heart structures, while right-sided structures are rarely affected[2]. Right-sided infective endocarditis (RIE) accounts for 5%-10% of all cases of infective endocarditis and frequently affects intravenous drug users or patients with central venous catheters or intracardiac devices[3]. RIE usually involves the tricuspid or pulmonary valves and often leads to secondary right heart failure[3]. This report shows an elderly hemodialysis patient with coronary sinus (CS) endocarditis who presented with septic shock. CS endocarditis has rarely been reported and usually has a good prognosis[4], but it was fatal in our case. We review the clinical challenges that caused a poor clinical outcome in our case, including differential diagnosis and therapeutic options in hemodialysis patients with endocarditis.

CASE PRESENTATION

Chief complaints

A 91-year-old male patient admitted to a nursing hospital was transferred to our hospital with dyspnea, cough, and a confused mental state.

History of present illness

Prior to patient transfer, a single dose of moxifloxacin was administered at the nursing hospital without blood culture tests. Oxygen was administered *via* a nasal cannula during the transfer.

History of past illness

The patient has undergone regular hemodialysis for one year. He had a medical history of atrial fibrillation, severe grade of tricuspid and mitral regurgitation, and chronic obstructive pulmonary disease. He also had a stroke 5 years previously.

Physical examination

At the time of admission in our institution, blood pressure, heart rate, respiratory rate, and body temperature of the patient were 85/40 mmHg, 110 /min, 20 /min, and 36.5 °C, respectively.

Laboratory examinations

Laboratory tests revealed a white blood cell count of 8780 /μL (neutrophils: 78%), C-reactive protein level of 1.6 mg/dL (normal range < 0.5 mg/dL), procalcitonin level of 0.571 μg/L (normal range < 0.046 μg/L), creatinine level of 5.6 mg/dL (glomerular

filtration rate by the modification of diet in renal disease study equation = 10 mL/min/1.73 m²), and brain natriuretic peptide level of 124 ng/L (normal range < 100 ng/L). Arterial blood gas analysis during oxygen supply revealed severe acidosis with a pH of 7.173, partial pressure of carbon dioxide of 66 mmHg, partial pressure of oxygen of 100 mmHg, and bicarbonate of 24 mEq/L.

Imaging examinations

Chest radiography revealed a large right pleural effusion and pulmonary edema (Figure 1A). Chest computed tomography showed consolidation in the right upper and middle lobes and total atelectasis of the right lower lobe (Figure 1B). The pleural effusion was a transudate, according to the following Light's criteria[5]: Pleural fluid protein-to-serum protein ratio of 0.4; pleural fluid lactate dehydrogenase (LDH) -to-serum LDH of 0.6; and pleural fluid LDH to the upper normal limit for serum of 0.6. There was no growth of microorganisms on pleural fluid culture. Transthoracic echocardiography showed a moderate grade of eccentric tricuspid regurgitant jet flow directed towards the CS and a mobile band-like echogenic mass (longitudinal dimension of approximately 8 cm) attached to the ostium of the CS and posterolateral wall of the right atrium, suggesting the presence of vegetation (Figure 2 and Supplementary Videos 1-3, which demonstrate a vegetation on echocardiography). He also had a moderate grade of pulmonary hypertension (estimated systolic pulmonary arterial pressure = 61 mmHg) and right atrial enlargement (30 cm²). Other cardiac structures, including the valves, were unaffected, and the left ventricular ejection fraction was normal. There was no radiographic evidence of pulmonary thromboembolism or deep vein thrombosis. Sequential blood culture results were negative.

FINAL DIAGNOSIS

The patient was diagnosed with septic shock due to CS endocarditis and pneumonia. Respiratory acidosis with metabolic decompensation was accompanied by severe infection, acute exacerbation of chronic obstructive pulmonary disease, hypoventilation, and a large pleural effusion.

TREATMENT

Broad-spectrum antibiotics, including teicoplanin and meropenem, were administered. A chest tube was inserted at the site of pleural effusion. A mechanical ventilator and continuous renal replacement therapy were administered. Fluid resuscitation and inotropic support were provided to maintain blood pressure.

OUTCOME AND FOLLOW-UP

Hypercapnia and respiratory acidosis were improved to some extent after insertion of the chest tube and application of the mechanical ventilator (pH = 7.347, partial pressure of carbon dioxide = 41 mmHg, partial pressure of oxygen = 97 mmHg, and bicarbonate = 22 mEq/L on arterial blood gas analysis). Follow-up echocardiography after one week showed remnants of vegetation (Figure 3), suggesting migration of the cardiac vegetation into the lung. There was no abrupt hypoxic event that could lead to deterioration. However, leukocytosis and metabolic and respiratory acidosis worsened over time [white blood cell count, 33830 /μL (neutrophils, 90%); pH = 7.160, partial pressure of carbon dioxide = 52 mmHg, partial pressure of oxygen = 73 mmHg, bicarbonate 18 mEq/L, lactic acid = 3.6 mmol/L on arterial blood gas analysis]. Septic shock persisted despite medical support and he eventually died.

DISCUSSION

The CS is a structure that receives approximately 60% of the total cardiac venous supply and drains into the right atrium[6]. It is mainly injured during right cardiac procedures, including insertion of central venous catheter, cannulation of the CS during heart surgery, electrophysiologic study, and placement of intracardiac devices

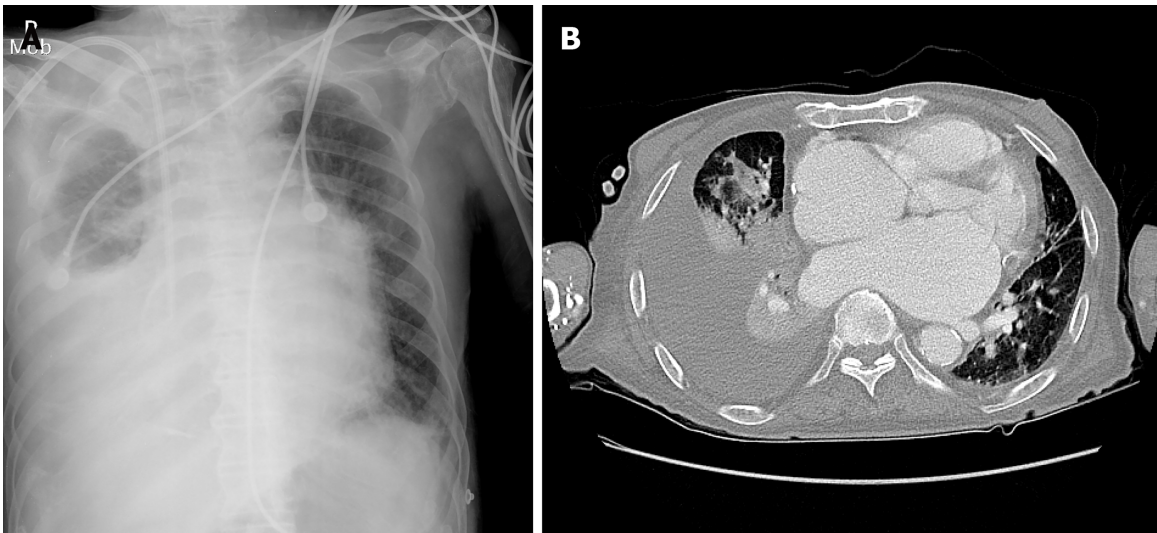


Figure 1 Radiographic imaging. A: Chest radiograph showing a large right pleural effusion and pulmonary edema; B: Chest computed tomography showing consolidation of the right middle lobe, total atelectasis of the right lower lobe, and a large right pleural effusion.

[6]. Unlike traumatic injury, spontaneous endothelial injury of the CS has been reported in some conditions causing accelerating or turbulent jet flow in the CS, such as coronary arteriovenous fistula between the left circumferential artery and CS and eccentric tricuspid regurgitation towards the CS[4,7-9]. In our case, the substantial tricuspid regurgitant jet flow directed towards the CS and posterolateral right atrial surface might have caused endothelial denudation; subsequently, bacteremia during hemodialysis or sepsis due to pneumonia probably led to the formation of vegetation at that location.

Vegetation of the CS resembles thrombosis in appearance. However, both need to be distinguished because they require different treatments. CS vegetation, including infective endocarditis or septic thrombophlebitis, has been reported in 13 cases, including our case (Table 1)[4,7-17]. According to these cases, patients with CS vegetation mainly had symptoms and signs of infection, such as fever and leukocytosis, and had typical imaging or pathologic findings involving the CS. In rare cases [7,14], blood culture results were negative; however, pathogens were identified on tissue culture. The major pathogen was *Staphylococcus aureus* (four in infective endocarditis cases and three in septic thrombophlebitis) which is associated with complicated clinical courses, including huge vegetation, destructive abscess and fistulation, and septic embolization[3]. Indeed, five (71%) of the seven cases with *Staphylococcus aureus* infection underwent surgery or died suddenly. Patients with complex CS vegetation, including septic embolism and tissue destruction leading to heart failure, underwent surgery or died before treatment, while patients with isolated CS vegetation without complications recovered with only antibiotics. Septic thrombophlebitis has both characteristics of thrombosis and infection of the CS. It was fatal when there was acute mechanical obstruction of the CS[14,15]. Similar to CS vegetation, spontaneous CS thrombosis without iatrogenic injury has been reported in less than 15 cases. CS thrombosis mostly occurred in conditions leading to a static status of the right atrium, including atrial fibrillation[18-21], right heart failure with severe pulmonary hypertension[18,19,22,23], or hypercoagulable conditions such as severe inflammation/infection[20,24-26] and malignancy[27]; this presentation is similar to that of thrombosis at other sites. Patients with CS thrombosis primarily have symptoms and signs associated with partial or complete obstruction of the CS, but no symptoms and signs of infection. For example, chest pain, ST-segment elevation[28], or hemodynamic collapse and subsequent sudden death[22,24,25,27] have been reported. CS thrombosis with complete obstruction required emergency surgery[19,26]; however, thrombosis without obstructive signs responded to anti-coagulation therapy [18,20].

Although vegetation or thrombosis of the CS is confirmed based on histopathological findings, echocardiography is the best imaging technique for screening. However, minor structures of the right heart, such as the CS and Chiari network, are sometimes overlooked during echocardiography. This might be one of the reasons why cases were rarely reported. In our case, infective endocarditis was not suspected before echocardiography because typical microorganisms did not grow in blood

Table 1 Cases of infective endocarditis and septal thrombophlebitis involving the coronary sinus reported in the literature

Ref.	Age/sex	Symptoms	Characteristics of vegetation in the CS	Associated pathology	Pathogen	Therapy	Events
Cases of infective endocarditis							
Takashima <i>et al</i> [7], 2016	64/M	Fever, fatigue	A sessile mass with mobile multi-lobules on the CS lumen	CAVF, vegetation on the MV and AV with moderate regurgitation, acute HF	Negative results in BC, <i>Corynebacterium</i> species in TC	Surgery	Multi-organ failure, DIC, died
Kasravi <i>et al</i> [8], 2004	31/M	Fever, pleuritic chest pain	A mobile and multi-lobulated mass protruding from the CS to the RA	CAVF	MSSA in BC	Surgery	SE (lung) and DIC, recovered
Song <i>et al</i> [4], 2018	71/M	Fever, chest pain, hemoptysis	A banded mobile mass in the CS	ASD, PLSVC, severe eccentric TR jet to the CS, RV dysfunction with RAE, moderate PHT	MSSA in BC	Surgery	SE (lung), recovered
Kumar <i>et al</i> [10], 2016	27/F	Septic shock	A pedunculated mobile mass 1 cm proximal from the CS orifice to the Eustachian valve	IVDU	MSSA in BC	Antibiotics	SE (lung, viscera), recovered
Machado <i>et al</i> [11], 2010	44/M	Fever, dyspnea	A mobile mass originating in the CS orifice, extending to the RA	Purulent pericardial effusion	MSSA in BC	Surgery	Recovered
Gill <i>et al</i> [12], 2005	37/M	Fever, weight loss	A mobile mass in the CS and CAVF	CAVF	<i>Streptococcus mitis</i> in BC	Antibiotics	Recovered
Theodoropoulos <i>et al</i> [9], 2016	28/F	Fever, hemoptysis	Two mobile masses towards the CS orifice and in the CS lumen	IVDU, eccentric moderate TR jet to the CS	Group C <i>Streptococcus</i> in BC	Antibiotics	Recovered
Kwan <i>et al</i> [13], 2014	23/F	Fever	A mobile round mass protruding from the CS orifice	HD	<i>Acinetobacter baumannii</i> in BC	Antibiotics	Recovered
Our case	91/M	Septic shock	A mobile band-like mass protruding from the CS orifice	HD, eccentric moderate TR jet to the CS	Negative results in BC	Antibiotics	Died
Cases of septic thrombophlebitis							
Ross <i>et al</i> [14], 1985	31/M	Fever, dyspnea	Occlusion of the CS orifice by fungal thrombi (in necropsy)	Lymphoma, occlusion of the LCA by fungal thrombi (in necropsy)	Negative results in fungal culture, <i>Aspergillus fumigatus</i> in the lung, LCA and CS	Antibiotics	Died
Dryer <i>et al</i> [15], 1976	20/M	Fever, disturbed mental state	Occlusion of the CS orifice by septic thrombophlebitis (in necropsy)	IVDU, vegetation on the MV, multi-organ embolic infarction (in necropsy)	MSSA in BC	Antibiotics	SE (multi-organs), died
Jones <i>et al</i> [16], 2004	50/M	Fever	A mass protruding from the CS orifice to the RA, and extending to the posterior interventricular vein	Previous pericardiectomy due to purulent pericarditis, recurrent furunculosis	MSSA in BC	Surgery	SE (lung), recovered
Fournet <i>et al</i> [17], 2014	38/F	Fever, chest pain,	A mobile mass originating from the CS ostium with heterogeneous solid material	Purulent pericardial effusion	MSSA in BC	Antibiotics	SE (lung), recovered

CS: Coronary sinus; M: Male; F: Female; CAVF: Coronary arteriovenous fistula between left circumferential artery and coronary sinus; MV: Mitral valve; AV: Aortic valve; HF: Heart failure; BC: Blood culture; TC: Tissue culture; DIC: Disseminated intravascular coagulation; RA: Right atrium; MSSA: Methicillin-sensitive *Staphylococcus aureus*; SE: Septic embolism; ASD: Atrial septal defect; PLSVC: Persistent left superior vena cava; TR: Tricuspid regurgitation; RV: Right ventricle; RAE: Right atrial enlargement; PHT: Pulmonary hypertension; IVDU: Intravenous drug user; HD: Hemodialysis; LCA: Left coronary arteries including left anterior descending and left circumflex arteries.

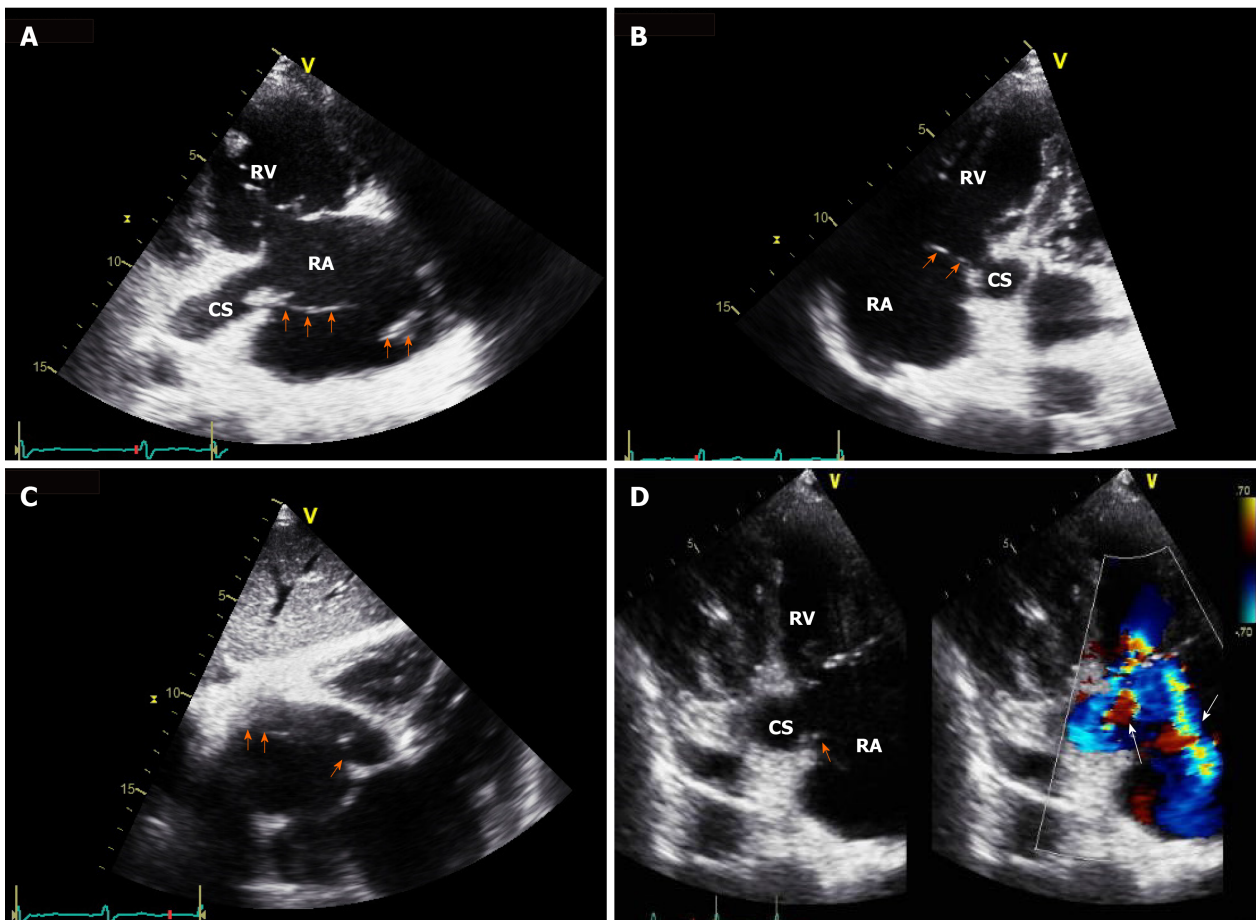


Figure 2 Echocardiographic imaging. A: Right ventricular inflow view showing a mobile band-like vegetation, approximately 8 cm in size, attached to the coronary sinus ostium and the posterolateral wall of the right atrium; B: Modified apical four-chamber view showing a vegetation attached to the ostium of the coronary sinus; C: Subcostal view showing a vegetation; D: Right ventricular inflow view showing eccentric tricuspid regurgitant jet flow directed towards the coronary sinus and concentric tricuspid regurgitant jet flow directed towards the posterolateral wall of the right atrium, observed through color Doppler imaging, and an attached vegetation at the site, observed via two-dimensional imaging. Orange arrow indicates vegetation; White arrow indicates directed tricuspid regurgitant jet flow. RA: Right atrium; RV: Right ventricle; CS: Coronary sinus.

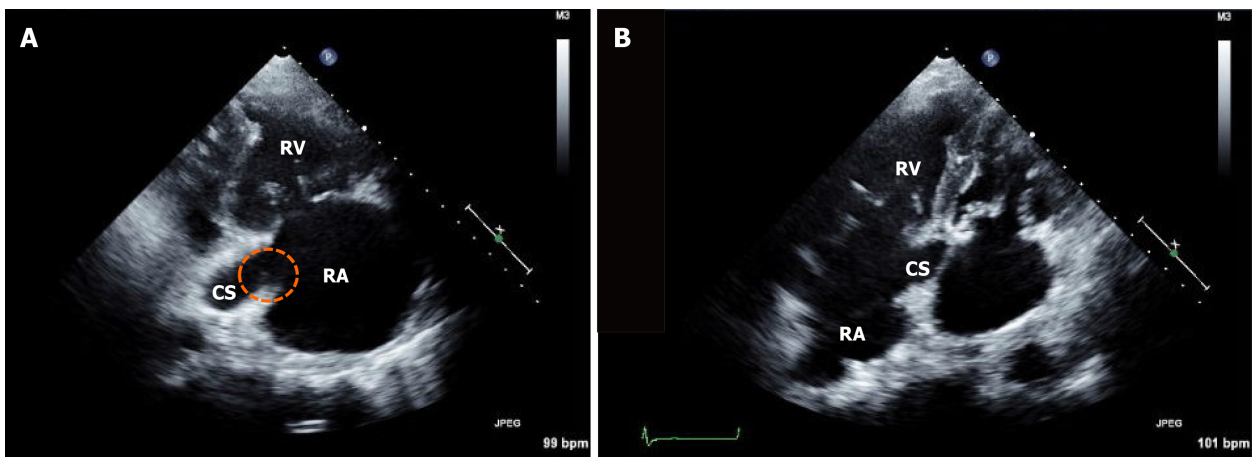


Figure 3 Follow-up echocardiographic imaging. A: Right ventricular inflow; B: Modified apical four-chamber views showing a remnant vegetation at the coronary sinus ostium (dotted circle). RA: Right atrium; RV: Right ventricle; CS: Coronary sinus.

cultures, and only non-specific hypotension and mild elevation of C-reactive protein and procalcitonin levels were observed without typical infection signs. In fact, echocardiography was performed to screen for cardiogenic shock, and CS vegetation was incidentally observed. We immediately started broad-spectrum antibiotics for infective endocarditis and found no vegetation on the follow-up echocardiography. If the

echogenic mass had continued to increase, it might have caused other clinical problems due to mechanical obstruction or destruction of structures adjacent to the CS. Although immediate treatment with antibiotics did not alter the prognosis of this patient, early detection of CS vegetation is essential for appropriate management.

The mortality rate of RIE including CS endocarditis[4] is considered to be lower than that of left-sided infective endocarditis (LIE)[3]. However, a high proportion (60%-80%) of intravenous drug users, who generally display a benign clinical course, might account for the good prognosis of RIE in previous studies[29,30]. Indeed, recent studies that enrolled patients with no history of intravenous drug use or with cardiac devices showed similar in-hospital mortality rates between RIE and LIE[31,32]. Furthermore, hemodialysis patients with inherently impaired immune systems are highly susceptible to bacteremia and septic shock. In our case, the patient died because his immune systems were impaired due to old age, chronic renal impairment, fragility, and severe infection. Moreover, the diagnosis of infective endocarditis was delayed due to atypical symptoms. His negative blood culture results might also be associated with poor outcome because it made antibiotic selection difficult.

The diagnosis and treatment of infective endocarditis in hemodialysis patients is challenging. Clinical manifestations, including fever and leukocytosis, may be atypical because of impaired cellular host defense; thus, diagnosis or management of infective endocarditis may be delayed[2]. Indwelling intravascular catheters may be a primary source of pathogens; however, removal of the catheter is not always possible or necessary[2]. Infective endocarditis caused by methicillin-resistant *Staphylococcus aureus* is a common clinical entity. However, empirical use of vancomycin before the isolation of microorganisms should be determined carefully because vancomycin has a slower bactericidal effect on methicillin-susceptible *Staphylococcus aureus* compared to beta-lactam antibiotics[2,33].

CONCLUSION

Infective endocarditis is more common and has a poorer prognosis in hemodialysis patients than it does in the general population. Thus, echocardiography should be thoroughly investigated in hemodialysis patients suspected of having sepsis or shock. In addition, CS vegetation is easy to be misdiagnosed when the clinician is not cautious or inexperienced. The treatment of infective endocarditis in hemodialysis patients is more challenging when blood cultures are negative. More careful consideration and a team-based approach involving cardiologists, nephrologists, and infectious disease specialists, is needed for the treatment of infectious diseases in hemodialysis patients.

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