CASE PRESENTATION

Septic pulmonary emboli secondary to an infected indwelling haemodialysis catheter in a patient with chronic kidney disease

Haider Noori Dawood,[a] Ehsan Hassan Karim,[b] Jabbar S. Hassan,[c]

ABSTRACT

Septic pulmonary embolism (SPE) is a rare but serious complication of bloodstream infections, particularly in patients with indwelling vascular catheters. The case report describes a 39-year-old male with chronic kidney disease undergoing haemodialysis via a non- tunnelled catheter who developed septic pulmonary emboli. He presented with fever, chills, sweating, rigours, and anorexia, then developed dyspnoea, pleuritic chest pain, followed by cough, sputum, and haemoptysis. Imaging studies, including a CT scan of the chest, showed bilateral pleural effusion and multiple thick-walled cavitary lesions with an enhancing nodule. The echocardiogram revealed that the catheter had reached the right atrium, with a mass attached to it (thrombus). Investigation showed elevated inflammatory markers (CRP and ESR), anaemia, and neutrophilic leucocytosis. Although blood cultures were negative, possibly due to prior antibiotic use, the catheter tip culture grew Staphylococcus aureus. The patient was treated with appropriate antibiotics based on sensitivity testing, along with immediate removal of the infected catheter and insertion of a new one at a different site. His fever and dyspnoea subsided, and he was discharged to continue regular haemodialysis. This case highlights the importance of early recognition of catheter-related bloodstream infection and prompt management to prevent complications such as septic pulmonary emboli..

Key words: Septic pulmonary embolism; Haemodialysis catheter; chronic kidney disease; Catheter-related bloodstream infection.

INTRODUCTION

Septic pulmonary embolism (SPE) is an uncommon complication in which infected thrombi from a primary infectious site lead to infarctions in the pulmonary vasculature. [1] In adult patients, this complication is often reported in association with long-term use of central venous catheters for chemotherapy, parenteral nutrition, or haemodialysis.[2] Imaging studies play a pivotal role in diagnosing intracardiac masses, providing valuable insights for timely medical or surgical treatment.[3]

The radiographic features of pulmonary SE have been described; in general, computer tomography (CT) seems to be more sensitive than chest radiography early in the course of infection and may reveal different features, including multiple peripheral nodules, feeding vessel signs, pleural-butting, wedge-shaped peripheral lesions, cavity formation, and pleural effusion.[3]

In this case, we aim to highlight the critical importance of recognising potential life-threatening complications associated with the use of haemodialysis catheters. The presented patient's catheter dependence led to the development of a mass in the tip of the catheter that caused multiple septic pulmonary emboli.

CASE PRESENTATION

A 39-year-old male farmer living in Al-Taji, north of Baghdad, with his wife and three sons, was admitted to Al-Imamain Al-Kadhimain

a: MBChB, FIBM, FABM, FIBM (Resp.). Consultant Respiratory Physician. Al-Emamain Al-Kadhmain Medical City, AL-Karkh Health Directorate, Baghdad, Iraq b: MBChB, Al-Emamain Al-Kadhmain Medical City, AL-Karkh Health Directorate, Baghdad, Iraq. c: Department of Medical Microbiology, College of Medicine, Al-Nahrain University, Iraq.

***Corresponding Author:** **Haider Noori Dawood**, E-mail: **haidernoori2001@gmail.com**.*

Medical City on 22 April 2025 with shortness of breath for 2 days. The patient was a known case of end-stage renal disease on scheduled haemodialysis (HD) through a non-tunnelled catheter. He was doing well till the last three weeks, when he suffered from chills during dialysis for a few days. Then his condition progressed over time to be continuous, documented, high body temperature associated with sweating, rigour especially at night, fatigue, decreased appetite and weight loss of two kilograms. He received multiple courses of antibiotics and antipyretics, without response. Four days before admission, the patient developed acute-onset shortness of breath that progressively became severe enough to limit his daily activities and was present even at rest, prompting him to seek medical attention. The dyspnoea was associated with sharp, nonradiating right-sided chest pain of moderate severity, pleuritic in nature, worsening with coughing and deep inspiration, and a productive cough with foul-smelling, yellowish, and blood-tinged sputum. He denied a history of orthopnoea, paroxysmal nocturnal dyspnoea, syncope, palpitations, or dizziness.

He reported no recent contact with anyone with similar symptoms. Other systemic reviews were unremarkable, except for decreased urine output. The patient has a history of unknown end-stage renal failure, and he is on regular haemodialysis (HD) of three sessions per week. He was admitted to the hospital many times during the last month due to elevated body temperature. He reported no surgical intervention other than double-lumen insertion and planning for creation of an arteriovenous (AV) fistula. His mother also has end-stage renal failure, which could not be attributed to an obvious cause despite thorough investigations, and was kept on regular HD. He lives in a small house with his family in a rural area and has a low socioeconomic status. He denied a history of smoking, alcohol consumption or similar symptoms in his family.

Examination: A Middle-aged male lying in bed, conscious, oriented to time, place, and person. Looks ill, pale, dyspneic, tachypneic with an intravenous (IV) line in his hand and a non-tunnelled catheter placed on the right side of his neck without obvious redness, discharge or tenderness. No jaundice, cyanosis, clubbing, tremor, lymphadenopathy, splinter haemorrhage, Janeway lesion or Osler node. There is bilateral pitting oedema extending to the mid-shin. The vital signs showed a regular, large-volume, noncollapsing pulse at 130 bpm, without delay. Blood pressure was 135/70, SPO2 was 93% without oxygen, respiratory rate was 25/min, and corrected axillary temperature was 39.8 °C. Respiratory examination showed Limited chest expansion on the right, dullness to percussion with reduced vocal fremitus on the same side, with the trachea centrally located. Chest auscultation showed normal vascular breathing, with absent air entry in the right lower zone, and coarse chest crepitation that did not change with cough on the right. Cardiac examination showed normal S1 and S2, no added sounds. There is a pansystolic murmur best heard at the apex, radiating to the axilla, and the JVP was not raised.

Investigations: for the results of the haematological,biochemical,andbacteriological laboratory tests on the blood and pleural fluid, see **Table 1**. Patient's chest X-ray shows multiple lesions with rt sided pleural effusion, **Figure 1**. The chest CT scan shows bilateral multiple cavitary and non-cavitary lesions with bilateral pleural effusion mainly on the right, **figure 2**. The fluid was aspirated, yielding a bloody fluid.

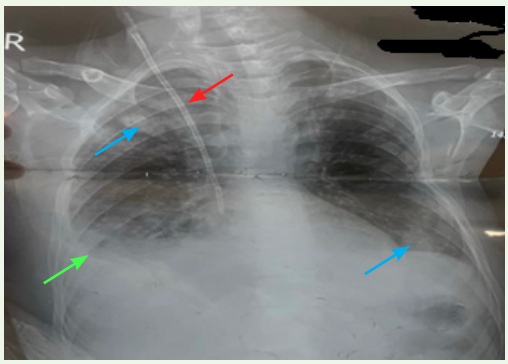

Figure 1 | Chest X-ray of the patient, PA view shows an intravenous catheter (central Line) on the right side, red arrow. The tip of the catheter is in the right heart chambers, with a right-sided pleural effusion (meniscus sign) (green arrow). Although the film was not taken in full inspiration, we can clearly see at least two opacities (blue arrows): the first in the upper zone of the right lung and the second in the pleural-based lower zone of the left lung. .

Table 1 | The results of haematological, bacteriological, and biochemical tests conducted on admission of the patient to the hospital.

Full Name		Result	Units	Normal Range
Blood Aspirate				
Hemoglobin	Hb	4.8	g/dL	Male: 13.5–17.5 g/dL; Female: 12.0–15.5 g/dL
Mean Corpuscular Volume	MCV	97	fL	80–100 fL
White Blood Cell Count	WBC	28.7	$\times 10^3/\mu L$	$4.0–11.0 \times 10^3/\mu L$
Platelet Count	PLT	238	$\times 10^3/\mu L$	$150–450 \times 10^3/\mu L$
C-Reactive Protein	CRP	173	mg/L	<5 mg/L
Erythrocyte Sedimentation Rate	ESR	80	mm/hr	Male: 0–15 mm/hr; Female: 0–20 mm/hr
Random Blood Glucose	RBS	96	mg/dL	70–140 mg/dL
Blood Urea	BU	140	mg/dL	15–40 mg/dL
Serum Creatinine	SCr	8.15	mg/dL	0.6–1.3 mg/dL
Serum Sodium	Na	124	mmol/L	135–145 mmol/L
Serum Potassium	K	5.6	mmol/L	3.5–5.0 mmol/L
Serum Chloride	Cl	87.5	mmol/L	98–106 mmol/L
Alanine Aminotransferase	ALT	61	U/L	7–56 U/L
Aspartate Aminotransferase	AST	73.2	U/L	10–40 U/L
Prothrombin Time	PT	15	seconds	11–13.5 seconds
Partial Thromboplastin Time	PTT	27.5	seconds	25–35 seconds
Blood culture	Sputum culture: Growth of Candida, sputum Gram Stain: Gram-positive diplococci, sputum AFB: NEGATIVE.			
Plural fluid aspirate	Colour: bloody, LDH: 298 U/L, total protein: 3.4 g/dl, glucose: 102mg/dl, ULTRA, Gene Expert for Mycobacterium Tuberculosis (MTB) was negative: Not Detected			
Catheter tip culture	Staphylococcus aureus with > 16 CFUs.			

The biochemical results are shown in Table 1. Transthoracic echocardiographic examination showed that the catheter is inserted into the right atrium with a big thrombus attached to its tip, in addition to severe mitral regurgitation, Figure 3.

Diagnosis: Septic pulmonary emboli secondary to catheter-related bloodstream infection.

Therapeutic Intervention: We started antibiotics: Meropenem 500 mg twice daily and vancomycin 1000 twice weekly, adjusted according to renal function. Catheter removed immediately after echo diagnosis of the tip thrombus and insertion in another site. After two days, the fever and dyspnea began to decrease and resolved progressively. The

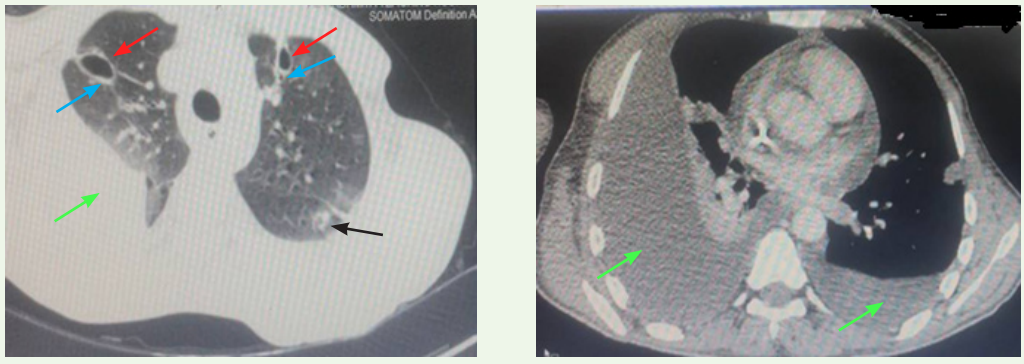

Figure 2 | A: Non-contrast CT scan of chest with lung window shows 2 cavitary lesions involving both lungs' upper lobes anterior segments, red arrows, with surrounding ground glass opacity. The lesions have feeding arteries indicated by blue arrows. Another opacity appears in the upper segment of the left lower lobe, indicated by the black arrow. The green arrow indicates a right-sided pleural effusion. B: CT scan of chest (mediastinal window) shows bilateral pleural effusion (green arrow) mainly on the right side with collapsed right lung, and pericardial effusion.

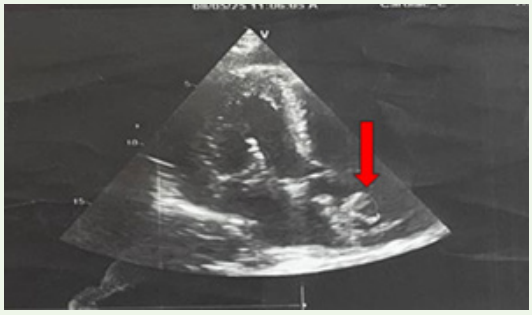

Figure 3 | Transthoracic echocardiographic four-chamber view shows a mass (thrombus) attached to the HD catheter that reached the right atrium, with severe mitral regurgitation.

patient was discharged home in good health and continued on regular HD.

DISCUSSION

There are increased risks of SPE in patients with chronic kidney disease because of decreased immunity and frequent venous puncture with the use of indwelling venous catheters or arterio-venous graft (AVG). In a study in Japan analysing 11,367 cases of pulmonary embolism identified on postmortem examination, SPE was identified in 247 (2.2%).[5] Septic emboli can originate from different sources; one source is infected catheters/lines, such as venous lines (e.g., central venous catheters).[6]

Our patient, who is a known case of end-stage renal disease on scheduled haemodialysis (HD) through a non-tunnelled catheter, presented with fever, rigour, anorexia and weight loss, then developed chest pain, dyspnea, followed by cough, sputum, and hemoptysis. The characteristics of septic pulmonary embolism (SPE) at presentation are nonspecific and are usually unrecognised by clinicians. The initial clinical features include low-grade fever and respiratory symptoms such as cough, haemoptysis, chest pain, and dyspnoea. Most patients with SPE present similarly to those with pneumonia. The diagnosis of SPE is, therefore, frequently delayed, which consequently influences prognosis. With the increasing use of indwelling catheters and devices, cardiac SPE is now generally associated with cardiac device implants, intravascular catheters, and an immunocompromised state.[7,8] On examination, the patient showed features of right-sided pleural effusion, which was proven by CXR and aspiration, which showed red in colour. A case report of two patients with septic pulmonary embolism showed bloody pleural effusion.[9]

In this case report, CT chest shows multiple cavitary lesions in both lungs, with nodular lesions, bilateral pleural effusion mainly on the right side, collapsed right lung, and pericardial effusion. In a study of 10 patients with Septic Pulmonary Embolism, the various radiological appearances include 9 (90%) patients showed the classical feeding artery sign, 8 patients showed bilateral involvement (80%), 7 (70%) patients shows nodular cavitatory and noncavitatory lesion, 6 (60%) patients show patchy ground glass opacity (GGO), 2 (20%) shows pleural effusion.[10] Other study as stated by the type of lesions on the chest CT, bilateral opacities were the most common radiographic type of lesions finding and were presented in all the patients (100%), followed by nodular (73.9%), cavitation (57.9%), consolidation (47.4%), non-nodular (26.3%), pleural effusion (26.3%) and Feeding vessel sign (15.8%).[11] While a study including 168 cases of SPE by Ye et al. showed that multiple nodular opacities (66%) were the most commonly observed radiographic sign, followed by cavitary lesions (56%), local infiltrations (36%), pleural effusions (30%), feeding vessel sign (28%), and peripheral wedge-shaped lesions (17%).[12]

The transthoracic echocardiographic image shows a mass (thrombus) attached to the HD catheter, which has reached the right atrium. In a study of 55 patients with a Hickman catheter, an abnormal mass consistent with a thrombus was found in 12.5% of patients by transesophageal echocardiography. In all patients with the mass, the catheter tip was placed in the RA.[13] In adults, infection of a right atrial thrombus is a very rare complication of central venous catheters.[14] Cohen et al. [15] evaluated the superiority of transoesophageal echocardiography to image intracardiac masses in 19 patients with central venous lines.

Transthoracic echocardiography identified a mass in only 5 patients (26%). Gilon et al.[16] studied 55 patients by transoesophageal echocardiography within 1 week and after 6–8 weeks after Hickman catheter implantation. In the baseline study, 13 had the tip placed in the right atrium, eight at the superior vena cava-atrium junction, and 27 in the superior vena cava. An abnormal mass, consistent with a thrombus, was found in 12.5% of patients, all in the group with the Hickman catheter tip in the right atrium.

In this case, the blood culture showed no growth of pathogenic bacteria, possibly because the patient was on antibiotics before the test. Catheter tip culture is Staphylococcus aureus with >16 CFUs. The microorganisms most commonly associated with septic emboli include Staphylococcus aureus, coagulase-negative staphylococci, the streptococcal and enterococcus groups, and polymicrobial infections. Less frequently, gram-negative organisms are implicated. In addition, fungal pathogens, most notably Candida and Aspergillus species, have been reported.[17] In a study by Mohamed et al.,[10] Staphylococcus aureus was the most frequently identified pathogen in cultures (52.6% of cases) among patients with septic pulmonary embolism.

Once catheter-related bloodstream infections (CRBSI) are suspected, prompt empiric therapy with an antibiotic that covers methicillin-resistant Staphylococcus aureus (preferably vancomycin) and gram-negative bacilli (e.g., a third-generation cephalosporin, carbapenem, or beta-lactam/beta-lactamase inhibitor combination) should be started.[18,19] The infected catheter should always be removed for patients with infection due to Staphylococcus aureus, Pseudomonas species, or Candida species, and a temporary (nontunneled) catheter should be inserted into another anatomical site. The catheter should also be removed in the presence of severe sepsis, suppurative thrombophlebitis, or metastatic infection (e.g., endocarditis or osteomyelitis), or in the absence of response to antibiotic therapy within 72 hours.[18] The treatment for our patient was vancomycin and meropenem with removal of the catheter, with a good response.

CONCLUSION

In conclusion, septic pulmonary embolism can be due to an infected intravenous catheter in a patient with chronic kidney disease on haemodialysis. Clinical features, ECHO, and chest CT findings are important for diagnosing this condition; removal of the catheter and appropriate antibiotics are essential for treatment.

REFERENCES

1. Bach AG, Restrepo CS, Abbas J, Villanueva A, Lorenzo Dus MJ, Schöpf R, et al. Imaging of nonthrombotic pulmonary embolism: biological materials, nonbiological materials, and foreign bodies. *Eur J Radiol*. 2013;82(3):e120–41.
2. Wong KS, Lin TY, Huang YC, Hsia SH, Yang PH, Chu SM. Clinical and radiographic spectrum of septic pulmonary embolism. *Arch Dis Child* 2002;87:3125.
3. Delgado V, Ajmone Marsan N, de Waha S, et al.: 2023 ESC Guidelines for the management of endocarditis. *Eur Heart J*. 2023; 44:3948-4042. 10.1093/eurheartj/ehad193
4. Stawicki SP, Firstenberg MS, Lyaker MR, et al. Septic embolism in the intensive care unit. *Int J Crit Illn Inj Sci*. 2013;3(1):58. doi:10.4103/2229-5151.109423
5. Baidya A, Ganakumar V, Jadon R, Ranjan P, Manchanda S, Sood R. Septic Pulmonary Emboli as a Complication of Peripheral Venous Cannula Insertion. *Drug Discov Ther*. 2018;12(2):111-3. doi:10.5582/ddt. 2018.01016
6. Gamaza S, Camacho SJ, León J, Agarrado A, Daroca T, Oneto MJ, et al. Acute coronary syndrome due to septic embolism secondary to infective endocarditis in a prosthetic mitral valve. *Rev Esp Cardiol* 2017;70:501–514.
7. Cook RJ, Ashton RW, Aughenbaugh GL, Ryu JH. Septic pulmonary embolism: presenting features and clinical course of 14 patients. *Chest* 2005;128:162–166.
8. Aminzadeh Z, Behzad HR. Bloody Pleural Effusion In Septic Pulmonary Emboli: A presentation of Right-Eided Endocarditis: A Report of Two Cases. *Jundishapur J Microbiol*. 2012;5(3):516-8.
9. Tapas Sh, Hari P, Purvi D. A Study of Radiological Appearances of Septic Pulmonary Embolism in Contrast-Enhanced Computed Tomography Thorax. *International Journal of Contemporary Medical Research*. 2020; 7(2):37. DOI: http://dx.doi.org/10.21276/ijcmr.
10. Mohamed F, Mahad S. Presenting Clinicoradiological Features, Microbiological Spectrum and Outcomes Among Patients with Septic Pulmonary Embolism: A Three-Year Retrospective Observational Study. *International Journal of General Medicine* 2022:15 5223–5235. https://www.dovepress.com/terms.php
11. Ye R, Zhao L, Wang C, Wu X, Yan H. Clinical characteristics of septic pulmonary embolism in adults: A systematic review. *Respir Med*. 2014;108:1–8. doi: 10.1016/j.rmed.2013.10.012. doi: 10.1016/j.rmed.2013.10.012.

12. Gilon D, Schechter D, Rein AJ, Gimmon Z, Or R, Rozenman Y, et al. Right atrial thrombi are related to indwelling central venous catheter position: insights into time course and possible mechanism of formation. *Am Heart J* 1998;135:457–62.
13. Korzets A, Katz S, Chagnac A et al. An infected right atrial thrombus–a new complication of haemodialysis associated subclavian vein catheterisation. *Nephrol Dial Transplant* 1994; 9: 1652–1654.
14. Sakuma M, Sugimura K, Nakamura M, Takahashi T, Kitamukai O, Yazu T, et al. Unusual pulmonary embolism: septic pulmonary embolism and amniotic fluid embolism. *Circ J* 2007; 71:772-5.
15. Cohen GI, Klein AL, Chan KL, Stewart WJ, Salcedo EE. Transesophageal echocardiographic diagnosis of right-sided cardiac masses in patients with central lines. *Am J Cardiol* 1992; 70: 925–929.
16. Gilon D, Schechter D, Rein AJ et al. A. Right atrial thrombi are related to indwelling central venous catheter position: insights into time course and possible mechanism of formation. *Am Heart J* 1998; 135: 457–462.
17. Chambers HF, Bayer AS. Native-Valve Infective Endocarditis. *N Engl J Med*. 2020 Aug 06;383(6):567-576.
18. Mermel LA, Allon M, Bouza E, Craven DE, Flynn P, O'Grady NP, Raad, II, et al. Clinical practice guidelines for the diagnosis and management of intravascular catheter-related infection: 2009 Update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2009;49(1):1-45.
19. Allon M. Dialysis catheter-related bacteremia: treatment and prophylaxis. *Am J Kidney Dis*. 2004;44(5):779-791.

Abbreviations list: Arteriovenous (AV), Arterio-venous graft (AVG), Catheter-related bloodstream infections (CRBSI), Chest X Ray (CXR), Colony Forming Unit (CFU), Computer tomography (CT), Ground glass opacity (GGO), Haemodialysis (HD), Intravenous (IV), Jugular Venous Pressure (JVP), Septic pulmonary embolism (SPE).

Conflict of interest: Authors have nothing to declare.

Funding: Nothing apart from personal fund.