American Journal of Medical Case Reports, 2021, Vol. 9, No. 1, 35-39
Available online at http://pubs.sciepub.com/ajmcr/9/1/10
Published by Science and Education Publishing
DOI:10.12691/ajmcr-9-1-10

Implantable Port Developing Septic Pulmonary Emboli and Secondary Spontaneous Pneumothorax

Rohan Madhu Prasad[1,*], Fazal Raziq[1], Tyler Kemnic[1], Ahmed Abubaker[2]

[1]Department of Internal Medicine, Michigan State University - Sparrow Hospital, Lansing, Michigan, United States
[2]Department of Infectious Disease, Michigan State University - Sparrow Hospital, Lansing, Michigan, United State
*Corresponding author: rohanmaprasad@gmail.com

Received September 25, 2020; Revised October 27, 2020; Accepted November 04, 2020

Abstract This case report illustrates the rare occurrence of an implantable port becoming infected, forming septic pulmonary emboli (SPE), and eventually a secondary spontaneous pneumothorax (SSP). A 43-year-old male presented to the emergency department for a five-day duration of fevers, generalized malaise, difficulty in breathing, non-productive cough, and left chest pain. Past history revealed right carotid body paraganglioma that required resection, adjuvant chemotherapy via a port in the left subclavian vein, and radiation. The cancer was in remission for one year prior to this admission and the port had not been used in six months, but had not been removed. Chest computed tomography demonstrated bilateral pleural cavitations and parenchymal ground-glass opacities. Blood cultures and subsequent sensitivities grew methicillin sensitive *Staphylococcus aureus* (MSSA). We initiated empiric broad spectrum coverage and later switched to cefazolin. A left shoulder ultrasound illustrated a subclavian vein thrombus, so the port was removed. Culture of the catheter tip also grew MSSA. Four days later the patient developed acute dyspnea. Repeat imaging showed a new right-sided spontaneous hydropneumothorax with loculated pleural effusions along with progression of the bilateral opacities and cavitations. Therefore, chest tubes were placed with pleural fluid cultures growing MSSA. Additionally, video-assisted thoracoscopic surgery with decortication was performed. The patient was discharged home on six weeks of intravenous cefazolin via a peripherally inserted central catheter (PICC). This case demonstrates that the physicians should be aware of the lethal complications of a port and should attempt to remove them once they are no longer required.

Keywords: *case report, implantable port, central venous access, methicillin sensitive staphylococcus aureus, septic pulmonary emboli, secondary spontaneous pneumothorax*

Cite This Article: Rohan Madhu Prasad, Fazal Raziq, Tyler Kemnic, and Ahmed Abubaker, "Implantable Port Developing Septic Pulmonary Emboli and Secondary Spontaneous Pneumothorax." *American Journal of Medical Case Reports*, vol. 9, no. 1 (2021): 35-39. doi: 10.12691/ajmcr-9-1-10.

1. Introduction

Cavitary lung lesions have an innumerable amount of etiologies, but they are broadly classified as microbial infections, systemic diseases, and malignancies [1]. Septic pulmonary emboli (SPE), an important cause of cavitations, is strongly linked to patients with infective endocarditis, intravenous drug abuse, oropharyngeal infection, alcoholics, and septic thrombophlebitis like Lemierre's syndrome. However, SPE is now being seen in those with immunosuppression, vascular catheters, and prosthetics [1,2,3].

A catheter can develop complications like infections, thrombosis, and catheter breakage, but they are rarely seen in implantable ports with an incidence of 0.53-1.4% [2,4,5]. Upon our literature review, only two case reports have associated an infected port with the development of a SPE [2,6]. Among SPE patients, the prevalence of secondary spontaneous pneumothorax (SSP) was 3.4% and the in-hospital mortality was 28.2% [7]. We believe that our patient developed an infected port with methicillin-sensitive *Staphylococcus aureus* (MSSA), SPE, and SSP due to the fact that the port was left in for over two years and was not used in over six months. Thus, nonessential ports should be removed as soon as possible to decrease the potential complications. This case report was prepared following the CARE guidelines [8].

2. Case Presentation

A 43-year-old male presented to the emergency department for a five-day duration of fevers, difficulty in breathing, non-productive cough, left chest pain, and generalized malaise. The patient had a history of right carotid body paraganglioma. When it was diagnosed, two years before the current admission, it was six centimeters in size and had metastasized to a right neck lymph node. At that time, the patient underwent resection, adjuvant chemotherapy, and radiation. The patient had completed four chemotherapy cycles of carboplatin and etoposide through an implantable port in the left subclavian vein,

with the last one completed 18 months prior to this presentation. The cancer had been in remission for the previous year. Moreover, the port was last flushed six months prior and had not been used since. The patient admitted that he failed to follow up to remove the port. Social history is pertinent for being a 10-pack year smoker.

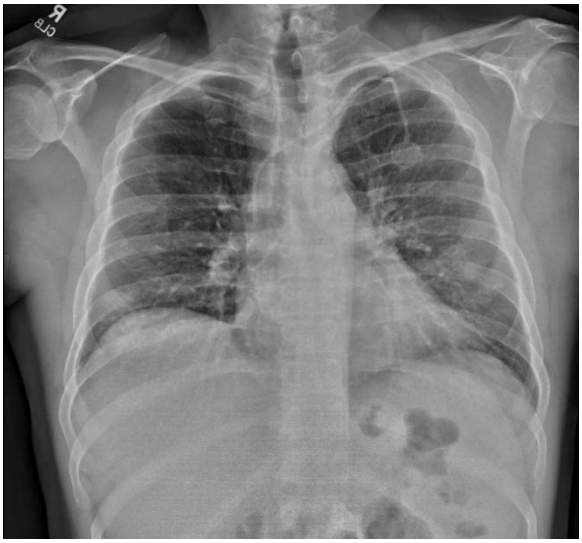

Figure 1. Admission chest x-ray. Multifocal bilateral consolidations

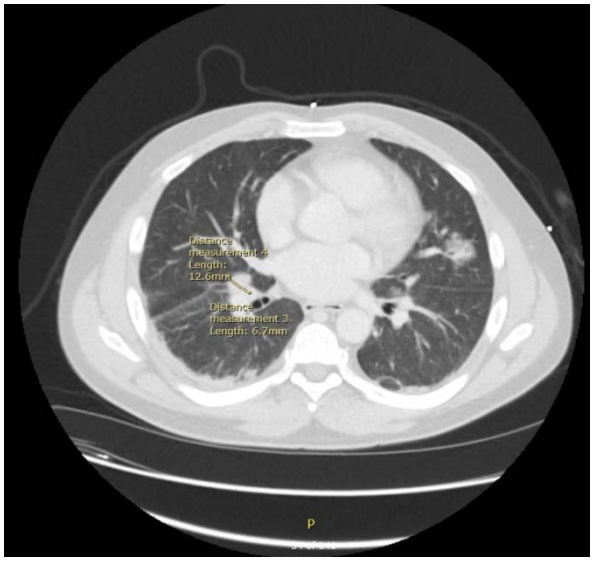

Figure 2. Admission computed tomography of chest, axial view at level of T6-T7. 1) Bilateral pleural cavitations, where right is bigger than left, and mild pleural effusion. 2) Bilateral parenchymal ground-glass opacities and nodules. 3) Enlarged mediastinal lymph nodes where one had central necrosis, measuring 12.6 x 6.7mm

On admission, he was afebrile and required a nasal cannula at two liters to maintain oxygen saturation. The physical exam revealed bilateral coarse breath sounds, hoarse voice, and tenderness over the left upper chest at the port site. We ordered a chest x-ray (CXR) that showed multifocal bilateral consolidations (Figure 1). Initial chest computed tomography (CT) demonstrated bilateral pleural cavitations, parenchymal ground-glass opacities and nodules, and enlarged mediastinal lymph nodes (Figure 2). The patient was initially started on empiric broad-spectrum coverage. Blood cultures and subsequent sensitivities grew MSSA that was sensitive to cefazolin, so the antibiotic regimen was switched appropriately. A comprehensive infectious workup was negative for bacterial, fungal, or mycobacterial etiology (Table 1). A repeat CXR demonstrated a new right lung base cavitation and worsened bilateral consolidations (Figure 3). The cavitary lesion was thought to be secondary to SPE. Transthoracic echocardiogram did not reveal any valvular vegetations or abnormalities, which ruled out infective endocarditis. A left shoulder ultrasound illustrated a subclavian vein thrombus, so the port was removed. The culture of the port tip also grew MSSA.

Table 1. Laboratory and Microbiology Investigations

Laboratory investigation (Reference Range)	Value
White blood cells (10^3/uL)	10.5
C reactive protein (mg/dL)	41.4
Sedimentation rate (mm/h)	50
D dimer (FEU/L)	4.74
Troponin I (pg/mL)	45
Ferritin (ng/mL)	535
Lactic (mmol/L)	1.5
Respiratory culture	Contaminated with oropharyngeal flora
Blood culture	MSSA x2
Left shoulder ultrasound	Subclavian vein thrombus near port
Port tip culture	MSSA
Pleural Fluid analysis	Exudative effusion and MSSA
Atypical pneumonia panel	Negative
Respiratory viral panel	Negative
Acid fast bacilli smear	Negative
Fungal precipitin panel	Negative
Transthoracic echocardiogram	No valvular vegetations. Ejection fraction 60-65%. Mild left ventricular hypertrophy.
Transesophageal echocardiogram	No valvular vegetations. Mild mitral and tricupsid regurgitation. Normal left ventricular systolic function.

Abbreviations: MSSA (Methicillin Sensitive *Staphylococcus aureus*).

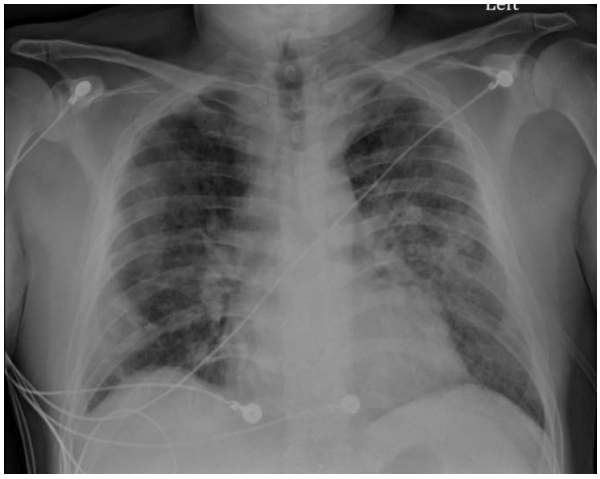

Figure 3. Repeat chest x-ray on day 2 of hospital course. 1) New cavitary lesion in right lung base. 2) Worsening of bilateral consolidations

Four days into the hospital course the patient developed acute worsening of the dyspnea with persistent coughing and fevers. At this time, the physical exam showed bilateral rhonchi, but the patient was still saturating well on a nasal cannula at two liters. Another repeat CXR revealed a new right-sided pneumothorax and a continual worsening of bilateral consolidations (Figure 4). Additionally,

a repeat chest CT illustrated a new right-sided spontaneous hydropneumothorax with a loculated pleural effusion, extension of the previous left-sided lung cavitation, and worsened bilateral ground-glass opacities and consolidations (Figure 5). For the pleural effusion, the patient received two right-sided chest tubes and one left-sided. Pleural fluid analysis and culture revealed an exudative effusion with MSSA. All chest tubes were removed after two days. Video-assisted thoracoscopic surgery with decortication was performed for the loculated pleural effusions. After 48 hours of negative blood cultures, a PICC (peripherally inserted central catheter) was inserted.

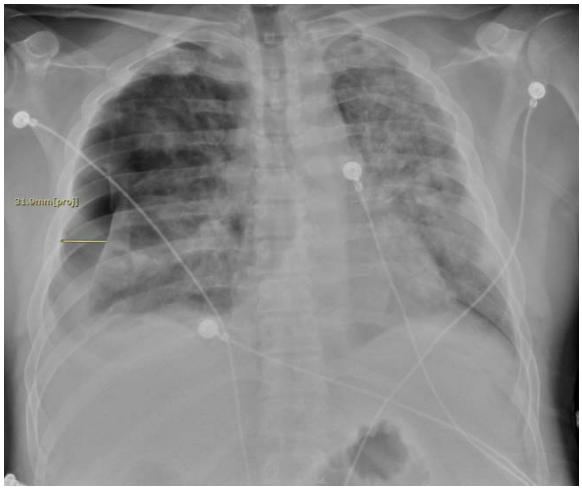

Figure 4. Repeat chest x-ray on day 4 of hospital course 1) New right-sided pneumothorax that is moderate sized and predominantly laterally. 2) Interval worsening of bilateral consolidations

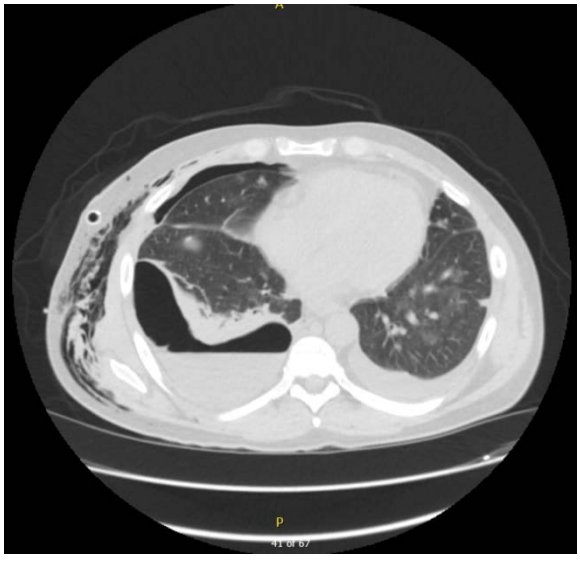

Figure 5. Repeat computed tomography of the chest, axial view at the level of T6-T7 1) Hydropneumothorax is right-sided and predominantly lateral. Loculated right-sided pleural effusion present that measures 4.4 x 6.1 cm. 2) Extension of previous left-sided lung cavitation that now measures 3.5 x 3.2 x 2.8 cm. 3) Bilateral ground-glass opacifications that are now diffuse. Bilateral patchy nodular consolidations.

Upon symptom resolution, the patient was weaned off supplemental oxygen and discharged home on a total of six weeks of intravenous cefazolin via his PICC. He was also instructed and agreed to follow-up with his primary care physician, pulmonologist, and infectious disease specialist. At the end of his antibiotic course, he felt much better and the PICC was removed. However, the patient did not follow-up with the pulmonologist for a repeat chest CT scan.

3. Discussion

For patients requiring long-term central venous access, either a central venous catheter or an implantable port can be used. The possible indications for long-term access include cancer chemotherapy, long-term antibiotic treatment, parenteral nutrition, hemodialysis, repeated blood transfusions, and repeated venotomies [9]. A port is tunneled underneath the skin and placed subcutaneously with the catheter tip inside a chest vein, which is typically either the subclavian or internal jugular vein. Typical catheter complications include infection, thrombosis, and catheter breakage. Ports are known to have the least amount of catheter-related infections. The incidence of port pocket infection was reported at 0.53-1.4% and port-related bacteremia was 0.8-1.4% [2,4].

The most common etiology of a long-term catheter becoming infected is direct contamination of the catheter [4,10]. In regards to catheter-related infections with positive blood cultures, coagulase-negative staphylococci were implicated at 60% with *Staphylococcus epidermidis* comprising 25.7%. Staphylococcus aureus, both MSSA and methicillin-resistant *Staphylococcus aureus*, was only seen in 10% of patients [11]. This may be caused by the staphylococci species producing an extracellular polymeric substance that can form a microbial biofilm layer on the catheter. This biofilm effectively encases the microbial organisms from the host immune system. *Staphylococcus aureus*, specifically, can create a prothrombotic state by expressing clumping factors (ClfA and ClfB) that can bind to adhesin, fibrinogen, and fibronectin. Additionally, *Staphylococcus aureus* binds to and activates platelets and coagulation proteins leading to a thrombus formation [2,4,9,12,13]. The incidence of symptomatic port-related thrombosis varies between the studies, with previous reports stating 28% and more recent ones indicating 5%, 2.9%, 12.8%, and 5.5% [5]. This thrombus can occlude the catheter tip or send emboli containing fibrin, microbial organisms, and purulent material into the lungs and cause a SPE [13]. SPEs have been uncommonly linked with central venous catheters and hemodialysis access and rarely with ports [2,3,6,14,15,16,17]. Imaging findings of SPE on chest CT include peripheral nodules (89%), non-nodular infiltrates (7%), wedge-shaped peripheral lesions (3.2%), peripheral cavitations (10.4%), and feeding vessel sign (6%) [15].

The etiology for the majority of SPE is strongly linked to patients with infective endocarditis, intravenous drug abuse, oropharyngeal infection, alcoholics, and septic thrombophlebitis like Lemierre's syndrome. Thus, it is pertinent to obtain a broad infectious workup along with an echocardiogram. However, SPE is now also being seen in those with immunosuppression, vascular catheters, and prosthetics [1,2,3]. It is rare for an infected port to produce SPE. Furthermore, a SPE-induced cavitation can rupture, effectively producing a SSP. The prevalence of this was 3.4% and the in-hospital mortality was 28.2% [7]. However, these figures include endocarditis-related SPE.

There have only been two reported cases of an SSP developing from a port-related SPE [2,3,4,5,6]. Our case adds to the limited literature where a SPE from a port caused multiple bilateral cavitations that spontaneously ruptured causing a SSP. Since the mortality associated with this condition is significantly high, physicians should be mindful of this port complication.

To establish that the infection originated from the catheter one should obtain a culture of the catheter. This is usually done via the roll plate method where the catheter tip is rolled on a culture plate, but this requires removal of the catheter. An option allowing the catheter to be left in-situ is the semiquantitative cleri technique, which is when one flushes the lumen with a nutrient broth and then cultures the broth [9]. Moreover, on ultrasound a catheter-related thrombus can be seen floating within the vessel lumen around the catheter site or as a ghost after catheter removal. The gold standard is venography, but it is invasive and requires exposure to intravenous contrast and radiation [12,13]. To demonstrate SPE-induced SSP, we hypothesized that pleural fluid should be obtained from the pleural effusions, which should grow the similar organism as the cultures of the blood and catheter. Our diagnostic investigations revealed MSSA on blood, catheter, and pleural fluid cultures and a thrombus near the port.

The current evidence-based treatment method regarding antibiotics for catheter-related MSSA bacteremia is intravenous penicillinase-resistant penicillin, such as nafcillin, oxacillin, cefazolin, or cefuroxime. The patients with uncomplicated infected ports require catheter removal and antibiotics for 7-10 days. If septic thrombosis or endocarditis is present; however, a 4-6 week regimen is necessary [10]. The antibiotic lock therapy is an interesting option to treat a catheter hub related infection that allows one to leave the catheter in-situ. Although, in cases of *Staphylococcus aureus* and candidemia the evidence shows that adequate treatment response requires catheter removal [9,10]. Before a thrombus develops, flushing the catheter with heparin or the usage of oral anticoagulants or low molecular weight heparin are associated with decreased catheter-related infection rates [9]. This further depicts the close relationship between the pathogenesis of a thrombus and an infection. The mainstay of treatment for spontaneous pneumothorax is generally chest tubes and possibly surgery for severe cases [18].

Although rare, an implantable port developing an infection, a SPE, and finally a SSP has drastic complications and mortality rates. Therefore, physicians should be cautious of this and attempt to remove the port once they are no longer required. We hypothesize that if our patient had followed up to remove his port, he would not have developed this sequela. As this is a case report, the limitations are that certain correlations may have been attributed as causations, they cannot be generalized, and they are not based on systematic studies. The results from a single patient may not be applicable to a broad population.

4. Conclusion

Central venous catheter-related infections are uncommon, but are well documented in the literature, except for implantable ports. Additionally, it is rare for these infected ports to form SPE. The emboli commonly causes cavitations; however, only a small percentage rupture to cause a SSP, which is life-threatening and fatal. Our patient developed each of these scenarios and eventually required long-term antibiotics, numerous chest tubes, and surgery. Therefore, we advise that in addition to being cautious of this complication, physicians should attempt to remove the port when possible.

Acknowledgements

The authors have no financial or proprietary interest in the subject matter of this article.

References

[1] Parkar AP, Kandiah P. Differential diagnosis of cavitary lung lesions. *J Belg Soc Radiol.* 2016; 100(1): 100.

[2] Hong G, Kim YS. Recurrent septic pulmonary embolism related to an implanted central venous access port device. *Chinese Medical Journal.* 2018; 131(24): 3009-3011.

[3] Okabe M, Kasai K, Yokoo T. Pneumothorax secondary to septic pulmonary emboli in a long-term hemodialysis patient with psoas abscess. *Nihon Naika Gakkai.* 2017; 56: 3243-3247.

[4] O'Grady NP, Alexander M, Burns LA, et al. Guidelines for the prevention of intravascular catheter-related infections. *Clinical Infectious Diseases.* 2011; 52: e162-e193.

[5] Didi M, Mejri N, Labidi S, et al. Implantable port thrombosis in cancer patients: a monocentric experience. *Cancer Biol Med.* 2016; 13(3): 384-388.

[6] Okada H, Taira K, Tokunaga S, et al. A case of a septic pulmonary embolism related implanted central venous port. *Gan To Kagaku Ryoho.* 2013; 40(3): 389-392.

[7] Gibson CD, Shah P, Jean RA, Jean RE. Prevalence and predictors of pneumothorax in patients with septic pulmonary embolism *Am J Respir Crit Care Med.* 2017; 195: A3948.

[8] Riley DS, Barber MS, Kienle GS, et al. CARE 2013 Explanation and elaborations: reporting guidelines for case reports *JClinEpi*. 2017; 89: 218-235.

[9] Galloway S, Bodenham A. Long-term central venous access. *British Journal of Anesthesia.* 2004; 92(5): 722-734.

[10] Mermel LA, Farr BM, Sherertz RJ, et al. Guidelines for the management of intravascular catheter-related infections. *Clinical Infectious Diseases.* 2001; 32: 1249-1272.

[11] Santarpia L, Buonomo A, Pagano MC, et al. Central venous catheter related bloodstream infections in adult patients on home parenteral nutrition: Prevalence, predictive factors, therapeutic outcome. *Clin Nutr ESPEN.* 2016; 35: 1394-1398.

[12] Crowley AL, Peterson GE, Benjamin Jr DK, et al. Venous thrombosis in patients with short- and long-term central venous catheter-associated Staphylococcus aureus bacteremia. *Critical Care Medicine.* 2008; 36(2): 385-390.

[13] Baskin JL, Pui CH, Reiss U, et al. Management of occlusion and thrombosis associated with long term indwelling central venous catheters. *Lancet.* 2009; 374(9648): 159.

[14] Cook RJ, Aston RW, Aughenbaugh GL, Ryu JH. Septic pulmonary embolism: Presenting features and clinical course of 14 Patients. *CHEST.* 2005; 128(1): 162-166.

[15] Lee SJ, Cha SI, Kim CH, et al. Septic pulmonary embolism in Korea: Microbiology, clinicoradiologic features, and treatment outcome. *Journal of Infection.* 2007; 54: 2230-234.

[16] Inamoto S, Hashimoto Y. A Case of pneumothorax secondary to septic pulmonary embolism due to central venous catheter infection caused by methicillin-resistant Staphylococcus aureus. *Nihon Kansensho Gakkai.* 2008; 82(1): 51-54.

[17] Yang SF, Yang WC, Lin CC. Infective endocarditis-related bilateral spontaneous pneumothorax in a hemodialysis patient. *Acta Clinica Belgica.* 2011; 61(1): 51.

[18] Schnell J, Koryllos A, Lopez-Pastorini A, Lefering R, Stoelben E. Spontaneous pneumothorax - epidemiology and treatment in Germany between 2011 and 2015. *Deutsches Arzteblatt International.* 2017; 114: 739-744.