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SEPTIC PULMONARY EMBOLISM IN A CASE OF PSORIASIS IN AN ADOLESCENT HOST

Respiratory Medicine

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ABSTRACT

Septic pulmonary embolism is an uncommon disease in which infected thrombi are mobilized from an infectious focus and transported hematogenously to the lungs causing vascular obstruction and ischemia along with local inflammation or abscess formation. It is commonly found in cases of tricuspid valve vegetation, septic thrombophlebitis or infected venous catheters. We report a female in adolescent age group during "COVID Era" who presented with fever, cough with expectoration, radiating chest pain and also dyspnea. HRCT revealed multiple randomly distributed cavitary nodules seen predominantly in both upper lobes and superior segment of both lower lobes. There were few wedge shaped areas of mixed consolidation and central ground glass opacities in lateral segment of right middle lobe, lingula and posterobasal segment of both the lower lobes suggestive of pulmonary septic emboli with peripheral septic infarct. She also presented with multiple well-defined hyperkeratotic scaly papules to plaques which were skin colored to slightly erythematous in color over bilateral foot since 5 years. All of the clinical presentations improved after antibiotics treatment and treatment of the primary psoriatic lesion.

KEYWORDS

INTRODUCTION

Septic pulmonary embolism is a rare clinical presentation and a difficult diagnosis. We present an unusual case of septic pulmonary embolism arising from an unknown source where the recurrent pulmonary embolism resolved after successful treatment of the "not so obvious" infectious focus.

Case Presentation

A 13 year old female patient presented with a history of high grade fever with chills for 8 days, cough with expectoration, retrosternal and left mammary chest pain and dyspnea on exertion with no history of diabetes, hypertension, asthma, or contact with Pulmonary Tuberculosis or COVID patient. RT-PCR test for COVID was negative.

The patient was then shifted to SARI (Severe Acute Respiratory Illness) ward from presumptive ward for further investigations and management. The patient appeared initially febrile, temperature was 101·2 F, had toxic look, showed pallor and no signs of icterus. Blood pressure was 90/70 mm of Hg, heart rate was 140/minute and respiratory rate was 28/minute. Oxygen saturation was 96% when supplied with 4 liters of oxygen with nasal prongs. Auscultation of lungs revealed decreased breathe sounds on the left side in infra scapular region with coarse crepitations present over both sides. On enquiry about skin infections she gave a history of scaly papules to plaque like skin lesions since 6 years of age over bilateral foot. She had recurrent exacerbations of the skin psoriatic lesions in winter and the current exacerbation was since 2 months for which the patient was getting homeopathic treatment. Thorough physical examination was done with consent, to exclude other sites of infection. Dermatology consultation reported adjacent raw area over right foot was excoriated by the patient.

Investigations

Chest radiography showed bilateral middle and lower zone nodular shadows, and blunting of left costophrenic angle (Fig.1). Laboratory investigations showed a mild anemia hemoglobin level was 8·8 g/dl and septicemia, marked increased in white cell count of 32·0 x 10^3/cmm, platelet count was 565 x 10^3/cmm. Sputum AFB and CBNAAT report for evaluation of Pulmonary Tuberculosis was negative. Sputum gram's stain suggested presence of gram positive cocci in clusters suggesting a Staphylococcal infection; however the culture report was negative and antimicrobial sensitivity report could not be done. The C - reactive protein noted was 91·78 mg/l. Random blood sugar level was 138 mg/dl initially which then transiently raised up to 186 mg/dl after few days but HbA1c was within normal range (5·5 mmol/mol). ECG revealed sinus tachycardia and 2-D Echo did not show any abnormality. A lower limb venous Doppler was also done which was normal. Urine culture test, blood culture test, Non specific Immunoglobulins showed rise in their value, Immunoglobulin G (IgG) 22·9 g/L and Immunoglobulin A (IgA) 4·51g/L. Other laboratory data including liver function tests, renal function tests were within normal range. Dengue antigen test, widal test, HRP_2 (Plasmodium falciparum antigen histidine rich protein 2) test were negative.

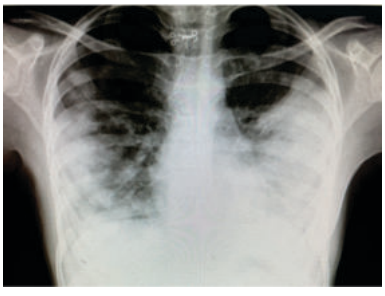


Figure 1- Chest Radiography Showing Bilateral Middle And Lower Zone Consolidation And Blunting Of The Left Costophrenic Angle.

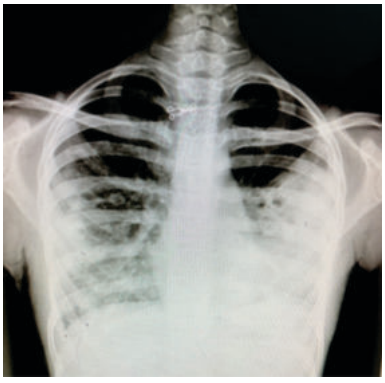
Plain HRCT thorax study revealed multiple randomly distributed consolidatory nodules in both lungs with most of them having central small cavitation seen predominantly in both upper lobes and superior segment of both lower lobes (average size 7 x 7 mm). There were few wedge shaped areas of mixed consolidation and central ground glass opacities in lateral segment of right middle lobe, lingula and posterobasal segment of both the lower lobes and mild left sided effusion with underlying sub segmental passive atelectasis. The features were suggestive of pulmonary septic emboli with peripheral septic infarct. 2D Echo report was normal and ultrasonography of abdomen showed borderline hepatomegaly and no abdominal abscess or infectious focus.

Treatment Follow-up And Outcome

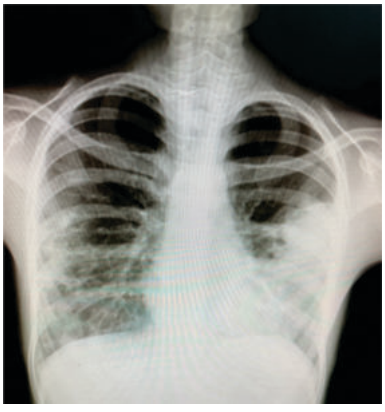
We searched on Google and PubMed for case reports of psoriasis causing septic pulmonary embolism but we could not find a single case report describing psoriasis as a risk factor. However as a damaged epithelial layer due to psoriasis can be vulnerable for spread of staphylococcal skin infection it was presumed to be the source of infection as also the psoriatic lesion was not properly treated for a long time. The patient was empirically started on Inj. Imipenem Cilastatin for initial treatment of bacterial pneumonia on urgent basis but the fever was not resolved within 3 days. Injection Vancomycin was therefore added to cover a possible MRSA infection as fever was not resolved with first line antibiotics and the psoriatic lesion was referred to Dermatology department for aggressive treatment and the patient was followed up with serial CXRs and CT thorax reports.

After 14 days of Vancomycin and Imipenem Cilastatin treatment along

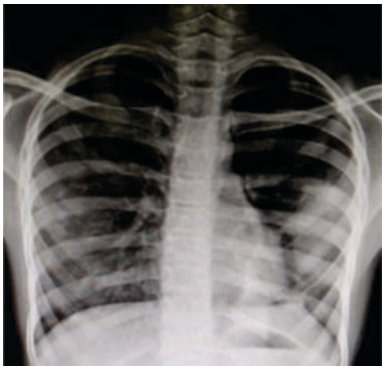
with Calcipotriol (0·005% w/w) ointment, betadine cleaning and Cotaryl cream (cream of urea with natural moisturiser) the patient showed significant clinical improvement. After discharge the patient was advised for tablet Faropenem for 7 days and then she was advised follow up after 15 days where she was asked to continue tablet Linezolid 300 mg BD and Amoxicillin and Clavulanic acid 625mg thrice a day for first 15 days. During each monthly follow up she underwent chest radiography and blood investigations. Anemia was corrected with oral iron supplements. Serum C-Reactive protein concentration and Leukocyte count returned to the normal range. Follow up Chest X-rays (CXR) (Figures 2 B and 2 C) after 6 weeks of treatment with antibiotics showed persistence of Left mid zone consolidation for which she was advised to do CT thorax for detailed evaluation. Figure 2, A to E shows the serial CXRs done for the patient during a total follow up period of 6 months.



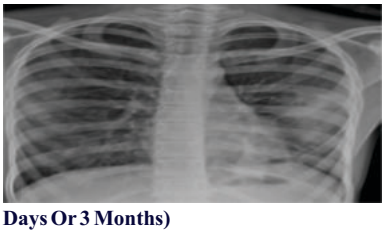
A) (after 9 Days)



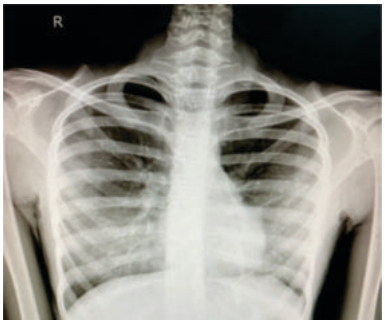
B) (after 20 Days) Persistent Consolidation In Left Mid Zone



C) (after 65 Days Or 2 Months)



D) (after 92 Days Or 3 Months)



E) (after 192 Days Or 6 &1/2 Months) Shows Fibrotic Stranding In Left Mid Zone

Figure 2 A) To E) - Sequential Chest Radiographs During Course Of Treatment

Follow up CT scan of thorax done after 6 weeks revealed multiple small areas of patchy consolidation and parenchymal nodules in both lungs. Two thin walled enhancing fluid filled cavities in left lung measuring 3·9 x 3 x 8 cm in the lingular segment and 2·5 x 2·9 x 4·5 cm in the left lower lobe posterior segment were seen. Minimal basal and cardiophrenic angle effusion was seen. Also small thin walled air containing pneumatoceles were seen in both lungs. As compared to previous scan there was mild resolution in lung lesions with the consolidation on left side showing transformation into lung abscess (Fig. 3). She was again asked to continue Amoxicillin and Clavulanic acid 625mg thrice daily for next 4 weeks because of poor radiological resolution and formation of new abscesses. She was also prescribed T. Linezolid 300 mg BD for 15 days. Meanwhile the patient was also being treated for her psoriasis from the Dermatology department.

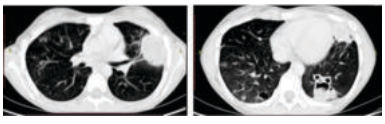


Figure 3-a. **Figure 3-b.**

Figure 3- Ct Thorax After 6 Weeks Showing Multiple Small Areas Of Patchy Consolidation And Parenchymal Nodules (a); Thin Walled Fluid Filled Cavities In Left Lingular Segment And Left Postero Basal Segment Showing Transformation Into Lung Abscess, Pneumatocoeles Were Seen In Both Lungs (b)

Second follow up MDCT scan of thorax done 4 months after the onset of illness showed small areas of consolidation with small internal cavitation in left lingula, multiple peripheral small parenchymal bands / peri-lobular fibrotic bands predominant in right lower lobe of lungs, suggestive of resolving infective changes (bacterial) with no evidence of pleural effusion.

As the patient had persistent necrotizing pneumonia with abscess formation as evidenced radiologically she was continued on T. Amoxycillin and Clavulanic acid for a total duration of 6 months since onset of illness for treatment of Staphylococcal Lung abscess and pneumatocoele until the cavitary lesions had completely healed. Respiratory symptoms were relieved; weight gain of 5 kg was also noted after 6 months of follow up.

The final HRCT (Fig. 4) thorax report done 7 months after the onset of illness showed post infective changes of patchy areas of fibrosis with Bronchiectasis seen in left lingula. A small cystic area of 1·5 x 1·8 cm was seen postero basal segment of right lower lobe, likely a post infective pneumatocoele. There were also few cystic lucent areas seen in left upper lobe and superior segment of left lower lobe suggestive of pneumatocoeles.

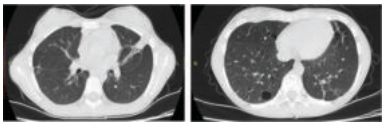


Figure 4-a. **Figure 4-b.**

Figure 4- Patchy Areas Of Fibrosis With Bronchiectasis In Left Lingula (a) And Left Postero Basal Segment With A Pneumatocoele In Posterobasal Segment Of Right Lower Lobe (b).

Also mild centrilobular emphysematous changes were seen in both

lung fields as reported by the radiologist. The skin lesions also improved with treatment from the dermatology department.

Figure 5 shows the skin lesion prior to treatment; Figure 6 reveals the results after treatment.

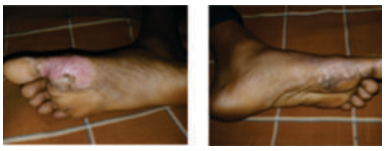


Figure 5- Psoriatic Skin Lesions Prior To Treatment

Figure 6- Improvement Of Skin Lesions After Treatment

DISCUSSION

This is an unusual case of a patient with no other documented past medical history aside from recurrent, poorly controlled plaque psoriasis who subsequently developed septic pulmonary emboli due to Staphylococcal Aureus infection.

Staphylococcal pneumonia is caused by Staphylococcus Aureus, gram-positive cocci in clusters, which usually spread to the lung either from an upper respiratory tract infection or through the blood from other infected sites, most often the skin. They are responsible for a wide variety of diseases ranging from superficial skin and soft tissue infections to deep-seated. S aureus is a ubiquitous colonizer of the skin and mucosal surfaces and can be found in the nares of 10% - 40% of individuals in the community and in the hospital. Primary Pneumonia due to Staphylococcus Aureus is mainly seen in infants[1] while secondary Staphylococcal pneumonia (disseminated infection) is seen more often in children between 3 - 15 years of age.[2] The infection is seen in a bimodal distribution, with the younger and elderly populations being more at risk. However, it should be noted that Pneumococcus is still the leading cause of post-viral pneumonia in this population.[3] Another notable etiology of S. aureus pneumonia is seen in patients who have contamination of the skin or equipment; IV drug abuse can lead to multiple systemic infections from S. aureus such as tricuspid valve endocarditis, bacteraemia, and septic emboli to the lungs.[4] In a case of staphylococcal pneumonia the infection is generally endogenous, the bacteria frequently being derived from the patient's skin or nose.[5] Staphylococcal pneumonia can sometimes get complicated by the formation of lung abscess following bacteraemia or septicemia, particularly in drug addicts with right-sided bacterial endocarditis.[6] The current case would be classified as "complicated" Staphylococcus Aureus Bacteraemia (SAB) because of the presence of septic emboli and metastatic infection, as per the Infectious Disease Society of America (IDSA) guidelines.[7]

Septic pulmonary embolism (SPE) is an uncommon and serious disorder associated with significant morbidity and mortality. It remains a difficult diagnosis in part because of its varied etiology and clinical presentations. The current case draws attention to the importance of psoriatic skin disease as a source of SAB and septic pulmonary emboli. This was a case of radiologically and clinically diagnosed septic pulmonary embolism with no apparent primary source until we discovered active self excoriated psoriatic skin lesions with a 6 year history. However, we could not find any literature describing psoriasis of the skin as the primary source of staphylococcal septic emboli although skin and soft tissue infections (SSTIs) have been described as an increasing cause of SAB.[4] Nevertheless, it is known that in psoriatic skin the epidermis divides much faster than normal causing a defective outer layer or barrier which under normal circumstances would protect from infection.[8] In addition Staphylococcal toxin α-Hemolysin (Hla; α-toxin) is known to play an important role in SSTIs by causing dermonecrosis. By activation of disintegrin and metalloprotease 10, Hla is known to contribute to proteolysis of E-cadherin, which leads to the disruption of the adherens junction in the epithelial layer, thereby prompting potential remodeling of the epithelial layer and consequently pathogen dissemination.[9,10] Similarly, Hla also contributes to the destruction of blood vessel endothelium integrity by causing proteolysis of the extracellular domain of vascular endothelial cadherin.[11] The pathogenesis caused by α-Hemolysin along with the epithelial damage caused by psoriasis itself can work in combination to allow staphylococcal skin infection to breach the epithelial barrier and consequently spread via the hematogenous route. An old study from 1973 has shown that about 50% of patients with psoriasis carried S aureus in low numbers, the cocci being more numerous on the lesions as compared to uninvolved skin.[12] In all likelihood a psoriatic skin lesion can serve as a potential nidus for Staph aureus infection which can then disseminate systemically to other locations in the body.

Interestingly, Psoriasis is known to have an increased risk of venous thromboembolism and Pulmonary embolism particularly in younger patients.[13] In this case we witnessed an adolescent patient with septic pulmonary embolism without deep venous thrombosis due to a skin lesion of psoriasis with staphylococcal infection. Whether a persistent Staphylococcal skin infection in young patients with psoriasis contributes to venous thromboembolism in cases of Psoriasis will be an interesting topic of research. Recent studies have shown that Psoriasis is an immuno-inflammatory disease associated with cardiovascular risk factors, embolic and thrombotic events, as well as hypercoagulability.[13]

A case report was published of a 78-year-old female patient, with psoriasis associated with pulmonary embolism, which was accidentally discovered.[14] This patient also had evidence of mild pulmonary fibrosis with no radiological criteria for diffuse parenchymal lung disease, which could not be associated with psoriasis directly. As this patient was not getting systemic therapy for psoriasis which could cause pulmonary fibrosis the cause of pulmonary fibrosis in this patient could not be established. In the case we present the patient developed pulmonary fibrosis as a result of healed septic pulmonary emboli. Could it be possible that cases of psoriasis have subclinical, asymptomatic, missed septic pulmonary emboli causing necrotic lung infections leading to fibrosis?

In the case we present, the psoriasis over plantar region was not properly treated for long times which lead to a defective epithelial barrier for protection against infection and the spread of infection through hematogenous route leading to the pulmonary septic emboli. Staphylococcus aureus can provoke an intense inflammatory reaction, with direct endothelial damage via cytotoxins (e.g. Panton-Valentine leukocidin) and enzymatic mechanisms (e.g. Coagulase), culminating in septic pulmonary emboli and septic thrombophlebitis in few cases.[15] Although skin and soft tissue infections have been known to be the least common source for potential SPE, recent studies show that the incidence of skin and soft tissue infections (SSTIs) as the primary source of SPE is increasing (upto 44%).[16] It is important to identify the primary source of infection and eliminate it along with the systemic infection to ensure complete cure. Proper diagnosis i.e. plantar psoriasis was made and treated. Treatment of the root cause i.e. plantar psoriasis broke the vicious pathophysiological cycle of the disease. It was not alone the antibiotics that treated the disease but also proper treatment of the causative factor i.e. psoriasis that contributed to the complete relief from the disease process. Also a long duration of antibiotics of about 6 months was required for complete elimination of the infection.

CONCLUSION

In conclusion, the epidemiology of septic pulmonary embolism (SPE) has broadened over time with an increase in identified extra pulmonary and non-cardiac sources related to contiguous infections. By being aware of the various sources of the septic pulmonary emboli we can facilitate the early diagnosis and management of this potentially fatal condition. It is important to eliminate the primary source of infection to ensure complete resolution of the disease process. Psoriatic lesions have never been mentioned in literature as a potential source of SPE and this particular case should arouse interest in further research into Psoriasis as a potential source of recurrent SPE causing pulmonary fibrosis and persistent systemic inflammatory response.

Conflict of Interest

The authors have no personal interest or conflict of interest in presenting this case.

REFERENCES

1. Goel A, Bamford L, Hanslo D, Hussey G. Primary staphylococcal pneumonia in young children: a review of 100 cases. J Trop Pediatr. 1999 Aug;45(4):233-6. doi: 10.1093/tropej/45.4.233. PMID: 10467836.
2. Chartrand SA, McCracken GH Jr. Staphylococcal pneumonia in infants and children. Pediatr Infect Dis. 1982 Jan-Feb;1(1):19-23. doi: 10.1097/00006454-198201000-00006. PMID: 7177890.

3. Cuquemelle E, Soulis F, Villers D, Roche-Campo F, Ara Somohano C, Fartoukh M, Kouatchet A, Mourvillier B, Dellamonica J, Picard W, Schmidt M, Boulain T, Brun-Buisson C; A/H1N1 REVA-SRLF Study Group. Can procalcitonin help identify associated bacterial infection in patients with severe influenza pneumonia? A multicentre study. Intensive Care Med. 2011 May; 37(5):796-800. doi: 10.1007/s00134-011-2189-1. Epub 2011 Mar 3. PMID: 21369807; PMCID: PMC7080069.
4. Tong SY, Davis JS, Eichenberger E, Holland TL, Fowler VG Jr. Staphylococcus aureus infections: epidemiology, pathophysiology, clinical manifestations, and management. Clin Microbiol Rev. 2015 Jul;28(3):603-61. doi: 10.1128/CMR.00134-14. PMID: 26016486; PMCID: PMC4451395.
5. Wertheim HF, Melles DC, Vos MC, van Leeuwen W, van Belkum A, Verbrugh HA, Nouwen JL. The role of nasal carriage in Staphylococcus aureus infections. Lancet Infect Dis. 2005 Dec;5(12):751-62. doi: 10.1016/S1473-3099(05)70295-4. PMID: 16310147.
6. MacMillan JC, Milstein SH, Samson PC. Clinical spectrum of septic pulmonary embolism and infarction. J Thorac Cardiovasc Surg. 1978 May;75(5):670-9. PMID: 642559.
7. Liu C, Bayer A, Cosgrove SE, Daum RS, Fridkin SK, Gorwitz RJ, Kaplan SL, Karchmer AW, Levine DP, Murray BE, J Rybak M, Talan DA, Chambers HF. Clinical practice guidelines by the infectious diseases society of america for the treatment of methicillin-resistant Staphylococcus aureus infections in adults and children: executive summary. Clin Infect Dis. 2011 Feb 1;52(3):285-92. doi: 10.1093/cid/cir034. PMID: 21217178.
8. Roberson ED, Bowcock AM. Psoriasis genetics: breaking the barrier. Trends Genet. 2010 Sep;26(9):415-23. doi: 10.1016/j.tig.2010.06.006. Epub 2010 Aug 6. PMID: 20692714; PMCID: PMC2957827.
9. Wilke G.A., Bubeck Wardenburg J. Role of a disintegrin and metalloprotease 10 in Staphylococcus aureus alpha-hemolysin-mediated cellular injury. Proc Natl Acad Sci U S A. 2010;107:13473–13478.[PMC free article] [PubMed] [Google Scholar]
10. Maretzky T., Reiss K., Ludwig A., Buchholz J., Scholz F., Proksch E., de Strooper B., Hartmann D., Saftig P. ADAM10 mediates E-cadherin shedding and regulates epithelial cell-cell adhesion, migration, and beta-catenin translocation. Proc Natl Acad Sci U S A. 2005;102:9182–9187. [PMC free article] [PubMed] [Google Scholar]
11. Powers M.E., Kim H.K., Wang Y., Bubeck Wardenburg J. ADAM10 mediates vascular injury induced by Staphylococcus aureus alpha-hemolysin. J Infect Dis. 2012;206:352–356. [PMC free article] [PubMed] [Google Scholar]
12. Marples RR, Heaton CL, Kligman AM. Staphylococcus aureus in Psoriasis. Arch Dermatol. 1973;107(4):568–570. doi:10.1001/archderm.1973.01620190044010
13. Ahlehoff O, Gislason GH, Lindhardsen J, Charlot MG, Jørgensen CH, Olesen JB, Bretler DM, Skov L, Torp-Pedersen C, Hansen PR. Psoriasis carries an increased risk of venous thromboembolism: a Danish nationwide cohort study. PLoS One. 2011 Mar 25;6(3):e18125. doi: 10.1371/journal.pone.0018125. PMID: 21464995; PMCID: PMC3064586.
14. Milić Rade AU, Šarac Sanja AU, Lazović-Popović Biljana AU, Dinić Miroslav JN, Psoriasis as a risk factor of pulmonary embolism, Vojnosanitetski pregled 2019 Volume 76, Issue 9, Pages: 968-972.
15. Brenes JA, Goswami U, Williams DN. The association of septic thrombophlebitis with septic pulmonary embolism in adults. Open Respir Med J. 2012;6:14-9. doi: 10.2174/1874306401206010014. Epub 2012 May 8. PMID: 22611460; PMCID: PMC3355351.
16. Goswami U, Brenes JA, Punjabi GV, LeClaire MM, Williams DN. Associations and outcomes of septic pulmonary embolism. Open Respir Med J. 2014 Jul 24;8:28-33. doi: 10.2174/1874306401408010028. PMID: 25184008; PMCID: PMC4150379.