A non-smoker 38-year-old male presented with complain of sudden onset of progressive chest pain and breathlessness of 12-hour duration. Chest pain was sharp and localized to right anterior chest region. Patient denied any history of trauma sustained, fever, hemoptysis, expectoration, orthopnea, sweating, vomiting, abdominal pain and/or epistaxis. His personal, family, occupational and drug history were non-contributory.

On presentation to us, patient appeared in respiratory distress, his pulse was 108 beats/minute, blood pressure 110/80 mmHg and respiratory rate of 26 breaths/minute. His resting SpO\textsubscript{2} was 93% on room air. Clubbing was noted in all the fingers but cyanosis was absent. There was no pedal edema. Skin and mucus membrane examination were also normal. Examination of chest was suggestive of massive right-sided pleural effusion. Cardiovascular, abdominal and central nervous system examination were unremarkable.

Complete blood counts, routine urine examination, renal and hepatic functions including coagulation profile were within normal limits. Hemoglobin was 10.6 gm%. Electrocardiogram showed sinus tachycardia. Skiagram chest showed opaque right hemithorax with shift of mediastinum to left side. Left lung field was apparently normal. Diagnostic thoracocentesis revealed grossly hemorrhagic aspirate with hematocrit of 24% and proteins 5.2 gm%. Differential counts of pleural fluid showed polymorphs 64% and lymphocytes 36%. Pleural fluid cytology was negative for malignant cells.

In view of the presence of clubbing with hemothorax, closed pleural biopsy was performed with Abrams needle with aspiration of remaining pleural fluid. Histopathological examination of pleural biopsy was not conclusive. Follow-up chest X-ray after pleural biopsy showed minimal pleural effusion.

Contrast-enhanced computed tomography (CECT) of thorax was performed to know the state of underlying pleural space and lung parenchyma. This study showed residual small pleural effusion on right side with conglomerate of multiple soft tissue masses in peripheral part of right anterior chest in relation to anterior segment of right lower lobe [Figure 1]. Contrast echocardiography with injection of agitated saline (to create bubble) in peripheral vein was done. Bubbles appeared in left atrium after fourth cardiac cycle suggestive of a right to left shunt.

CECT and echocardiography findings favoured a strong possibility of pulmonary arteriovenous malformation (PAVM). Therefore, multidetector computed tomography (MDCT) thorax angiography was performed to confirm PAVM and to delineate the number and size of the feeders. MDCT angiography showed a lobulated vascular mass of 25.7 mm × 13.8 mm in right anterior lower chest with a single feeder vessel of 5.5-mm diameter. Efferent was seen draining right inferior pulmonary vein [Figures 2 and 3]. In view of a single PAVM with a feeder vessel of 5.5 mm in diameter, patient was referred to thoracic surgeon. Video-assisted thoracoscopic wedge resection of malformation was done with ligation of the feeder. Postoperatively patient had favorable recovery with normalization of his resting SpO\textsubscript{2} and was doing well till the fourth week of follow-up.

Spontaneous hemothorax is a rare entity. The most common cause of this condition is metastatic pleural disease.\textsuperscript{[1]} Other rare causes include anticoagulation therapy, rupture of aortic aneurysm, pleural endometriosis, extramedullary hematopoiesis, hemophilia, thrombocytopenia, spontaneous pneumothorax, acute pancreatitis, and PAVM.\textsuperscript{[2]}

Dyspnea, hemoptysis, cyanosis and clubbing constitute the usual presenting features of PAVM. Occasionally,
Case Letters

Patient may also present with neurological complications for e.g. stroke, transient ischemic attacks, migraine and headache. Hemothorax as a presenting feature of PAVM is a very rare occurrence. Ali et al. [2] found only 32 reported cases of hemothorax associated with PAVM till 2008. Half of these cases had documented HHT. On the basis of negative family history, and absent signs/symptoms like epistaxis, mucocutaneous telangiectasia HHT can be ruled out in the present case adding to its rarity.

Cottin et al. [3] described four patients of hemothorax in a series of 126 patients with PAVM. Ference et al. [4] reviewed the incidence of pulmonary hemorrhage in patients with HHT and PAVM. Six out of 143 patients had hemothorax secondary to rupture of PAVM and in all these patients hemothorax was the presenting illness. Recently, Quinones et al. [5] described a 39-year-old female with HHT and PAVM having hemothorax as a presenting symptom.

Presence of clubbing and hemothorax in our patient led us to initially consider the possibility of malignant pleural effusion and thence to pleural biopsy. However, pleural biopsy turned out to be negative for malignancy.

There was no other clue on clinico-radiological examination that could guide us about the possible cause of hemothorax in this patient. Therefore, we decided to have CT thorax enhanced to delineate the morphology of underlying lung and pleura. This investigation brought us closer to the etiological diagnosis of hemothorax.

Conventional pulmonary angiography and MDCT thoracic angiography were the two options before us to confirm the diagnosis of PAVM in this patient. Pulmonary angiography and MDCT angiography chest both have their own merits and demerits. MDCT thoracic angiography was preferred as it is superior in demonstrating small arteriovenous malformations, non-invasive and relatively inexpensive than conventional pulmonary angiography. Further, MDCT thorax also gives information about other pathologies in thorax, if any. But, vascular lesions may also give false positive result on this modality. [6] Contrast-enhanced MR angiography has also been found to be more sensitive than pulmonary angiography in detecting PAVM. Further, although contrast enhanced MR angiography has the advantage of acquiring MIP reconstructions of complex PAVM in multiple planes but it is inferior in delineating lung parenchymal pathology as compared to MDCT angiography. [7]

Perfusion scan and contrast 2D echo may also detect a right to left shunt but these modalities also give positive results in presence of intracardiac right to left shunts. [8] The two available options to treat this patient were embolization versus surgical excision. Risk of future recurrence and the presence of a single malformation tilted the balance in favor of surgical excision of the lesion. Video-assisted thoracoscopic wedge resection was performed as it is associated with less morbidity and mortality than conventional thoracotomy.

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Nocardia farcinica as a causative agent of lung abscess

Sir,

Nocardia farcinica lung abscess in an immunocompetent adult without any other systemic manifestation of Nocardia is an extremely rare entity. Peculiar pathogens of lung abscess are Staphylococcus aureus, Klebsiella, Pseudomonas aeruginosa and Proteus species. Candida albicans, atypical pathogens like Legionella and Pneumocystis jiroveci may be encountered as well in immunocompromised individuals. Patients with impaired immune mechanisms and cough reflex are most vulnerable for aspiration and abscess formation.

A 45-year-old non-diabetic male, known case of severe persistent bronchial asthma for 10 years (controlled on regular treatment with inhaled beclomethasone 800 µg and formoterol 12 µg per day) presented with high grade continuous fever coupled with cough with phlegm production (not foul smelling) for 10 days and breathlessness for 4 days. Cough had postural variation and was more in right lateral position. On admission temperature was 102°F and oxygen saturation was 92%.

Routine hemogram revealed normal picture except a raised total leukocyte count of 18,000/mm³ with 93% polymorphonuclear neutrophils. Sputum was negative for acid fast bacilli by Ziehl-Neelsen stain and concentration method. Mantoux test revealed induration of 4 mm at 72 hours. Grade 1 clubbing was present. Auscultation revealed decreased entry in left lower lung field with few crackles in upper lung fields of the same side. Chest X-ray revealed a large cavity in left lower lung with irregular margins and air fluid level inside [Figure 1]. Ultrasound-guided aspiration of 100 ml frank pus was done which was examined for culture and sensitivity and the patient was started on empirical injectable antibiotic therapy with amikacin, piperacillin tazobactam and clindamycin. There were no signs of improvement clinically and radiologically even after 1 week of antibiotic therapy and his phlegm production increased in amount and purulency with persistent running fever. Oxygen saturation dropped down to 86% and the patient was now supplemented with oxygen therapy. Meanwhile sputum culture revealed Pseudomonas species sensitive to amikacin and piperacillin tazobactam and subsequent modified Ziehl-Neelsen stain of the Broncho alveolar lavage (BAL) sample revealed typical Nocardia species with delicate beaded branching filaments [Figures 2 and 3]. Sputum was further examined by polymerase chain reaction (PCR) which confirmed the diagnosis of N. farcinica species. Culture on blood agar plates revealed typical chalky white wrinkly colonies of Nocardia after 5 days.

In vitro drug sensitivity revealed resistance of Nocardia to amikacin, piperacillin tazobactam, ceftriaxone and sensitive to cotrimoxazole (20 mg/kg) and linezolid (40 mg/kg).

Based on the poor response to antipseudomonal therapy he was put on injectable cotrimoxazole and linezolid therapy. A dramatic response to combination therapy was evident as the phlegm production and fever spikes reduced substantially within 5 days of injectable therapy. Total leukocyte counts settled down to 8000/mm³ with normal polymorphonuclear neutrophils. Radiological