Case Report

Cavitating Pulmonary Infarct in Absence of any Co-Existing Cardiopulmonary Disease

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Abstract

In a patient who had no co-existing cardiopulmonary disease, pulmonary infarction (PI) and cavitation in the infarct is a rare phenomenon. Here, we are reporting such a rare case of cavitating pulmonary infarct who presented with pneumonia. A 65-year-old woman presented with typical infective symptoms such as high-grade fever, dyspnea, and pleuritic chest pain. Her vitals were within normal limits except hyperthermia. Chest X-ray showed right basal consolidation at admission that showed cavitation on the 5th-day chest radiograph. Computerized tomogram of the thorax showed cavitated pulmonary infarct and bilateral pulmonary thromboembolism. All microbiological investigations were negative. She denied having any previous cardiac problem and her current echocardiogram was also unremarkable. This case represents the transition phase of PI and cavitation that was misdiagnosed as a community-acquired pneumonia due to similar clinical and radiological features. PI can present as cavitating pneumonia, even in the absence of any co-existing cardiopulmonary disease.

Keywords: Cavitation, pulmonary infarct, pneumonia

Introduction

The incidence of pulmonary embolism (PE) in community residents is around 2.3–3.6/10,000 person years. Ten percent of PE develop pulmonary infarction (PI) and 4%–7% of PI complicate in cavitation.[1,2] PI and cavitation in the infarct occurs mostly in elderly patients who already had comorbid conditions, usually congestive heart failure. Both of these conditions are rare in absence of any co-existing cardiopulmonary disease.[3] We are reporting here such a rare case of cavitating PI in an elderly lady who had no co-existing cardiopulmonary disease and presented as community-acquired pneumonia (CAP).

Case Report

A 65-year-old, apparently obese (body mass index [BMI] 29.62 kg/m^2) lady presented with complaints of right pleuritic chest pain, cough with blood-tinged expectoration, exertional dyspnea, and fever for 10 days. Her past medical history was not significant except repeated upper respiratory tract infection for the past 2 months. Family history was also unremarkable. Her vitals were within normal limits except hyperthermia. Her initial blood analysis was as follows: white blood cells: 12,500 cells/μL (90.2% neutrophils and 7.8% lymphocytes); hemoglobin: 11.7 g/dL; platelets: 372,700 cells/μL; and C-reactive protein (CRP): 232 mg/L. Chest radiograph (posteroanterior [PA] view) showed right basal consolidation with pleural effusion [Figure 1].

She was hospitalized as a case of CAP and about 30 ml of bloody fluid was obtained from the right pleural cavity. The fluid was exudative (fluid protein: 3.95 g/dL vs. serum proteins: 6.56 g/dL) and showed neutrophilic leukocytosis (total cell count: 8000 cells/mm^3 with 80% neutrophils). The pleural fluid sugar and adenosine deaminase were 117 mg/dl and 11 U/L, respectively. Microbiological examination of the blood, sputum, and pleural fluid did not show any growth. The Ziehl–Neelsen stain of induced sputum and pleural fluid was also negative.

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With the antibiotic therapy, her fever subsided, dyspnea improved, CRP decreased, and leukocyte count became normal, but she had persisting pleuritic chest pain and hemoptysis. Repeat chest radiograph (PA view) on the 5th day showed cavitation in the consolidation patch [Figure 2].

Contrast computerized tomogram (CT) of the thorax was done. The lung parenchyma showed irregular thick-walled cavity with internal air–fluid level, adjacent areas of consolidation, and pleural effusion. There was an intraluminal thrombus in inferior divisions of both the pulmonary arteries [Figures 3 and 4]. Venous Doppler study of bilateral lower limbs showed deep-vein thrombosis in the left leg extending from the tibial veins to the common iliac vein. The diagnosis was revised to venous thromboembolism (VTE) with cavitating lung infarct.

Detailed clinical history was again taken with special emphasis on risk factors of VTE and any preexisting cardiopulmonary diseases. She denied any trauma, hospitalization, hormone replacement therapy, or prolonged bed rest but admitted that she had left leg edema and calf pain 20 days back that subsided with analgesics. Her previous records on cardiac evaluation and current echocardiogram were unremarkable. Her coagulation profile was also negative. She responded positively to anticoagulants with complete resolution of symptoms in the next 4 days and got discharged. She was asymptomatic till 3-month follow-up while on anticoagulants. The cavity size markedly regressed in follow-up radiographs but did not disappear.

Discussion

The common conditions favoring venous thrombosis among females are pregnancy, estrogen therapy, hormone replacement therapy, and oral contraceptives. Obesity (BMI >29 kg/m^2), recent respiratory infections, and raised inflammatory markers were also found to be risk factors for VTE.[4-6] PE is sometimes difficult to diagnose because of nonspecific clinical presentation. Clinical prediction scores such as Wells score, and revised Geneva score and plasma D-dimer are sensitive but have variable specificity. Classical radiological signs such

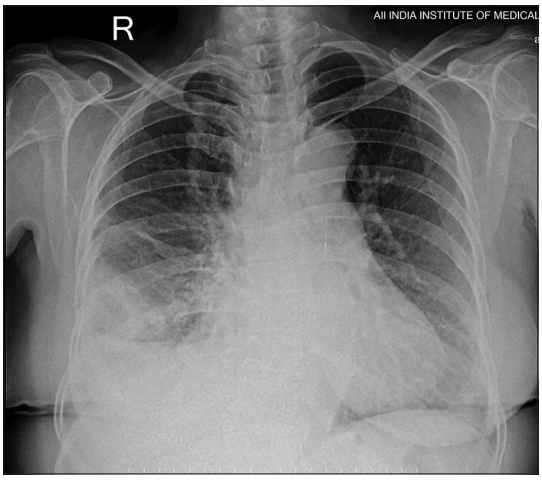

Figure 1: Chest X-ray posteroanterior view (day 1) showing patch of consolidation in the right lower zone

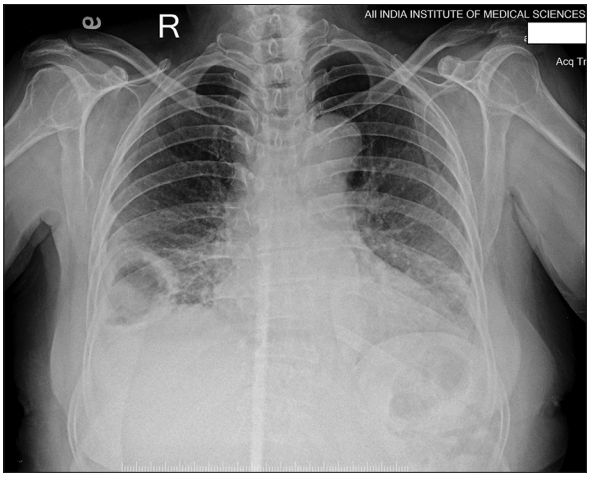

Figure 2: Chest X-ray posteroanterior view (day 5) showing cavitation in the consolidation

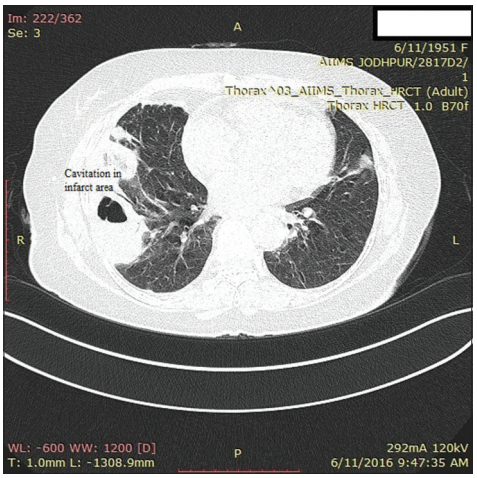

Figure 3: Computerized tomogram film lung window axial view showing cavitation in consolidation

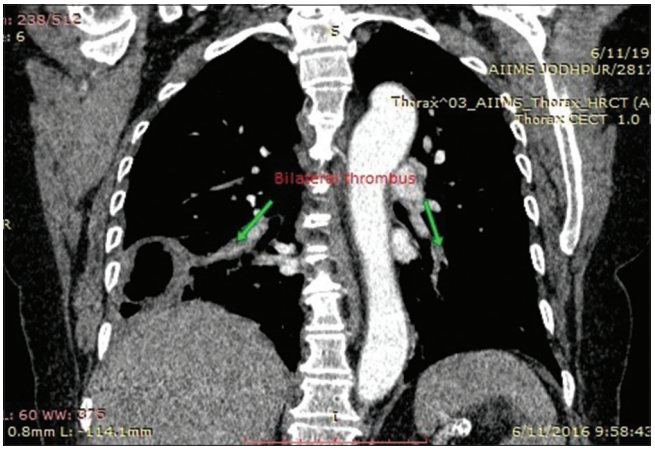

Figure 4: Computerized tomogram contrast film coronal view showing thrombus in inferior divisions of both pulmonary arteries

as Fleischner sign , Hampton hump, and Westermark's sign are found only in a small number of chest radiographs.[7] CT pulmonary angiography is considered as the standard modality for the diagnosis of PE. Failure to enhance the lumen due to filling defects suggests acute PE. Pulmonary infarct usually appears as peripheral wedge-shaped areas of hyperattenuation in lung windows that may have scalloped inner margins and cross-cavity band shadows.[8] The treatment of PE is anticoagulation in hemodynamically stable patients who do not have significant contraindications. Hemodynamically unstable patients may require thrombolysis. Application of inferior vena cava filters is used in recurrent emboli, while pulmonary endarterectomy is a preferred choice for chronic thromboembolic pulmonary hypertension.

Our case represents a transition phase of PI and cavitation that was misdiagnosed as CAP on a clinicoradiological basis. Cavitation occurs only in 4%–7% cases of pulmonary infarcts, mostly in the elderly and in the presence of co-existing cardiopulmonary disease. In our case, we were unable to detect any previous or current coexisting cardiopulmonary disease. Cavitation in the PI may occur either aseptically in large infarcts (more than 4 cm in diameter) or due to some necrotizing infection. Cavitation occurs earlier in infected infarcts (mean: 18 d, range: 6–40 d) as compared to noninfected one (mean: 28 d, range: 7–120 d).[9,10]

The clinical presentation and radiological features of pulmonary infarct may simulate pneumonia, especially when it is infected. The cavity in chest radiograph has broad differentials including common causes such as tuberculosis, necrotizing pneumonia, and lung mass and uncommon causes such as PI. Cavitation in pulmonary infarct occurs predominantly on the right side with a single cavity. Constant cavity size, irregular cavity outline, and no fluid level may suggest an aseptic cavity, while air–fluid level, prominent fever, and purulent sputum indicate superadded infection.[8,9] Although all microbiological investigations were negative in this patient, improvement in symptoms by antibiotic and air–fluid level in the cavity may indicate some superadded infection. In rare conditions, these cavities may complicate as pneumothorax, empyema, and bronchopleural fistula.

Conclusion

The suspicion of PE is not a common consideration in patients who present with classical infective symptoms and unremarkable medical history. PI can present as cavitating pneumonia, even in the absence of any co-existing cardiopulmonary disease. Clinicians must be aware of the uncommon causes of cavitations in chest radiographs.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her names and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest

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