Serendipitous Unearthing of Silent Multiple Giant Rasmussen's Aneurysms by Fluorine-18 Fluorodeoxyglucose Positron Emission Tomography/Computerized Tomography

Abstract

The authors report multiple giant bilateral pseudoaneurysms of pulmonary artery, also known as Rasmussen's aneurysms, which remained silent and unrevealed despite the large size and multiplicity unearthed by fluorine-18 fluorodeoxyglucose positron emission tomography/computerized tomography.

Keywords: *Computerized tomography, fluorine-18 fluorodeoxyglucose positron emission tomography/computerized tomography, pulmonary angiography, Rasmussen's aneurysm*

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Introduction

Rasmussen's aneurysm is an infrequently occurring entity usually associated with pulmonary tuberculosis cavity or focus eroding a small peripheral pulmonary arterial branch with grave consequences often with massive life-threatening hemoptysis.[1] Nonhemoptytic and yet large aneurysms are still rare and are being revealed by fluorine 18-fluorodeoxyglucose positron emission tomography/computerized tomography (F18-FDG PET-CT) during clinical and imageological workup in oncology practice.

Case Report

A 34-year-old young male presented with severe bouts of cough, shortness of breath, and generalized weakness. There was no association of fever, hemoptysis, chest pain, or weight loss. Initial clinical assessment revealed well-preserved apyrexial, normotensive, nonanemic, and a cyanotic individual with no clubbing or pedal edema. Hematological, biochemical, and metabolic parameters were within normal limits. The patient is a known and diagnosed case of chronic rheumatic heart disease since the age of 9 years on regular follow-up by a single cardiologist. He has progressive severe calcific aortic stenosis with moderate aortic regurgitation and mild mitral regurgitation with dilated left ventricle and good left ventricular systolic function [Figure 1]. Plain radiograph chest revealed homogeneous shadows of the nature of consolidation in the right mid zone and left upper zone [Figure 2]. A course of antibiotics was given but symptoms persisted unabated. Repeat radiograph chest after 4 weeks has shown no change in the radiological findings with the review radiograph identical to the preantibiotic skiagram chest [Figure 3]. In view of the nonresolving chest opacity, a high resolution contrast-enhanced CT of the chest was done which showed bulky lobulated mass-like opacities in both upper lobes and right lower lobe showing central dense enhancement and peripheral hypodensity in all the lesions [Figure 4]. CT-guided tru-cut biopsy performed showed lung tissue with marked increase in vascularity with extensive hemorrhage. There were scattered lymphocytes with no granulomas or malignant cells. Acid-fast bacilli and fungal staining were negative. A confirmatory real-time polymerized chain reaction-based *Mycobacterium tuberculosis* complex detection was also negative. Faced with this inconclusive situation, a F18-FDG PET-CT was requested to metabolically characterize the pulmonary lesions and also to identify a possible mitotically incriminating primary organ focus. F18-FDG PET-CT revealed multiple large lobulated soft-tissue attenuation

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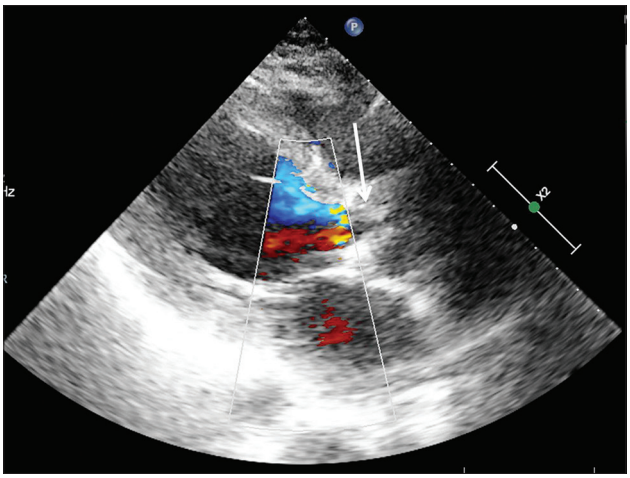

Figure 1: Echocardiography showing aortic stenosis (arrow) and mild mitral regurgitation

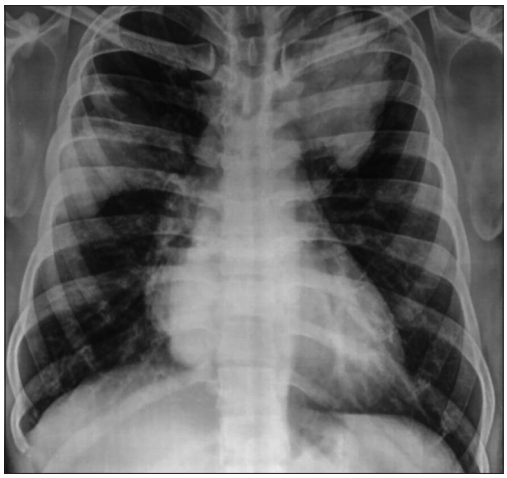

Figure 2: Plain radiograph chest showing homogeneous shadow of the nature of consolidation in the right mid zone and left upper zone

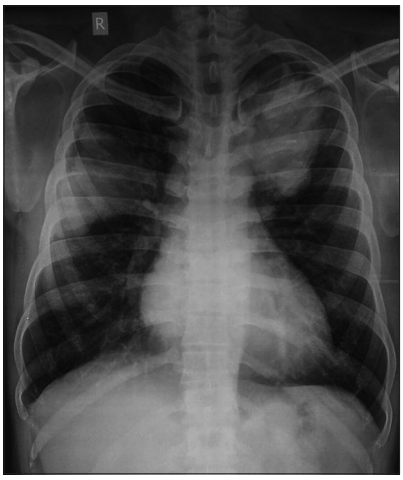

Figure 3: Repeat chest skiagram 4 weeks after a course of antibiotics showing no change in radiological findings

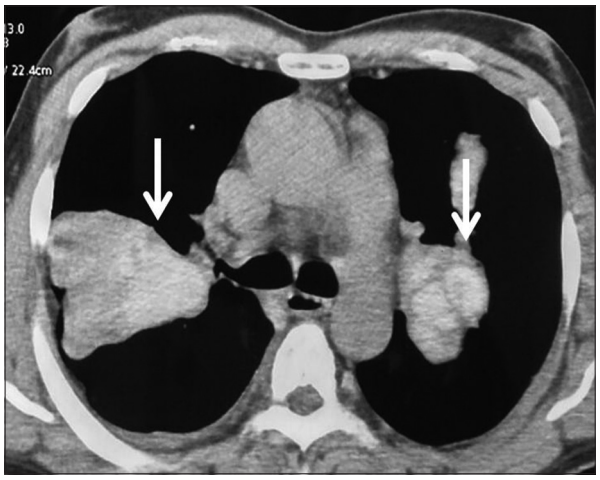

Figure 4: Axial computerized tomography of the chest revealing bulky lobulated mass-like opacities in both upper lobes and right lower lobe showing central dense enhancement and peripheral hypodensity identical in all the lesions

mass-like lesions in both lung upper lobes which are hypodense in the periphery (36–61 Hounsfield units [HU]) with enhancing, hyperdense central core (88–110HU) and specks of calcification. Both lung masses despite the large size were conspicuously devoid of any FDG avidity suggesting bland nonmetabolic nature [Figure 5]. A thin rim of lung tissue showing uniform FDG uptake around the left lung lesion represents an infective rim of consolidation. There was no mediastinal adenopathy. In view of the nonmetabolic nature of the large pulmonary lesions ruling out a mitotic or infective pathology and cascading centrifugal incremental density in the lesions, a vascular bleeding process has been contemplated and an immediate CT pulmonary angiography encompassing the chest was performed which unearthed central feeding vessel with extravasated contrast in the central portion of the left upper lobe mass [Figure 6] confirming it to be a hematoma. Having ruled out all possible known causes of Rasmussen's aneurysms, bilaterality of the lesions, large size and small feeding vessel, nonhemoptytic clinical state, a conservative approach was envisaged and the patient is on follow-up and corrective surgery for the aortic stenosis contemplated.

Discussion

Bilateral large nontraumatic, noninfective, spontaneous Rasmussen's aneurysm is a rare occurrence and largely unreported. Usual causation is small pulmonary artery branch vessel erosion by infective focus usually of tuberculous etiology, sometimes causing massive revealed hemoptysis often necessitating emergency bronchial artery embolization.[1] Erosive pseudoaneurysm of small caliber pulmonary arterial branch caused by an adjoining infiltrating pulmonary lesion is termed as Rasmussen's aneurysm. Fritz Waldemar Rasmussen, a 19th century Danish physician, first described the occurrence of dilation of the pulmonary artery in a tuberculosis cavity, rupture of which causes hemorrhage and hemoptysis,

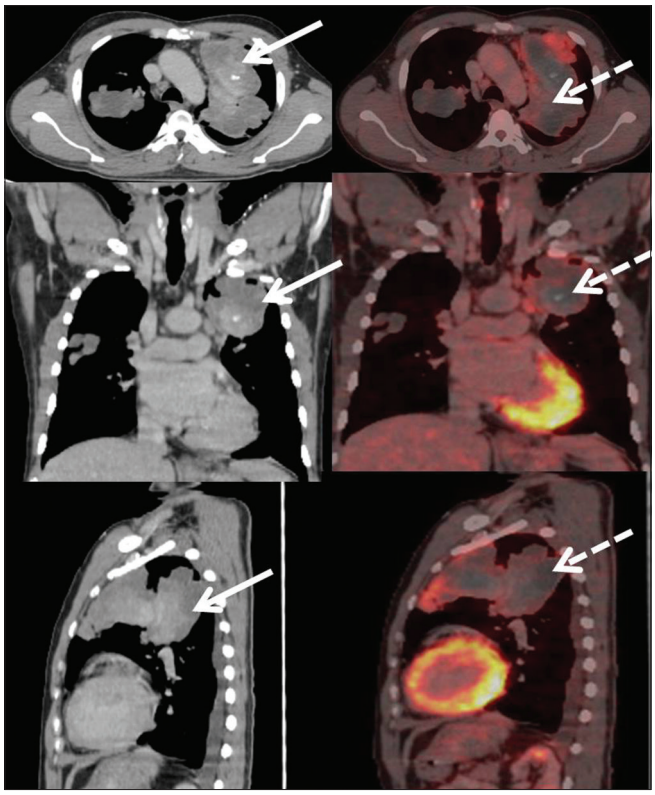

Figure 5: Axial, coronal, and sagittal fluorine 18-fluorodeoxyglucose positron emission tomography computerized tomography showing multiple large mass-like lesions in both lung upper lobes hypodense in the periphery with enhancing, hyperdense central core (arrow). Both lung masses despite the large size devoid of any fluorodeoxyglucose avidity (dotted arrow)

often massive and life threatening. Other documented causes are atherosclerosis, bronchiectasis, sarcoidosis, trauma, postcardiac catheterization, and postnecrotizing pneumonias.[2,3] Pathogenesis implicated is the progressive weakening of pulmonary arterial wall adventitia and media by granulation tissue, resulting in thinning of the arterial wall and formation of pseudoaneurysm. Subsequent rupture with hemorrhage produces hemoptysis. Angiographic intervention is warranted to unearth the source of bleeding and simultaneously attempt to embolize the bleeding source from Rasmussen's transformed culprit artery.[4,5] Chiruganti *et al*., have reported a rare occurrence of metastatic squamous carcinoma mediastinal necrotic lymph nodal mass eroding a large pulmonary artery branch causing a solitary giant Rasmussen's aneurysm with out any haemoptysis.[6] This case is the revelation of another such rare synchronous multiple bilateral giant pulmonary artery pseudoaneurysms of nonubiquitous spontaneous nature, by F18-FDG PET-CT in the course of investigation for suspected large bilateral pulmonary masses. The suspicion of pseudoaneurysm has been aroused by the absolute bland non-FDG avid nature of the large pulmonary lesions and the transitional radiographic contrast enhancement more in the center decreasing peripherally suggesting small leaks which become hypodense with time and migrate

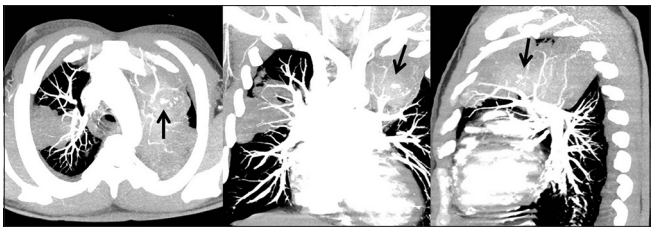

Figure 6: Axial, coronal, and sagittal sections of computerized tomography pulmonary angiography revealing the culprit vessel with extravasated contrast in the central portion of the left upper lobe mass (arrow)

peripherally displaced by fresh bouts of bleeding happening over long periods of times. The diagnostic confirmation has been achieved by the urgent and immediate CT pulmonary angiography performed immediately after the F18-FDG PET-CT to confirm and localize the bleeding source as a small pulmonary vessel and containment of bleeding within the hematoma itself with no revealed hemoptysis at any stage. This case outlines a second incidence of massive concealed giant Rasmussen's aneurysms identified surreptitiously by F18-FDG PET-CT in the course of investigation of cancer workup**.** Review of literature has not shown any cardiac or valvular disease as a causation of Rasmussen's aneurysm.

Conclusion

Rasmussen's aneurysm is usually a consequence of pulmonary tuberculosis focus involvement of a small peripheral pulmonary arterial or bronchial artery branch causing hemoptysis often fatal. Concealed large asymptomatic hematomas are a rare entity. Our case represents serendipitous revelation of giant, multiple bilateral Rasmussen's aneurysm with no trace of hemoptysis at any stage of patient's illness. The culprit vessel and the cause have been traced and identified but the etiology remains evasive.

Declaration of patient consent

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

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Nil.

Conflicts of interest

There are no conflicts of interest.

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