

Non haemoptytic massive Rasmussen's pulmonary artery aneurysm caused by aggressive cavitating squamous cell carcinoma metastasis

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ABSTRACT

The authors report an extremely rare occurrence of a massive aneurysm of a major pulmonary artery branch vessel caused by adjacent necrotizing aggressive squamous cell carcinoma metastatic mediastinal nodes. Despite the huge size, there was no hemoptysis due to the walling off effect by the necrotic nodes.

Keywords: F18-fluro deoxy glucose, necrotic nodal mass, pulmonary artery aneurysm, Rasmussen's aneurysm

INTRODUCTION

Rasmussen's aneurysm is a less frequently noticed entity usually associated with pulmonary tuberculosis presenting with massive hemoptysis due to involvement of small peripheral pulmonary or bronchial artery by the tubercular cavity or lesion.^[1] Involvement of a large central pulmonary artery by metastatic mediastinal nodal lesions is unreported so far. Despite the large size, hemoptysis was conspicuously absent adding to the rarity of occurrence.

CASE REPORT

A 65-year-old male of primary squamous cell carcinoma left buccal mucosa treated by left composite resection, and modified radical neck dissection 8 months back. Postsurgery local radiation with 25 fractions of 2 Grey (Gy) each completed just 4 months before, presented with generalized weakness and pain right hip region. There was no history of fall, fever, loss of weight or bleeding from any site. Clinical examination revealed no evidence of disease at the local site and no palpable masses anywhere in the body and there was no neurological deficit.

Hematological, bio chemical and metabolic parameters were within normal limits, except mild hypo chromic normocytic anemia. Initial imaging with magnetic resonance imaging of lumbo sacral spine revealed T1 hypointense and T2 hyperintense signal intensities in right sacral ala and iliac bone lesions suggestive of metastatic lesions. Patient was subjected to fluorine 18-fluro deoxy glucose positron emission tomography/computerized tomography (F18-FDG) for restaging and further evaluation. The primary site of left buccal region showed postoperative status with no morphological or metabolic abnormality. There was a metabolically active enhancing nodular mass lesion on the dorsum of the tongue measuring 30 × 19 mm with a standardized uptake value maximum (SUV max) of 5.77 [Figure 1a], similar nodular deposit in the cervical nuchal muscle measuring 22 × 21 mm with an SUV max of 4.82 [Figure 1b]. There were multiple bilateral metabolically active necrotic, nodular, sub pleural and parenchymal pulmonary metastasis with associated mild degree pneumothorax [Figure 2]. Mediastinum showed multiple metabolically active enlarged lymphadenopathy encompassing bilateral hilar, sub aortic, and para aortic lymphadenopathy which also showed conglomeration and central necrosis. Interestingly there was a large contrast filled cavitory area measuring 66.6 × 54.8 × 72.6 mm seen amidst the necrotic lymph nodal mass, devoid of any FDG avidity [Figure 3]. On close scrutiny the radiographic contrast collection was traceable up to one of the adjoining first order branch of left pulmonary artery [Figure 4]. There were multiple metabolically active mixed lytic sclerotic disseminated skeletal metastasis as well

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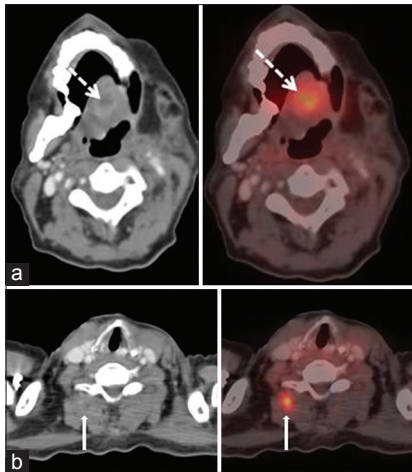


Figure 1: (a) Metabolically active enhancing nodular mass lesion on the dorsum of the tongue (dotted arrow). (b) Metabolically active nodular deposit in the cervical nuchal muscle (thick arrow)

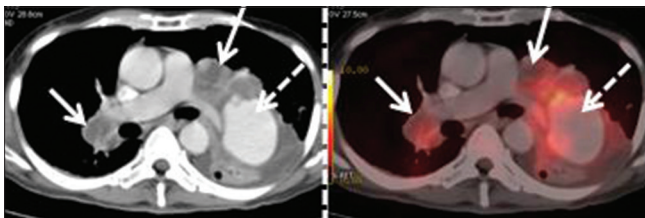


Figure 3: Axial positron emission tomography/computed tomography images of chest showing multiple metabolically active enlarged mediastinal lymphadenopathy with conglomeration and central necrosis (arrows) and a contrast filled cavitary area amidst the necrotic lymph nodal mass devoid of any fluoro deoxy glucose avidity (dotted arrow)

[Figure 5]. Due to his poor physical condition and the extensive disease load. Small asymptomatic unilateral pneumothorax requiring no intervention and the large extravasated contrast being restricted within the confines of the cavitating nodal metastasis explaining the stark absence of hemoptysis, patient was managed conservatively without any active intervention shifting from a curative to palliative intent.

DISCUSSION

Erosive pseudo aneurysm of small caliber pulmonary arterial branch caused by an adjoining infiltrating pulmonary lesion is termed as Rasmussen aneurysm. Fritz Waldemar Rasmussen a 19th-century Danish physician first described the occurrence of dilation of the pulmonary artery in a tuberculous cavity, rupture of which causes hemorrhage and hemoptysis, 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. The distribution is usually peripheral and beyond the branches of main pulmonary arteries.^[4] In the present era of selective catheter angiography, the entity of

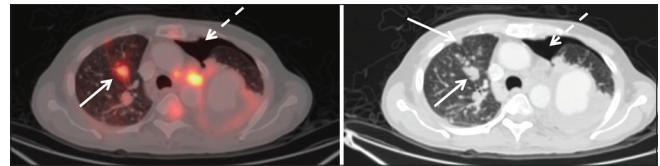


Figure 2: Axial positron emission tomography/computerized tomography images of chest showing multiple bilateral metabolically active sub pleural and parenchymal pulmonary metastasis (arrows) with associated left sided pneumothorax (dotted arrow)



Figure 4: High resolution axial and coronal computerized tomography chest showing the radiographic contrast collection traceable up to one of the adjoining first order branch of left pulmonary artery (arrow)

Rasmussen's aneurysm is a retrospective detection encountered while addressing suspected bronchial artery erosion being the cause of massive, intractable and life-threatening hemoptysis.^[5,6] Angiographic intervention is warranted to unearth the source of bleeding and simultaneously attempt to embolize the bleeding source from Rasmussen's transformed culprit artery.^[7,8] Our patient had extensive metastasis to skeleton, lungs, muscles, tongue and mediastinal adenopathy with significant necrosis. One of the aggressive and necrotizing lymph nodal mass has eroded the adjoining pulmonary artery branch, in this case a major branch vessel unlike smaller vessels usually seen in tuberculous cavities. The leaked out blood was contained within the necrotic lymph nodal mass which explains the absence of any revealed hemoptysis and a possible emergency bleeding situation despite the large sized extravasation. Conventional sites of aneurysm are peripheral and beyond the branches of main pulmonary arteries,^[4] however it was central in this case and involved a larger central pulmonary arterial branch. Reported cases of Rasmussen's aneurysm are the result of infective and chronic tuberculous cavity eroding the adjoining small caliber bronchial/pulmonary artery and none by an aggressive metastatic necrotic nodal mass. The case also highlights the aggressive, extensive and erosive nature of squamous cell carcinoma metastasis.

CONCLUSION

Rasmussen's aneurysm is a rare sequel of pulmonary tuberculosis presenting with massive hemoptysis usually involving a small peripheral pulmonary or bronchial artery. Involvement of a large central pulmonary artery by an aggressive necrotizing mediastinal lymph nodal mass is unreported so far. Our case is one such entity of this rarity with walled off collection of a large magnitude contained within the necrotic mass and nonhemoptytic manifestation being an additional associated rarity.

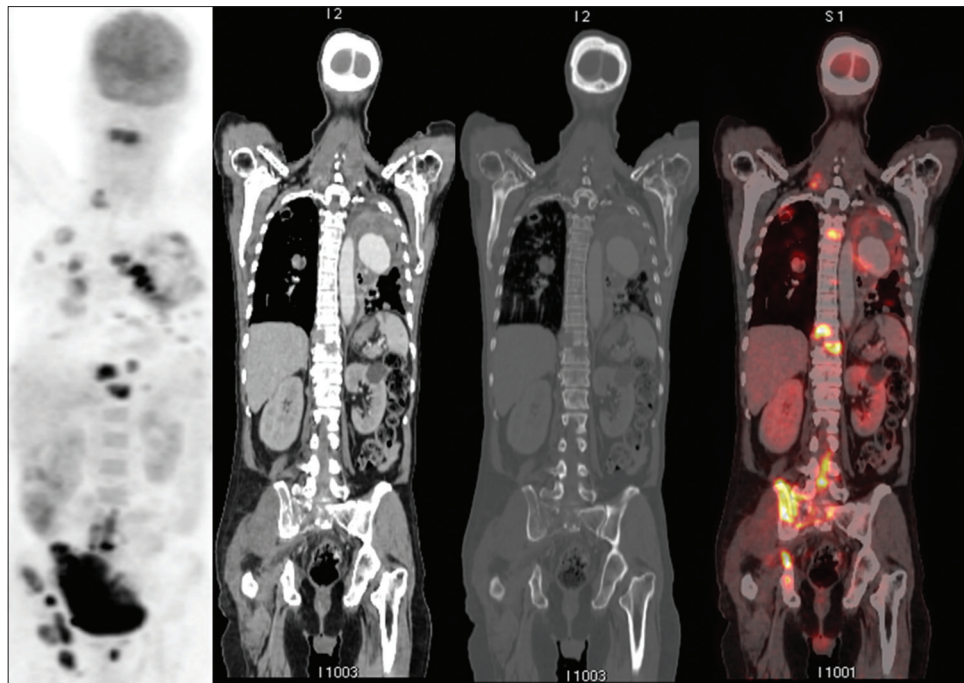


Figure 5: Maximum intensity projection and coronal positron emission tomography/ computerized tomography images revealing multiple metabolically active mixed lytic sclerotic disseminated skeletal metastasis (arrows)

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