DOI: 10.7860/JCDR/2019/38433.12444

Case Report

Internal Medicine Section

Curious Case of Myocardial Infarction Caused by Lung Abscess

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ABSTRACT

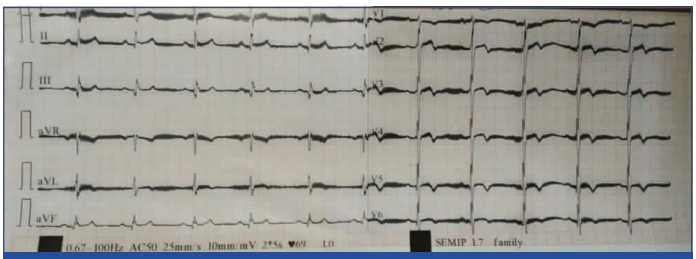
Lung abscess is a necrotizing infection of lung parenchyma with pus collection usually due to pyogenic organism. Embolic complications involving cardiac structures are extremely rare. Type 2 Myocardial Infarction (MI) denotes myocardial injury and necrosis secondary to coronary spasm, embolus, anaemia, hypotension, respiratory failure, etc. without obvious coronary artery disease. We report a case of type 2 MI complicating lung abscess which posed multiple challenges in diagnosis and management. On one hand, use of anti-ischemic therapy for MI enhances the risk of bleeding (haemoptysis) while on the other hand interrupting them to minimize bleeding predisposes to risk of thrombotic events.

Keywords: Coronary angiography, Embolic phenomenon, Lung parenchyma

CASE REPORT

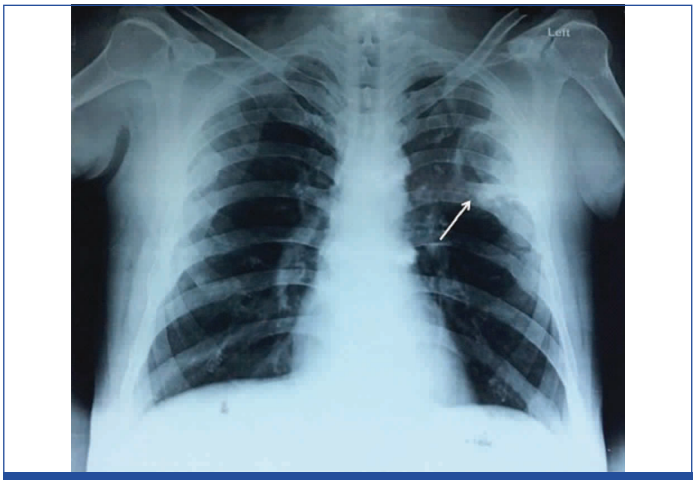
A 55-year-old male smoker presented to emergency department with retrosternal chest pain for eight hours. The pain was acute in onset with radiation to ulnar aspect of left arm and jaw. There was also associated diaphoresis. He was also suffering from cough with expectoration and effort dyspnea for last one month. There was also associated history of fever for the same duration. He was prescribed oral antibiotics for the same. As a known smoker his pack year was 15. There was no history of treatment for tuberculosis or bronchial asthma. There was no similar history in the family. He was suffering from type 2 Diabetes mellitus for last 5 years and was on Metformin and sulphonylurea combination therapy.

On examiantion, his vitals were stable. A 12 lead ECG revealed sinus rhythm with biphasic T waves in leads V1-V6 suggestive of anterior wall ischemia [Table/Fig-1]. His 2D echocardiography did not reveal any regional wall motion abnormality. Cardiac biomarkers (high sensitive troponins) were elevated and a diagnosis of Non ST Elevation MI (NSTEMI) was made. Patient was managed in coronary care unit with low molecular weight heparin, dual antiplatelets (aspirin 150 mg and clopidogrel 75 mg), statin (atorvastatin 80 mg), beta blocker (Metoprolol 50 mg) and nitrates (isosorbide mononitrate 20).



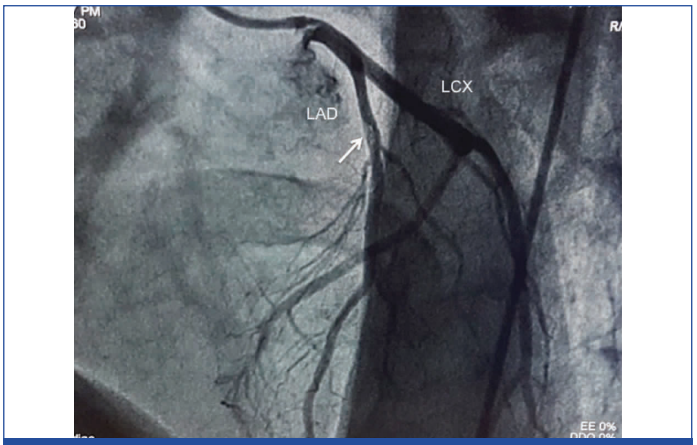
[Table/Fig-1]: A 12 lead ECG demonstrating sinus rhythm with biphasic T waves in leads V1-V6 suggestive of anterior wall ischemia.

On second day, he developed moderate haemoptysis, about 100 mL per day. He developed tachypnea (respiratory rate was 30 per minute) but was haemodynamic stable. His blood pressure was 110/80 mm Hg in right arm in supine position. A Chest-X ray PA view revealed thick walled cavity with air fluid levels on left upper lobe suggestive of lung abscess [Table/Fig-2]. Haematological tests revealed-haemoglobin (Hb)-12 gm%, total leucocyte count 12000/cmm^3, Differential Count (DLC) neutrophil 75%, lymphocytes 20%, eosinophil 2%, monocytes 1%, and basophils 1%. His serum urea was 60 mg/dL and serum creatinine was 1.8 mg/dL. PT/INR (Prothrombin Time/International Normalized Ratio) was within normal limits.



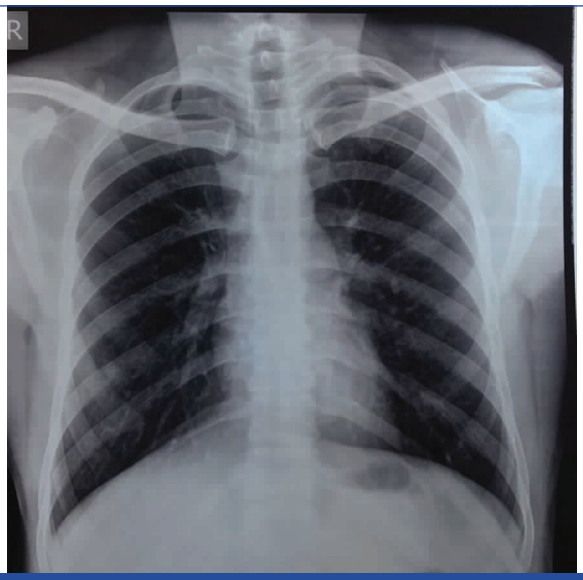
[Table/Fig-2]: Chest-X ray PA view depicting thick walled cavity (arrow) with air fluid levels on left upper zone.

Low molecular weight heparin and clopidogrel were discontinued in view of haemoptysis. A diagnostic coronary angiogram revealed grade II non occlusive thrombus in proximal part of Left Anterior Descending artery (LAD) without any underlying atherosclerotic plaque [Table/Fig-3]. Rest of the coronary angiogram was normal.



[Table/Fig-3]: Coronary angiogram showing grade II thrombus (arrow) in proximal part of left anterior descending artery (LAD), [LCX- left circumflex artery].

In view of concomitant lung abscess, presence of haemoptysis and absence of recurrence of chest pain, a decision to defer invasive procedure was taken and a symptom guided approach was planned. Patient was shifted to respiratory intensive care unit. Sputum smear was negative for Acid Fast Bacilli (AFB), gram stain and fungal elements. There was no induration in mantoux test. Molecular test for sputum was also not in favor of tuberculosis. Empirically intravenous antibiotics-cefoperazone, sulbactum and clindamycin were given for two weeks. He had improvement in his respiratory symptoms. Patient fortunately remained free of chest pain during hospital stay and was discharged on single antiplatelet (clopidogrel 75 mg), statin (atorvastatin 80 mg), beta-blocker (Metoprolol 50 mg) and oral nitrate (isosorbide mononitrate sustained release 30 mg). A follow-up chest X-ray at four weeks showed resolution of lung abscess [Table/Fig-4]. Patient refused a repeat angiogram and medical management was continued.



[Table/Fig-4]: Follow-up chest X-ray at four weeks showing resolution of lung abscess.

DISCUSSION

Lung abscess is defined as a collection of pus and necrotizing area in lung parenchyma [1]. There are multitude complications of lung abscess and embolic phenomenon is one of them. However, embolic complication are not very common and rarely have been reported in literature.

Acute MI most commonly occurs due to rupture of an atherosclerotic plaque with superimposed thrombus upon it [2,3]. However, there are other mechanisms for occurrence of MI [Table/Fig-5] [4]. Type 2 MI denotes myocardial injury and necrosis secondary to coronary spasm, embolus, anaemia, hypotension, respiratory failure etc., without obvious coronary artery disease [4]. Although non atherosclerotic causes contribute to 4-7% of all MIs, coronary embolism is definitely rare and exact incidence is not known [5]. There are multiple sources of coronary embolism, however, lung abscess has not been reported as a source in literature [6].

Myocardial infarction	Pathophysiology
Type 1	Spontaneous MI due to plaque rupture
Type 2	MI secondary to an ischemic balance like coronary artery spasm, coronary artery embolism, anaemia, hypertension hypotension with or without LVH.
Type 3	MI related to sudden cardiac death
Type 4a	MI related to percutaneous coronary intervention
Type 4b	MI related to Stent thrombosis
Type 5	MI related to coronary artery bypass grafting

[Table/Fig-5]: Classification of Myocardial infarction based on various pathophysiological mechanisms [4].

Erosion of a pulmonary vein by the lung abscess and embolization of septic material is the most probable cause of systemic embolism. Although literature has few case reports of cerebral embolism emanating from lung abscess, embolic complications involving cardiac structures are extremely rare and yet to be reported to the best of our knowledge [7]. In pre-antibiotic era mortality due to lung abscess was 75% but after antibiotics use mortality was reduced to 8.7% [8]. Most common microbial aetiology of lung abscess (>90%) is bacterial and anaerobes are the predominant organism isolated [9].

In the present patient, symptoms of lung abscess clearly preceded that of acute MI. The use of antithrombotics and antiplatelets led to haemoptysis which just unmasked the underlying upper lobe abscess. Co-existing systemic infection presents both diagnostic and therapeutic challenges in management of acute MI. Systemic infection can give rise to false rise in troponin levels and as well as non specific ST-T changes in electrocardiogram [10]. However, coronary angiogram in the present case unequivocally revealed thrombus in left anterior descending artery without any significant luminal narrowing - suggestive of type 2 MI. The use of antiplatelet, anti thrombotic agents and thrombolytic agents for acute MI predispose a patient for bleeding in presence of active systemic infections (lung abscess). In our patient due to haemoptysis we had to down size the antiplatelet and antithrombotic regimen putting the patient at higher risk of ischemic events. In such a scenario the clinician needs to strike a balance between the ischemic risk and bleeding risk for the patient.

Plaque rupture is not uncommon in the setting of infection [11]. Hence, an alternative theory can be postulated that coronary event (acute MI) was not result of septic emboli lodging into LAD but more likely to be *de novo* atherothrombotic lesion that was activated secondary to underlying inflammatory state. An additional argument against the "Embolic Hypothesis" from systemic circulation could be the proximal position of thrombus. However, it is now known from OCT (Optical Coherence Tomography) studies that coronary angiography underestimates the thrombus burden [12]. An IVUS or OCT, could have unequivocally demonstrated the presence or absence of underlying plaque rupture and thereby confirming the true cause.

CONCLUSION

Lung abscess with embolic phenomenon are not very common. Type 2 MI resulting from septic emboli is even rarer. Co-existing lung abscess poses both diagnostic and therapeutic challenges. The most common hurdle in this case is its management.

REFERENCES

[1] Seo H, Cha SI, Shin KM, Lim J, Yoo SS, Lee J, et al. Focal necrotizing pneumonia is a distinct entity from lung abscess. Respirology. 2013;18:1095-100.

[2] Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, et al. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC) Eur Heart J. 2016;37:267-315.

[3] Bugiardini R, Manfrini O, De Ferrari GM. Unanswered questions for management of acute coronary syndrome: risk stratification of patients with minimal disease or normal findings on coronary angiography. Arch Intern Med. 2006;166:1391-95.

[4] Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD. Third universal definition of myocardial infarction. Eur Heart J. 2012;33:2551-67.

[5] Henriques JP, Zijlstra F, Ottervanger JP, de Boer MJ, van't Hof AW, Hoorntje JC, et al. Incidence and clinical significance of distal embolization during primary angioplasty for acute myocardial infarction. Eur Heart J. 2002;23:1112-17.

[6] Taniike M, Nishino M, Egami Y, Kondo I, Shutta R, Tanaka K, et al. Acute myocardial infarction caused by a septic coronary embolism diagnosed and treated with a thrombectomy catheter. Heart. 2005;91:e34.

[7] Albrecht P, Stettner M, Husseini L, Macht S, Jander S, Mackenzie C, et al. An emboligenic pulmonary abscess leading to ischemic stroke and secondary brain abscess. BMC Neurology. 2012;12:133.
[8] Moreira Jda S, Camargo Jde J, Felicetti JC, Goldenfun PR, Moreira AL Porto Nda. Lung abscess: analysis of 252 consecutive cases diagnosed between 1968 and 2004. J Bras Pneumol. 2006;32:136-43.
[9] Wang JL, Chen KY, Fang CT, Hsueh PR, Yang PC, Chang SC. Changing bacteriology of adult community-acquired lung abscess in Taiwan: *Klebsiella pneumoniae* versus anaerobes. Clin Infect Dis. 2005;40:915-22.
[10] Lippi G, Targher G, Franchini M, Plebani M. Genetic and biochemical heterogeneity of cardiac troponins: Clinical and laboratory implications. Clin Chem Lab Med. 2009;47:1183-94.
[11] Kwong JC, Schwartz KL, Campitelli MA, Chung H, Crowcroft NS, Katz K, et al. Acute myocardial infarction after laboratory-confirmed influenza infection. N Engl J Med. 2018;378:345-53.
[12] Jia H, Abtahian F, Aguirre AD, Lee S, Chia S, Lowe H, et al. In vivo diagnosis of plaque erosion and calcified nodule in patients with acute coronary syndrome by intravascular optical coherence tomography. J Am Coll Cardiol. 2013;62:1748-58.

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FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Aug 26, 2018**
Date of Peer Review: **Oct 05, 2018**
Date of Acceptance: **Oct 26, 2018**
Date of Publishing: **Jan 01, 2019**