CASE REPORT

Occam's Razor? Not Always..! A Case Report of Acute Myocardial Infarction in a Patient with Fungal Pneumonia

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Abstract

Pneumonia is a leading cause of mortality worldwide. Cardiovascular problems affect up to 30% of patients admitted to hospitals for community acquired pneumonia (CAP), both immediately and up to 10 years afterward. Here we present a case of elderly patient with fungal pneumonia who developed acute myocardial infarction (AMI) on day 2 of admission. Pre-existing diseases, relative ischemia, sympathetic system activation, systemic inflammation, and direct pathogen-mediated injury to the cardiovascular system all play a role in cardiac problems. The actual processes governing direct host–pathogen interactions are of tremendous interest in the search for prospective CAP therapeutic and preventive strategies.

Keywords: Acute myocardial infarction, Community acquired pneumonia, Fungal pneumonia.

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Introduction

Pneumonia and cardiovascular diseases (CVD) are two of the most common causes of morbidity and mortality in the world.[1] Acute infections like CAP can harm the cardiovascular system through a variety of processes, causing or exacerbating cardiovascular problems such heart failure, acute coronary syndromes (ACS), cardiac arrhythmias, and strokes.[2] This problem tends to get worse with the age.

We present a case of elderly male patient with an isolated infection of the lung parenchyma with Candida in the form of focal pneumonia of the right upper lower lobe complicated by a myocardial infarction on day 2 of hospitalization.

Case Report

A 65-year-old male with no other comorbidities brought to emergency room with complaints of cough, fever, and hemoptysis since 3 weeks.

On examination patient was febrile, tachypneic, and SpO_2 was 86% at room air. On auscultation patient had bronchial breath sounds and crepts all over his right-sided lung fields. Other systems examination was fairly normal.

Relevant investigations were done; chest X-ray (CXR) revealed (Fig. 1) right-sided consolidation in upper zone and patient was admitted according to the CURB 65 score keeping pneumonia on mind and was commenced on antibiotics.

Patient was reviewed during morning rounds; cardiac monitor showed sinus tachycardia and ECG revealed extensive ST elevation in all chest leads. Sputum gram stain indicated the presence of fungal filaments which pushed us to ask for fungal culture which finally revealed the presence of Candida. Patient showed considerable improvement (Fig. 2) after being treated with antifungals (Oral Fluconazole 400 mg for 2 weeks), low molecular weight heparin, aspirin (commenced later on after cessation of hemoptysis), statins, and other supportive measures.

Discussion

Increased cardiac stress, hypoxemia, and inflammation may all have a role in acute cardiac events in pneumonia patients.[3] Pneumonia as such is a prothrombotic state, endotoxins, and coagulation activation,[4] and indicators of enhanced platelet activity have been linked to AMI in individuals with acute pneumonia.

The third universal definition of MI[5] can be used to define the type of MI. On coronary angiography, type I MI was characterized as ischemia caused by a main coronary event such as plaque erosion or rupture, intraluminal thrombus, or coronary dissection. At the

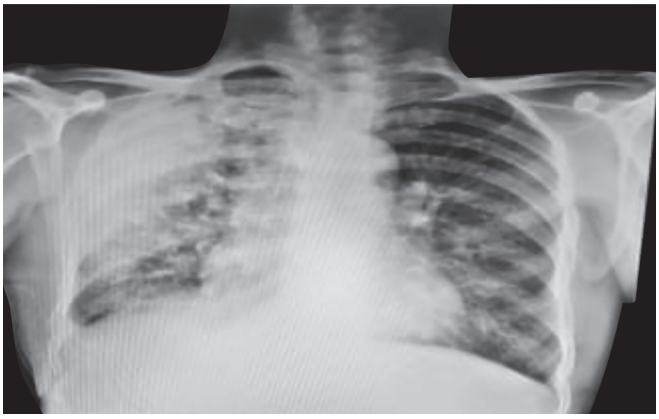

Fig. 1: CXR at the time of admission

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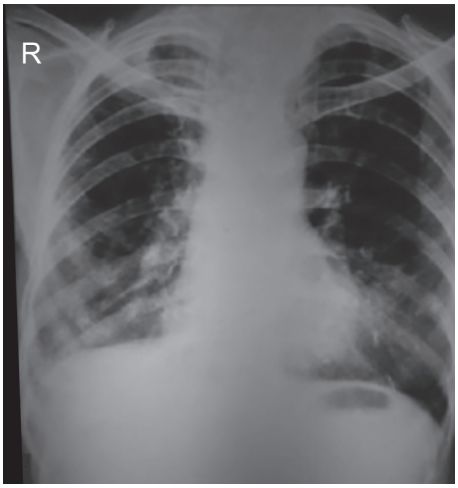

Fig. 2: CXR 5 weeks after initiation of antifungal treatment

commencement of MI symptoms, type II MI was defined as the lack of evidence of plaque rupture on coronary angiography and at least one of the prespecified supply/demand mismatch situations, such as acute infections.[6]

Acute pneumonia has also been linked to a four-fold increase in the risk of cardiovascular events in the first month[7] and a more than 100-fold increase in risk in the first 3 days following admission.[8] When compared to other inpatients, individuals hospitalized for pneumonia have a five times greater risk of acute MI.[7] Pneumonia, dubbed as the "friend of the elderly" by Sir William Osler, is still one of the most lethal infections in frail comorbid patients, acting more as a trigger for organ failure and mortality than as a cause of death.[9]

Conclusion

The law of parsimony, Occam's Razor, encourages doctors to look for a unifying diagnosis. However, it is advisable to investigate concurrent diagnoses when one ailment predisposes to or is related with another. In hospitalized patients with severe CAP, a combination diagnosis of CAP and AMI is prevalent. AMI should be evaluated as a potential cause in situations when the clinical course of a hospitalized patient with CAP is compounded by clinical failure.

Pneumonia and AMI are linked, which is important clinically because elderly patients who are at high risk for both may not present with the typical presentation of initial retrosternal chest pain with AMI, but rather with mental status changes or initial difficulty in breathing. Since these symptoms can easily be attributed to pneumonia in patients who have already received a diagnosis of CAP, there is a distinct risk of misdiagnosis, where treatment for AMI becomes more valuable.

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