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RESEARCH ARTICLE

ACUTE MYOCARDIAL INFARCTION & PULMONARY TUBERCULOSIS IN A 18 YEAR OLD FEMALE PATIENT

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ABSTRACT

Myocardial infarction in a young female without any known genetic or family history of heart disease nor any history of hyperlipidemia is rarely seen. We document a case of an 18 year old female with pulmonary tuberculosis developing myocardial infarction.

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INTRODUCTION

Tuberculous involvement of coronary arteries is very rare, although mycobacterium tuberculosis, as some other infectious agents, may also act in the coronary vessels by activating the inflammatory mechanism of atherosclerosis.

Case Presentation

We present here a case of a young 18 year old female who presented to us with complains of cough & fever since 15 days getting treatment from local general practitioner. There was no family history of ischaemic heart disease or any sudden unexplained deaths. On admission the chest x-ray done showed right sided consolidation with cavitation and left lower lobe consolidation with pleural effusion. (Fig.1) On examination, she had left sided bronchial breathing & bilateral coarse crepts. Saturation was 89% on room air, increased to 95% after giving nebulisation with bronchodilators. Hb was 8 gm %, WBC counts of 4000, ECG was within normal limits (Fig 2) and HIV, Hbsag were negative. Our impression was pulmonary tuberculosis with secondary bacterial / viral pneumonia. She was started on anti tuberculous treatment in view of chest x-ray findings. Her sputum AFB smear of 3 samples came negative.

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On day 1 the patient complained of breathlessness with SPo2 of 89 % on room air. On day 2 she became more breathless early morning as SPo2 60 % on room air. On day 3 she was shifted to ICU and NIV trial was given, but intubated after 3 hours. A differential diagnosis of Impending ARDS, H1N1 suspect was made. Blood transfusion was given as her Hb dropped to 7.8 gm%. H1N1, Malaria, Dengue, Leptospirosis were negative. Day 4 Bronchoscopy was done and Bronchoalveolar lavage sent for AFB & Bactec culture. She was maintaining with 90% FiO2, her heart rate remained around 120/min. Blood culture, gram stain for sputum & BAL was negative, however BAL came positive for AFB smear. Antibiotics were upgraded to iv piperacillin tazobactum, linezolid & amikacin Day 5 Her condition remained stable. In the night patient had acute increase in breathlessness. ST elevation in leads $V_1 – V_5$ on ECG (Fig 3) & elevated cardiac markers. Patient collapsed & could not be revived nor thrombolysed.

DISCUSSION

Epicardial coronary arteritis is a rare event in which the coronary injury may lead to myocardial ischaemia/infarction with or without associated coronary artery thrombosis. Various microorganisms cause vasculitis in vessels of any size and involve the vessel by extension of the acute or chronic infective process from an adjacent tissue or organ or from the lumen by hematogenous spread.

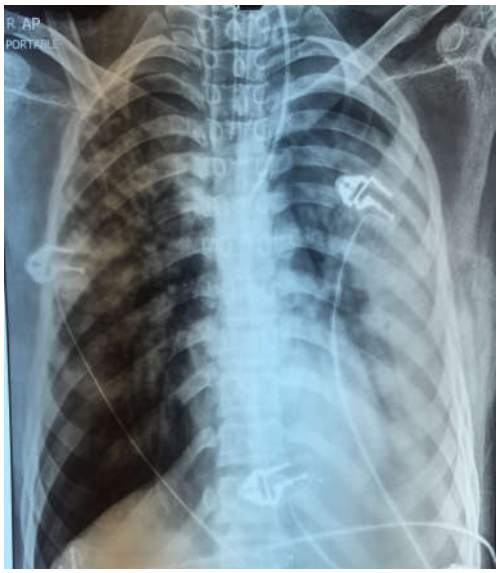

Figure 1. Chest Xray PA view showing right sided consolidation with caviation and left lower lobe consolidation with pleural effusion

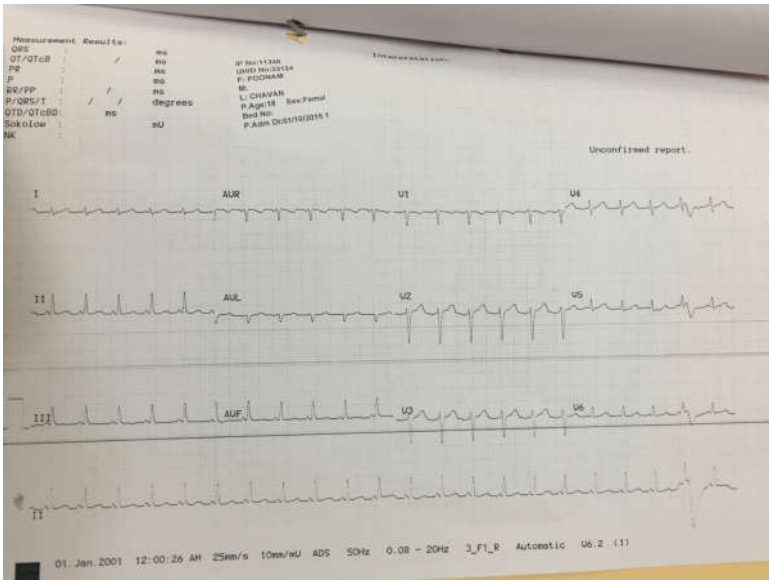

Figure 2. ECG showing sinus rhythm

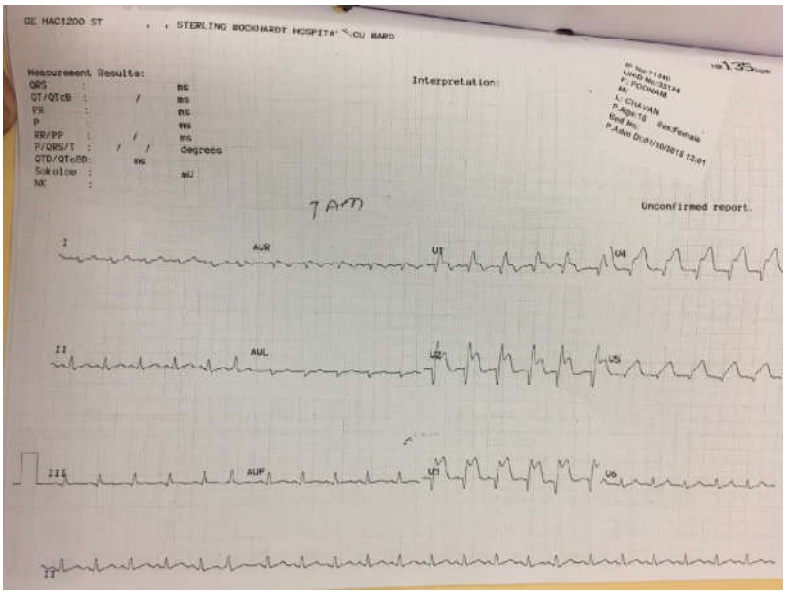

Figure 3. ECG showing ST elevation in leads V_1- V_5

The inflammatory response produces variable reactions including granulomatous response as seen in tuberculosis, syphilis, leprosy, suppurative inflammation bacteria, proliferative response as seen in typhoid. The most important angitic infections affecting the coronary arteries are tuberculosis and syphilitic arteritis. (Lie, 1987) Tuberculous granuloma may involve the advnetitia intima or the entire wall. Tb coronary arteritis can occur in myocardial and pericardial tuberculosis. (Rose, 1987) A study by Kazachkov EL. holds responsible coronaritis caused by tuberculosis as a cause of myocardial infarction (Kazachkov, 2001). Ditiatkov *et al*. reported the prevalence of coronary heart disease, in relation to age and gender, among patients with pulmonary tuberculosis. Coronary artery disease was found in 41 of 491 patients that they studied and it was more frequent among women and older age groups (Ditiatkov *et al*., 2006). Kinare *et al*., on the other hand, reported a case of a 19 year old male who ended fatally due to a large ventricular aneurysm obtained from myocardial infarction caused by tuberculous coronaritis of the left anterior descending branch (Rota *et al*., 2005). Although there are numerous cases of patients with acute myocardial infarction that are free of classic risk factors, we believe that the case we presented draws attention due to our patient's young age and female gender. Myocardial infarction is an uncommon condition in younger women in the absence of coronary risk factors (Kinare and Bhatia, 1971).

Conclusion

As the patient did not have any other known coronary artery disease, risk factors, we held responsible mycobacterium tuberculosis for causing coronary arteritis & occurrence of acute myocardial infarction in this young girl.

Vasculitis in tuberculosis is rare. We believe that the presentation of this rare case of myocardial infarction in a patient with active pulmonary tuberculosis should encourage researchers to investigate the potential role of mycobacterium tuberculosis in pathogenesis of coronary heart disease.

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