MULTIPLE MYELOMA PRESENTING WITH NSTEMI

M. Ravi kiran, A. Praveen

ABSTRACT:
Multiple myeloma, also known as plasma cell myeloma, is a cancer of plasma cells, a type of white blood cell normally responsible for producing antibodies. Multiple myeloma is associated with hyperviscosity of the blood depending on the properties of the para-protein. Here we are reporting a case of multiple myeloma presented with a cute coronary syndrome with Troponin T positive with regional wall motion abnormality but with normal coronaries.
Keywords: Multiple myeloma, coronary artery disease.

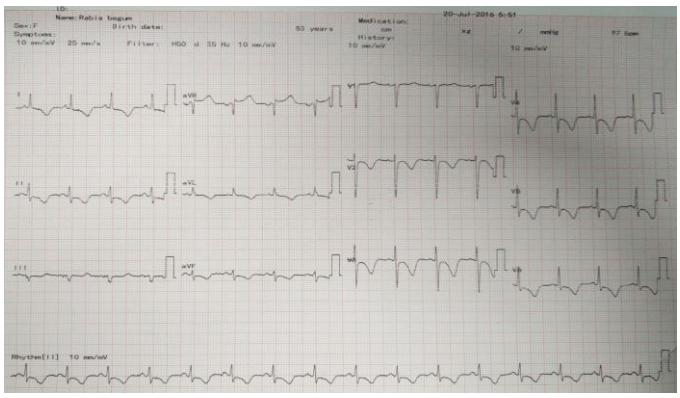
INTRODUCTION

Multiple myeloma, also known as plasma cell myeloma, is a cancer of plasma cells, a type of white blood cell normally responsible for producing antibodies [1]. Initially, there are often no symptoms. When advanced, bone pain, bleeding, frequent infections, and anemia may occur [2]. Multiple myeloma is considered treatable but generally incurable [3] . Remissions may be brought about with steroids, chemotherapy, thalidomide or lenalidomide and stem cell transplant[3]. Bisphosphonates and radiation therapy are sometimes used to reduce pain from bone lesions [1,3]. Multiple myeloma is associated with increased risk of thrombosis. We are presenting a case of NSTEMI due to coronary thrombosis.

CASE REPORT

A 53 year old female known case of multiple myeloma since 14 years now on chemotherapy with Bortezomib , Melphalan and omnicortil came with complaints of shortness of breath of 2 days duration associated with orthopnea and paroxysmal nocturnal dyspnea. There is no history of chest pain , palpitations , pedal odema , fever. Patient has hypertension , diabetes , hypothyroidism. Patient had a history of D10 vertebral plasma cytoma 14 years back for which transthoracic open biopsy, curettage with bone grafting was done. She underwent radiotherapy with cobalt^{60} to D8 – D12 following curettage. At admission pulse rate was 90 per minute and blood pressure was 160/100 mm of Hg. On examination patient has bilateral basal crepititions. ECG (Fig.1) showed T inversions in V1 to V6, I , aVL , II , Avf with ST coving. Chest X ray (Fig.2) showed expanding osteolytic lesions in left ribs. 2D echo showed regional wall motion abnormality in LAD territory with moderate LV systolic dysfunction. Troponin T (strip test) was positive. Creatinine phosphokinase was 663 IU/L , NT pro-BNP was 13918 pg/ml. Lab investigations showed hemoglobin of 12.1 gm/dl , total leukocyte count of 6000 cells/mm^3 , platelet count of 2.6 lakhs/mm^3 , blood urea of 16 mg/dl , serum creatinine of 0.7 mg/dl , serum albumin of 2.4 gm/dl. Thyroid profile was normal. HRCT chest (fig.4) showed areas of ground glass opacities in both lungs suggestive of pulmonary edema and multiple lytic lesions in bony thorax. Coronary angiogram showed recanalised LAD. Quantitative Troponin T was 0.072 ng/ml after 10 days which was done to see whether it was falsely elevated or has come to normal after acute coronary event.

Figure 1: ECG showing T inversions in V1 to V6 ,I , aVL , aVF and lead II.



Article received on 01 OCT 2016, published on 31 OCT 2016.
RAVI KIRAN. M[1], PRAVEEN. A[2]
[1] DM student, Department of Cardiology, NIMS, India
[2] DM student, Department of Medical oncology, NIMS, India
Corresponding author: RAVI KIRAN.M
Email: ravikiran927@gmail.com

Figure 2: Chest X ray showing osteolytic lesions in left ribs.

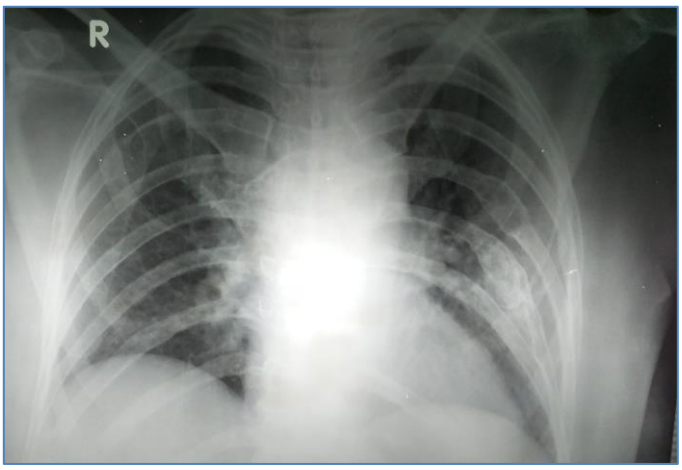


Figure 3: Parasternal short axis 2D echo image showing dilated globular LV (6.5x5.6 cm).

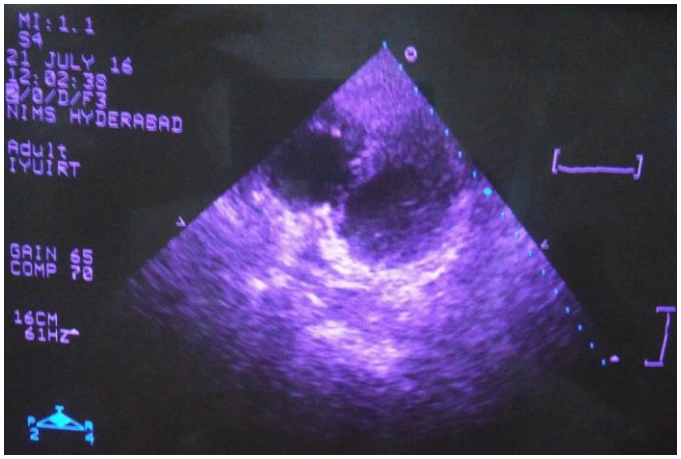
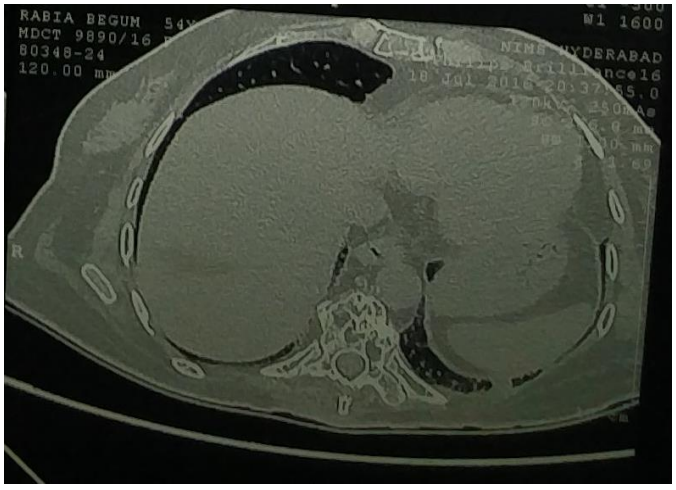
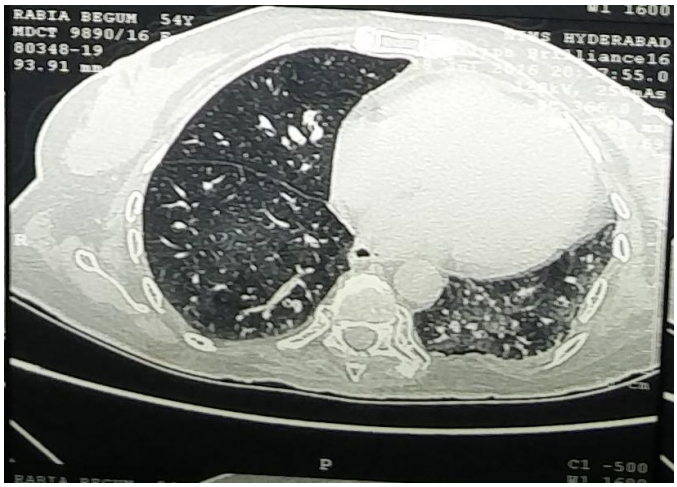


Figure 4: HRCT chest showing ground glass opacities and osteolytic lesions





DISCUSSION:

This patient presented with shortness of breath with raised cardiac enzymes with left ventricular systolic dysfunction and patient had risk factors for coronary artery disease like hypertension, diabetes mellitus, hypothyroidism, history of radiotherapy and chemotherapy. Coronary angiogram was performed 1 week after stabilization but showed no atherosclerotic coronary artery disease. Patient might had a thrombotic occlusion of the coronary artery which might have resolved with treatment.

Patients with multiple myeloma (MM) are at an increased risk of venous and arterial thrombosis. The pathogenesis remains unclear, but probably involves several factors such as activation of pro-coagulant factors, acquired activated protein C resistance, and inflammation. This patient is also on chemotherapy with bortezomib, melphalan and omnicortil. Chemotherapy augments the risk of cancer-associated thrombosis by inducing vascular damage. Patients with MM have a high risk of venous thromboembolism (VTE) [4]. The relationship between MM and arterial thromboembolism (ATE) is less clear. Libourel and colleagues reported a high incidence of arterial thromboembolism in MM patients undergoing chemotherapy [5].

A recent report used population-based data from Sweden that included 18,627 patients with multiple myeloma (MM) and 5326 patients with monoclonal gammopathy of undetermined significance (MGUS), and found that their risk of both ATE and VTE was

significantly higher than that of matched controls [7]. Similarly, Libourel et al report a higher-than-expected ATE frequency in 195 newly diagnosed MM patients receiving 3 different chemotherapy regimens. They found an unusually high incidence of arterial thrombosis, a complication generally considered rare in MM patients. In both series it was not mentioned whether the arterial thrombosis was in peripheral arteries or in coronaries arteries.

Increased serum viscosity is more frequently noted in patients with heavy chain immunoglobulin A. Serum electrophoresis of this patient showed M protein spike of Ig G type.

A false positive elevation of Troponin T was also considered in this patient because CAG showed normal coronaries.

Cardiac troponins play a central role in diagnosis and risk stratification in acute coronary syndromes, but Troponins are recognized as markers of cardiac myocyte injury, not of the etiology of injury. A wide range of clinical conditions has been associated with increased Troponin values [6], most or all of which have been shown to entail cardiac injury. Thus, it is necessary to consider these conditions when investigating unexpected increases in cardiac Troponins. But in this patient rise and fall in cardiac enzymes associated with left ventricular systolic dysfunction favors a coronary event.

In conclusion, in our case the cause for NSTEMI was considered to be atherosclerotic occlusion of LAD due to the presence of CAD risk factors like elderly female, diabetes and hypertension. But CAG demonstrated recanalised LAD; there is no evidence of any atherosclerotic changes. This may be due to the disease process. As peripheral arterial thrombosis was described in literature before, we want to report this case for its rarity for coronary thrombus.

REFERENCES:

1. Raab MS, Podar K, Breitkreutz I, Richardson PG, Anderson KC (July 2009).Multiple myeloma. Lancet. 374 (9686): 324–39.

2. Plasma Cell Neoplasms (Including Multiple Myeloma)—Patient Version. NCI. Retrieved 8 August 2016.

3. Plasma Cell Neoplasms (Including Multiple Myeloma) Treatment (PDQ®)–Health Professional Version. NCI. July 29, 2016. Retrieved 8 August 2016.

4. Palumbo A, Rajkumar SV, Dimopoulos MA, et al. Prevention of thalidomide- and lenalidomide-associated thrombosis in myeloma. Leukemia 2008;22(2):414-423.

5. Libourel EJ, Sonneveld P, van der Holt B, de Maat MPM, Leebeek FWG. High incidence of arterial thrombosis in young patients treated for multiple myeloma: results of a prospective cohort study. Blood 2010;116(1):22-26.

6. Thygesen K, Alpert JS, White HD, Jaffe AS, Apple FS, Galvani M, et al. Universal definition of myocardial infarction. Circulation2007;116:2634-2653.

7. Kristinsson SY, Pfeiffer R, Bjorkholm M, et al. Arterial and venous thrombosis in monoclonal gammopathy of undetermined significance and multiple myeloma: a population-based study [abstract]. Blood (ASH Annual Meeting Abstracts) 2009;114(22). Abstract 1872.