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Case Report

SEVERE "ASYMPTOMATIC" (? IDIOPATHIC) MYOCARDIAL CALCIFICATION: A CASE REPORT

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ABSTRACT
Myocardial Calcification, a rare clinical occurrence can occur due to 2 pathological mechanisms; 1) Metastatic calcification (due imbalance of Calcium Phosphate metabolism resulting from CRF, destructive bone lesion or hyperparathyroidism,), or 2) Dystrophic calcification (end result of myocardial injury). Calcification of myocardium without aforementioned reasons is very rare. In elderly age group, calcifications of Mitral/Aortic annulus are known. But Massive calcification of Myocardium is very rarely reported. In most of the reported cases causative factor could be identified. These cases had Cardiac symptoms &/or arrhythmias on ECG. In present two case reports, Myocardial Calcification was picked up when patients were investigated for No cardiac complaints. These two patients had no cardiac complaints. It was not possible to find a plausible explanation for Calcium deposition in myocardium. ECG shows no rhythm disturbances and hemodynamic studies were within normal range.

***Keywords*:** *Myocardial Calcification*

INTRODUCTION
Myocardial calcification is an uncommon clinical occurrence. Calcification of myocardium by two basic mechanisms. They are (I) Metastatic calcification, which occurs calcium phosphate metabolism is deranged either via increased calcium release from bone or from excessive gut absorption results in the deposition of calcium in normal tissue (leading to increased levels of calcium in blood and may be associated with calcification of other organs); *and*
(2) Dystrophic calcification, which occurs in dead or degenerative tissue in the presence of normal calcium/phosphate balance. In the heart, dystrophic calcification may occur in infarcts, ventricular aneurysms, and tumors. Mitral annular calcium or pericardial calcification may also invade the myocardium and cause dystrophic calcification.
Cases reported so far in literature have some symptoms or arrhythmias. In these 2 cases patient were asymptomatic, in sinus rhythm and calcification is detected by chance.

Keywords: *Myocardium, Calcification, Myocardial Injury, Endomyocardial Fibrosis, Normal Coronaries*

CASES
Case Report no 1
Mr.G, 68 yr. old male, is a physical labourer by profession and active in his profession till the present admission. He had no history of any physical illness in past including Tuberculosis. He is diabetic since 2-3 yrs., on regular follow up and controlled blood sugar levels, non-hypertensive and had no addictions.He consulted Orthopaedic dept. for back injury following a fall. Routine spine X-rays revealed radio opaque shadows in the thoracic region. Chest X-rays showed evidence of large calcium in cardiac shadow. He was referred to Cardiology for further evaluation.
As mentioned earlier, patient had no cardio respiratory. General Physical examination was unremarkable with normal BP and Pulse.. Cardiac Examination revealed normal heart sounds with normal split. No additional sounds or murmurs were heard.
Patient's haematology reports showed Hb%=12.5 gm.%, TC – 7,600 cells/ mm3, DC – N- 71, L- 27, E- 02, ESR – 35 mm/hr.,FBS – 133 mg/dL, PPBS – 204 mg/dL, RBS – 198 mg/dL, Glycosylated Hb – 5.4, Urea – 35 mg/dl, Creatinine – 0.73 mg/dL., CPK – 67 IU/Ml, CKMB – 20 IU/ml, Sodium – 139.5 mEq/L,

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Potassium – 4.4mEq/L, Chloride – 100.3 mEq/L, Bicarbonate – 26.8 mEq/, PTT – T- 19.3sec, C- 15.0sec, INR- 1.30. Sr.Calcium – 8.8 mg/dl, Sr.Phosphorus – 4.0 mg/dL, Albumin – 4.2 gm/dl\L, Alk. Phosp – 77, Urine analysis showed a Specific gravity- 1.010; PH- 6.5; Urobilinogen- Normal; leucocytes- 2-4/hpf; epithilial cells – 2-3/hpf.

From the figure 1ECG showedsinus rhythm, regular rate with no extra beats. However, inferior leads showed "q" waves, T-inversion in I and aVl& R wave in L-III was notched. Figure 2 shows theReview of Chest X-ray showed evidence of large chunks of calcium in cardiac area.

Echocardiogram showed hyper echoic calcium in basal and mid portions of LV (all walls) (Figure3); apex being free. RA, RV and LA showed no evidence of Calcium. Pericardium and endocardium were free of Calcium. All 4 valves were morphologically normal with no evidence of calcium and functioning normally. LV systolic function was impaired EF of 35% due "splinting" of myocardium and apex was on contracting effectively.

CT and Cardiac MRI revealed massive calcification of LV myocardium, particularly basal and mid portion, not confined to one coronary distribution, with sparing of pericardium and endocardium (including valves) as shown in the figure 4,5.

MRI Pictures

Right heart study coronary angiogram was performed. Heart rate was 68 bpm, Mean RAP = 3 mm of Hg, RV = 28/0/10 mm of Hg, PAP =28/16/20 mm of Hg, PCWP = 10/4/7 mm of Hg, and SAP = 120/80 mm of Hg. No attempt was made to enter LV with pigtail, for fear of dislodging any calcium speck which lead embolic phenomenon in asymptomatic patient. Plane cine fluoroscopy of thorax as shown in figure 5a showed massive calcification or myocardium especially of basal and mid segments of LV, not limited to any one coronary artery distribution. Coronary angiogram revealed normal coronaries (figure 6a, 6b).

Coronary Angiogram

At this juncture, the patient had no fractures or any orthopaedic issues and as his back pain reduced, he refused further investigation and got himself discharged. However, patient is on OP follow up and continues to be asymptomatic.

Case Report no 2

MrsU, 78 yr. old lady visited Medicine dept. with features suggestive of LRTI. She was not a known Hypertensive or Diabetic. No significant past Medical history. Her General Physical Examination and systemic examination of all systems were normal. On routine evaluation, her ECG was normal but Chest x-ray showed calcification of Cardiac.

Echocardiogram showed hyper echoic calcium in basal and mid portions of LV (all walls); apex being free. RA, RV, LA, pericardium and endocardium were free of Calcium. All 4 valves were morphologically normal with no evidence of calcium with evidence of Moderate MR. LV systolic function was impaired EF of 37%.

Patient's haematology reports showed Hb%=11 gm%, TC – 8,100 cells/ mm3, DC – N- 68, L- 30, E- 02, ESR – 09 mm/hr.,FBS – 98 mg/dL, PPBS – 137 mg/dL, Glycosylated Hb – 5.2, Urea – 45 mg/dl, Creatinine – 1.1 mg/dL., CPK – 70 IU/Ml, CKMB – 29 IU/ml, Sodium – 129 mEq/L, Potassium – 4.1mEq/L, Chloride – 98.4 mEq/L, Bicarbonate – 27.4 mEq/, PTT – T- 19.0sec, C- 15.0sec, INR- 1.20. Sr.Calcium – 9.0 mg/dl, Sr.Phosphorus – 4.3 mg/dL, Albumin – 4.4 gm/dl\L, Alk. Phosp – 56,

Urine analysis showed a Specific gravity- 1.010; PH- 6.5; Urobilinogen- Normal; leucocytes- 2-4/hpf; epithilial cells – 2-3/hpf.

Right heart study coronary angiogram was performed. Heart rate was 76 bpm, Mean RAP = 5 mm of Hg, RV = 24/3/12 mm of Hg, PAP =24/14/20 mm of Hg, PCWP = 12/5/8mm of Hg, and SAP = 141/84 mm of Hg. LV was not entered, for fear of dislodging any calcium speck which lead embolic phenomenon in asymptomatic patient. Plane cine fluoroscopy in figure 7of thorax showed massive calcification or myocardium especially of basal and mid segments of LV, not limited to any one coronary artery distribution. Coronary angiogram revealed normal coronaries as in Figure 7. At this stage patient refused further investigation and got discharged.

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Review of Literature

Finestone and Geschickter (1949) and Gore and Arons (1949) subdivided Myocardial calcification into three groups-*metastatic, dystrophic or an extension inwards from the pericardium*. Hypercalcaemia is an essential finding in the metastatic group, and calcium salts are deposited in normal myocardial tissues. The dystrophic calcification is related to the laying down of calcium salts in dead, degenerated or devitalized myocardial tissue.

Patterson *et al.,* (1953), reported a case of Idiopathic myocardial calcification, however rheumatic origin was suspected. The patient had failure with AF and episode of thromboembolism. Macieira-Coelho *et al.,* (1975) reported a similar case in 1975.

Recently Adel *et al.,* (2005), reported a case of myocardial calcification with normal LV ejection fraction and normal coronaries. But patient was symptomatic with evidence of CCF with valvular calcification (MAC) and moderate MR. CT and MRI were used to assess the myocardial calcification, which revealed involvement of mitral annulus and pericardium.

Brit *et al.,* (2011) reported patient with CCF and arrhythmia. CT and MRI revealed involvement of Myocardium, mitral annulus, papillary muscles and pericardium. CT also showed pulmonary nodules indicating healed granulomas.

DISCUSSION

The deposition of calcium in myocardium, as noted, are due to *Dystrophic*, i.e., calcium salt deposition in dead or injured myocardium; *Metastatic,* i.e., deposition of calcium salt in normal myocardium due to disturbance in Calcium Phosphorus axis.

Metastatic calcification is related to Hypercalcaemia resulting from CRF, destructive bone lesion or hyperparathyroidism. These patients have calcification of cardiac, renal and vascular system.

In these cases it was not possible to find a plausible explanation for Calcium deposition in myocardium.

Pomerance *et al.,* (1970) described mitral ring calcification with involvement of adjoining myocardium in elderly (age>70 yrs.) women.

If myocardium is involved, it is adjacent to valve. This is unlikely in this patient as the calcification was remote from the mitral valve ring as evidenced from Echocardiogram and CT scan.

Rheumatic fever leading to Myocarditis and later calcification has been described. Case of 11 yr. old boy with massive calcification of myocardium following bout of acute rheumatic fever was reported by Edelstein (1946).

In our cases, patients (a) were elderly (not in Rheumatic fever age group), (b) No history suggestive of rheumatic fever earlier in life, (c) No valvular involvement.

Therefore, Rheumatic etiology for myocardial calcification is remote possibility.

Parasitic infections of the heart are rarer causes of Myocarditis and calcification. Most of the parasitic infection present with chronic Myocarditis and conduction defect (Dilated cardiomyopathy) e.g., *Trypanosomiasis (Chagas' disease)* (Pomerance, 1970).

Amebic pericarditis (*E. histolytica)* is a rare but serious complication of liver abscess. Small series involving Greek patients with cardiac hydatid caused by *Echinococcus*species disease have been reported (Louis *et al.,* 2004).

But the resultant calcification, if any, will be nodular. Acute Myocarditis associated with tachyzoites (*Toxoplasma gondii),* focal inflammation and myonecrosis may accompany congenital toxoplasmosis or acute infection in adults (Leak and Meghji, 1979).

One case of extensive calcification of the right ventricle and interventricular septum has been reported in congenital toxoplasmosis.

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But in the cases reported, patient had no obvious evidence of any infection (though not serologically investigated), and had normal sinus rhythm ECG with no conduction defects.

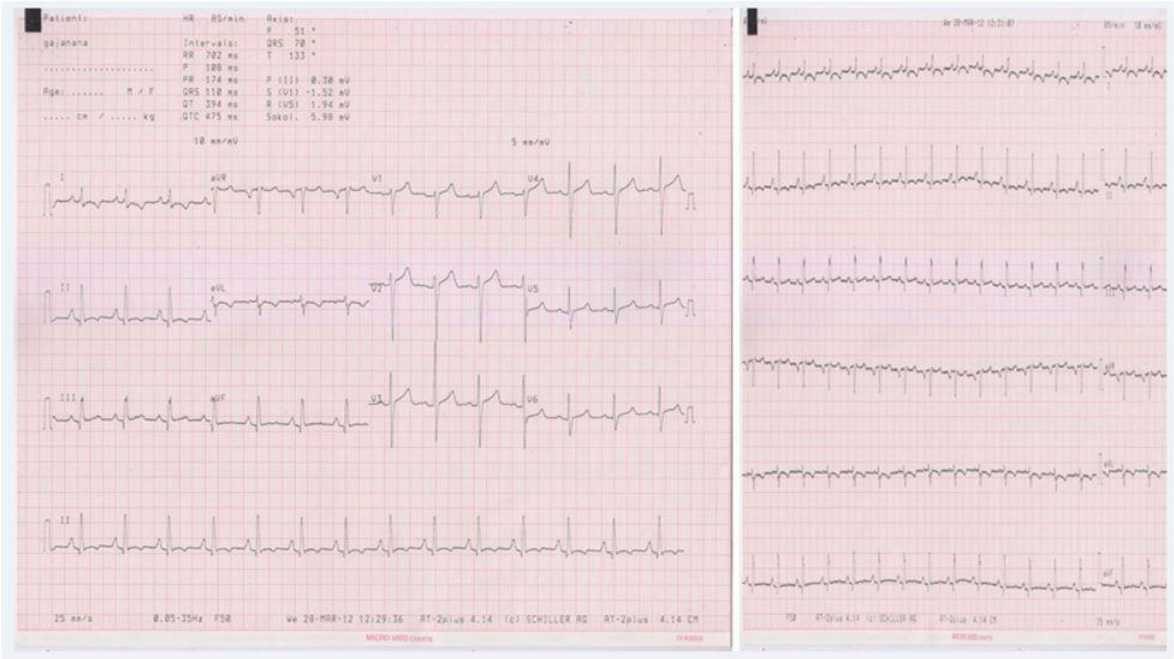

Figure 1

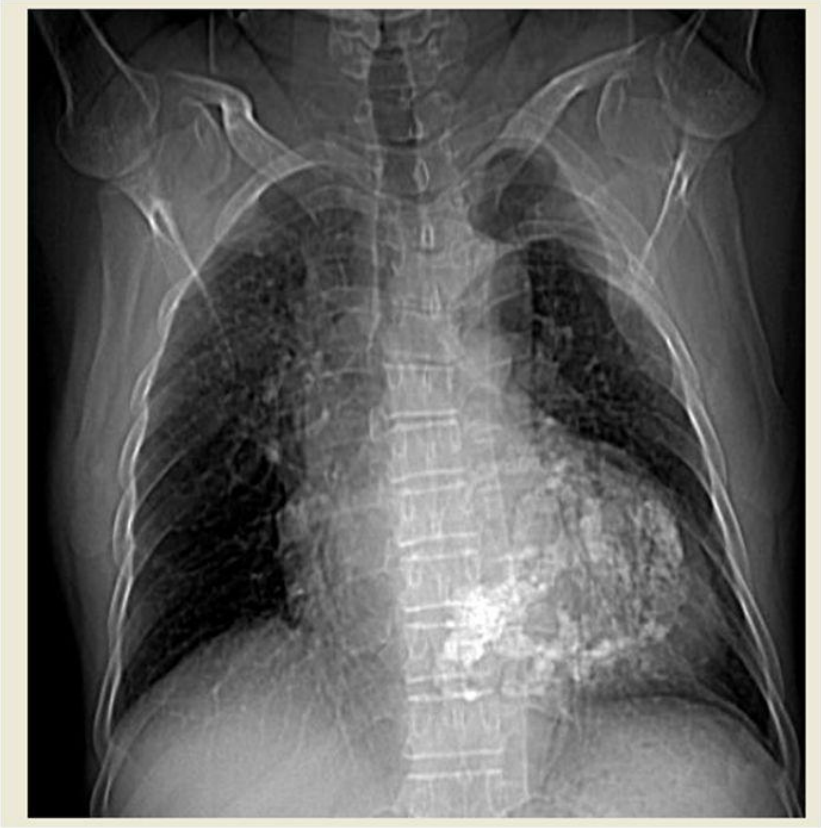

Figure 2: Chest x-ray PA view

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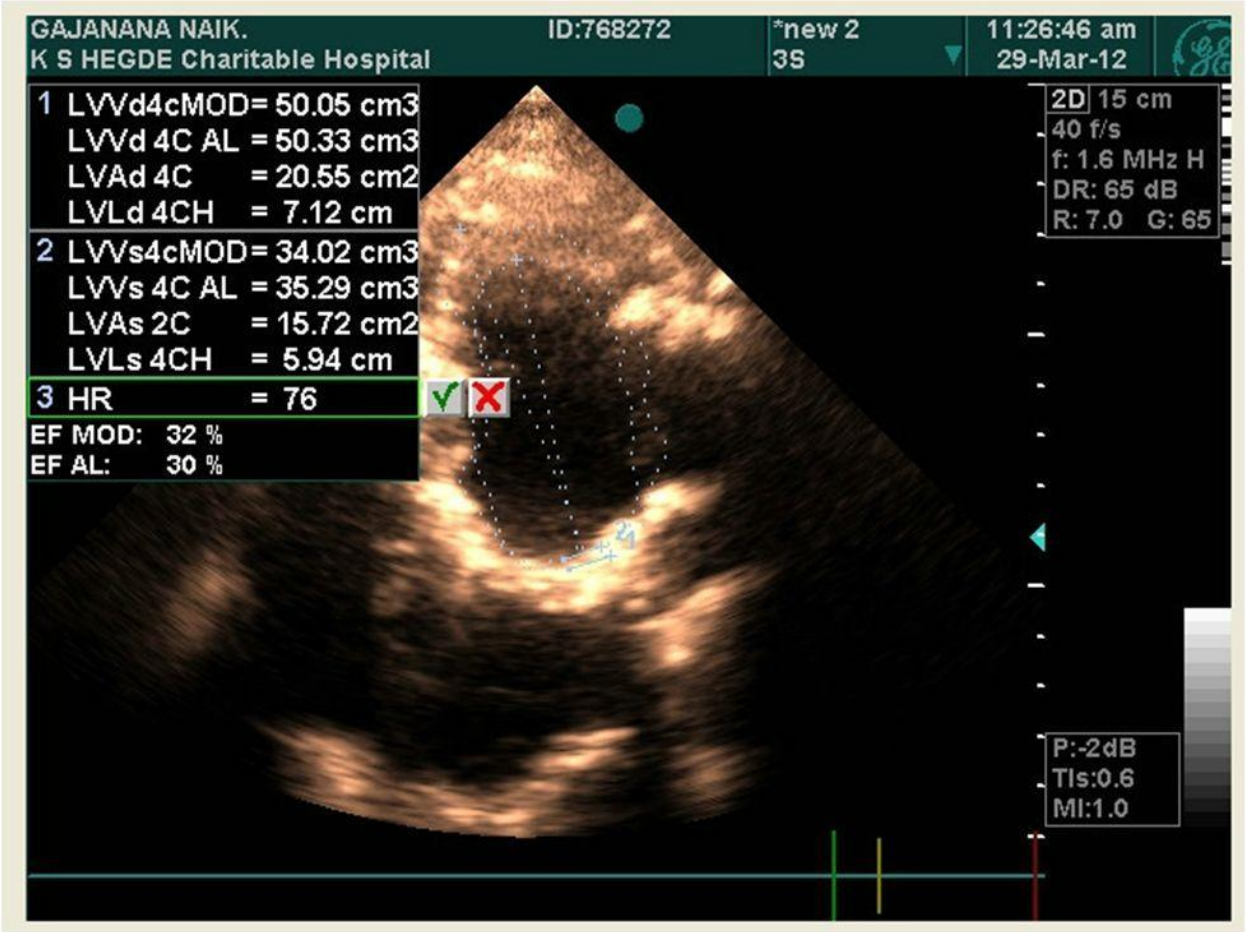

Figure 3: ECHO (EF calculation and chunks of Calcium)

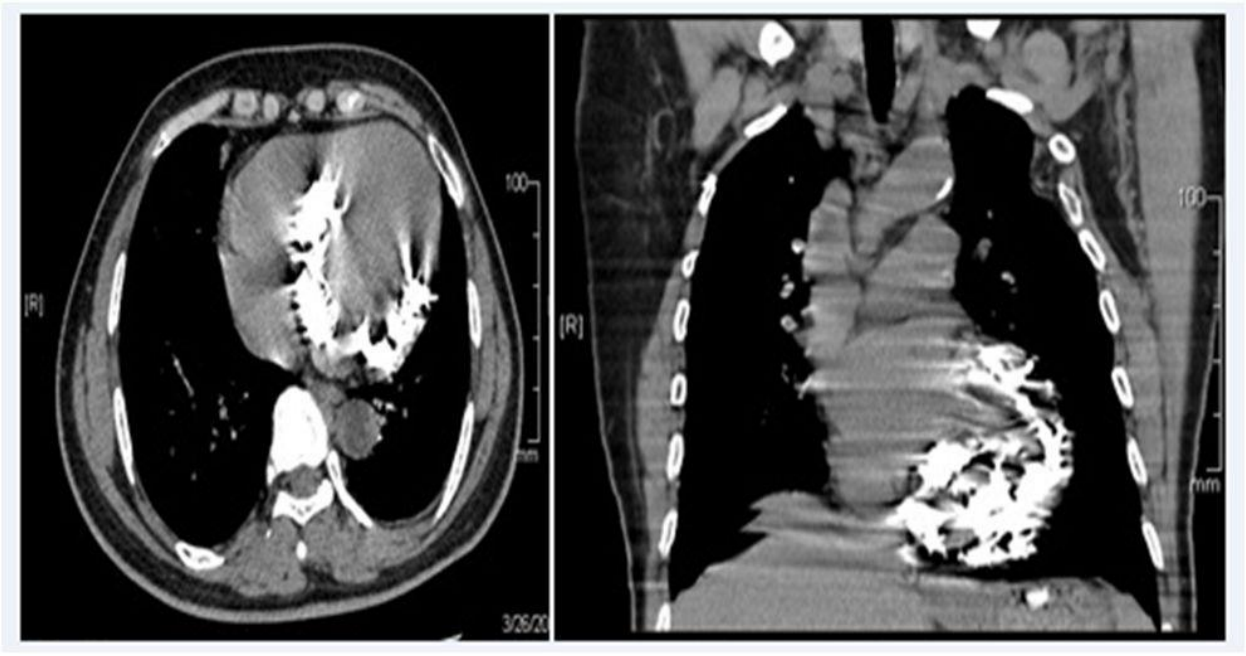

Figure 4: MRI

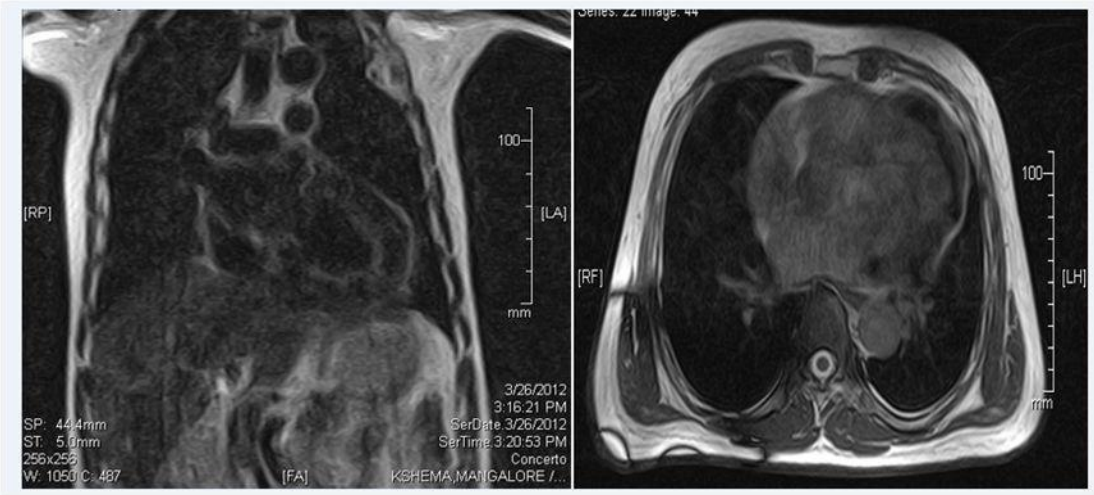

Figure 5: CT Scan

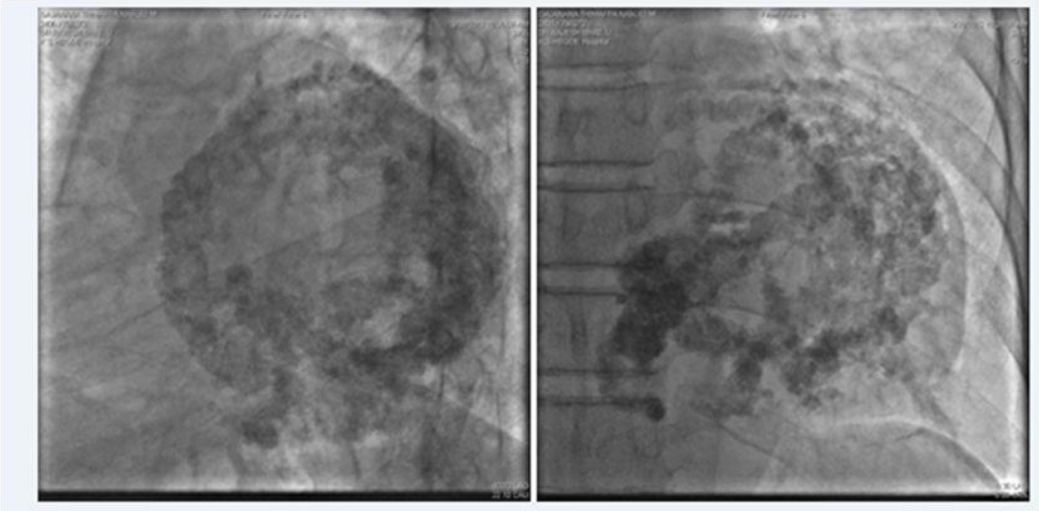

Figure 6a: Fluroscopy

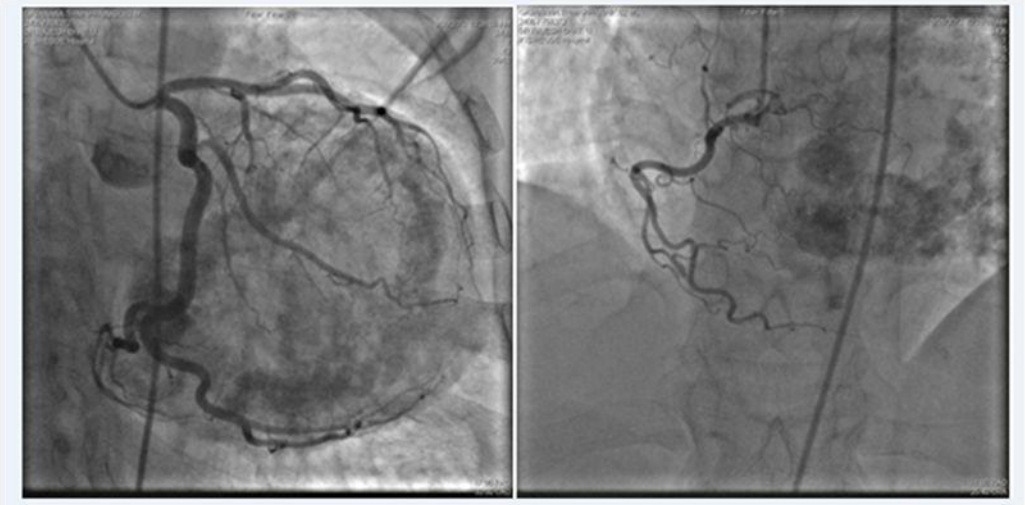

Figure 6b: Coronary

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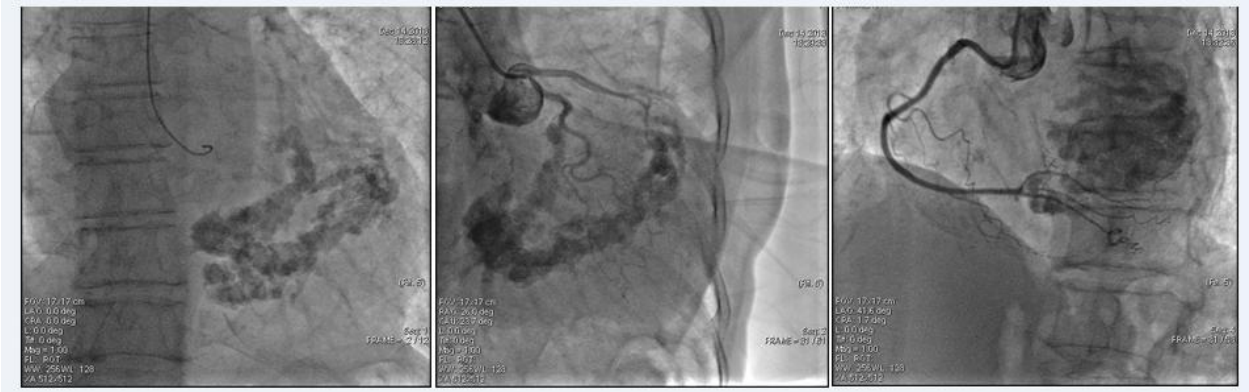

Figure 7: Fluroscopy left and right coronaries

Coronary artery disease is common cause of Myocardial calcification, especially in elderly age group. *Barnard et al.,* (1985) presented a 50-year-oId woman with massive myocardial calcification and normal coronary arteries. In the above presented case, though ECG had "q" waves in inferior leads, patient had no history of MI in past and coronary angiogram was normal.

Myocardial calcification is reported in granulomatous diseases such as Tuberculosis and Sarcoidosis ((Brit *et al.,* 2011). There was no hard evidence to support these diseases in present case.

Endomyocardial fibrosis causes endocardial fibrosis with later extension to myocardium and later, rarely, calcification (Brit *et al.,* 2011). These cases present with evidences of restrictive cardiomyopathy, pericardial effusion, valvular regurgitation and endocardial thrombus. The patients presented were (a) not from endemic area, (b) had no clinical risk factors for development for EMF, (c) Endocardium and Pericardium were not involved as evidenced by imaging studies.

Conclusion

Idiopathic Myocardial Calcification is a rare clinical occurrence. Many cases are reported in literature.

The cases reported in this report were unusual because, massive calcification developed with no obvious cause; patients were asymptomatic with no cardiac symptoms, ECG shows no rhythm disturbances and hemodynamic studies were within normal range.

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