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TYPE 2 MYOCARDIAL INFARCTION IN A 12-YEAR-OLD GIRL

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Abstract: Type 2 Myocardial infarction (MI) is caused by myocardial oxygen demand and supply mismatch. The etiology, risk factors, treatment and prognosis of Type 2 MI differs from Type 1. We are reporting a case a Type 2 MI in a young girl due to anemia caused by autoimmune hemolysis.

Introduction:

Myocardial infarction (MI) is classified into Type I and Type 2 based on pathophysiology by Fourth Universal Definition of myocardial infarction (UDMI) (1). Type 1 MI is caused by myocardial ischemia due to rupture of atherosclerotic plaque whereas Type 2 is caused by myocardial oxygen demand and supply mismatch. The etiology of Type 2 MI can be coronary cause like coronary spasm, coronary dissection or it can be non-coronary systemic cause like sepsis, anemia and arrythmias and hypotension. There is a wide variation in prevalence of Type 2 MI among all myocardial infarctions ranging from 2 to 58% (2). This is due to different cut off values of troponin used to define MI in different studies and difficult in differentiating this Type 2MI from other acute non ischemic myocardial injuries. Type 2 MI is common in female when compared to Type 1 and occurs in elderly age group with more comorbidities (3). The conventional risk factors for atherosclerotic Type I MI are also the additive factors for occurrence of Type 2 MI in acute illness (4). In the present case report, we are reporting a case of Type 2 MI in a young girl with no comorbidities.

Case report:

A 12-year-old female with no comorbidities presented with acute onset chest pain and fever for 5 hours. Physical examination was unremarkable except for pallor. Her vitals showed tachycardia at 110 beats per minute. 12 lead ECG showed ST elevation in leads II, III, avF with ST depression in V1 to V3 as shown in Fig 1.

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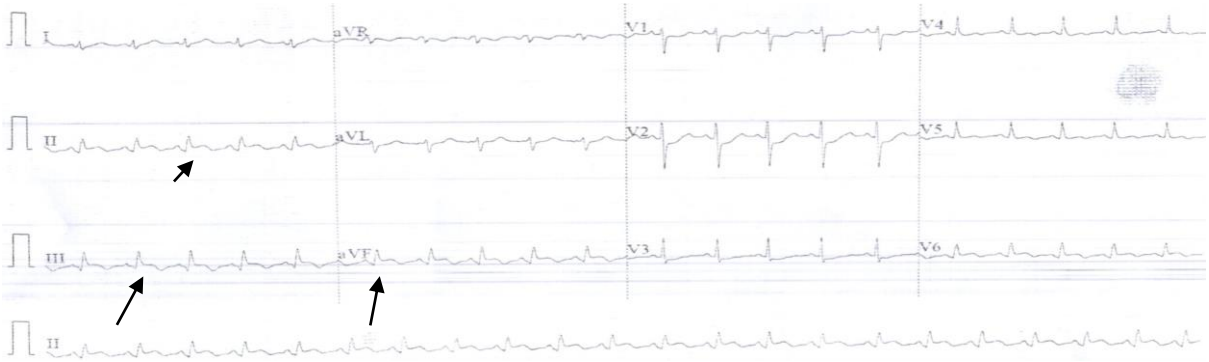

Fig 1 ECG showing ST elevation in leads II, III, avF as shown by black headed arrows.

Echocardiogram showed regional wall motion abnormality (RWMA) in inferior wall with ejection fraction of 40% and normal valvular function. High-sensitivity cardiac troponin (hs-cTn) was 27.4 ng/L(normal <14ng/L). Anginal pain with ST elevation in three consecutive leads on ECG and RWMA in echocardiogram along with elevated troponin(> 99th percentile), a diagnosis of MI was made based on UDMI. Blood investigations showed haemoglobin of 6 gm/dl, CRP(C-reactive protein) of 140 mg/dl, ESR of 150mm/hour. CTCA(CT coronary angiogram) revealed normal coronary origin and course with no stenosis as shown in Fig 2.

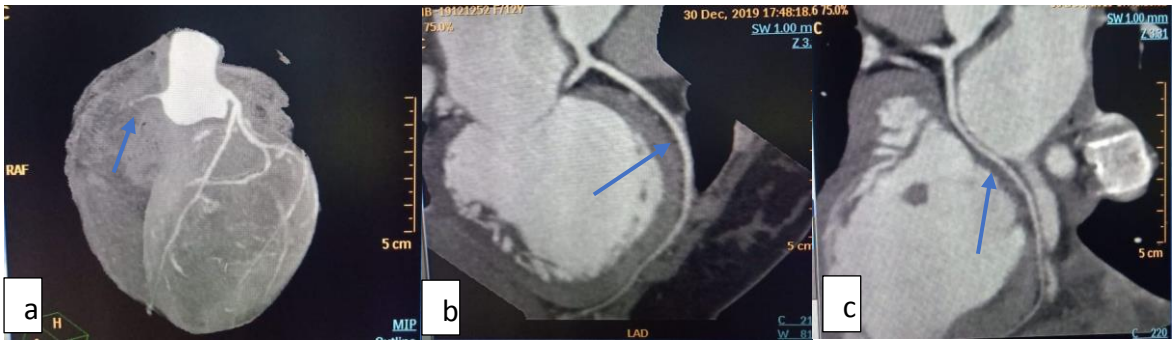

Fig 2: CT coronary angiogram showing non obstructed coronaries (a) arrow showing non dominant RCA ,(b) arrow showing normal(LAD), (c) showing normal (LCX) as indicated
RCA- Right coronary artery, LAD- Left anterior descending, LCX- Left circumflex artery.

Routine fever work up doesn't yield any positive results. So a diagnosis of Type 2 MI was made as criteria for MI were met with normal coronaries and anaemia as probable cause. The cause for anaemia was investigated in which the peripheral smear shows normocytic normochromic with anisopoikiocytosis and iron indices were within normal limits(serum iron- 103ug/ml, TIBC(Total Iron Binding Capacity)- 268 ug/ml, ferritin- 918ng/ml, Transferrin saturation- 38.4%). As the patient presents with fever and anaemia with anisopoikiocytosis on peripheral smear, haemolytic anaemia was suspected and Coombs test was done which was negative. She was treated conservatively with blood transfusion and with diuretics, beta blockers and ARBs(angiotensin receptor blockers) for LV dysfunction. She has improved symptomatically and haemoglobin increased to 9gm/dl. Cardiac MRI after

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2 weeks shows transmural scar in inferior wall as evidenced by LGE(Late Gadolinium Enhancement) transmurally Fig 3.

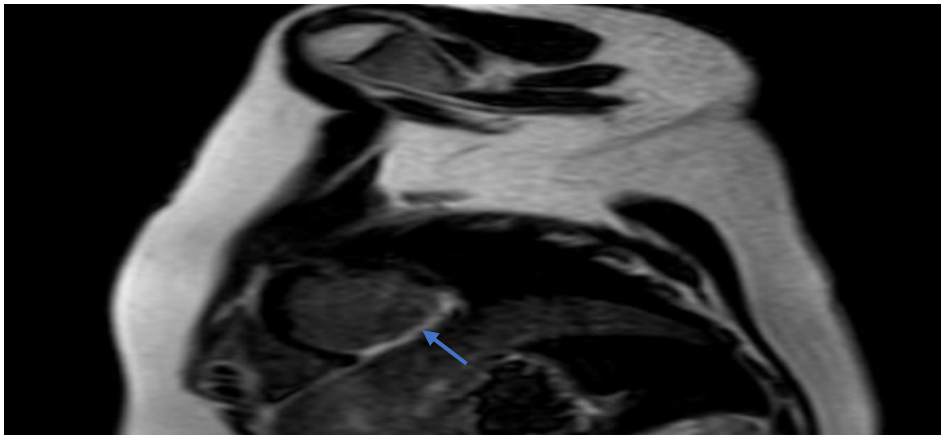

Fig 3:Cardiac MRI showing LGE enhancement in inferior wall transmurally as indicated by arrow suggestive of scar due to infarction. LGE- Late Gadolinium Enhancement.

This pattern of LGE scar is consistent with infarction ruling out myocarditis and other cardiomyopathies. Patient was discharged and follow up. Three from discharge she again presented with an episode of fever and drop in haemoglobin(Hb) but this time with elevated serum creatinine also. Recurrent episodes of fever and drop in Hb, elevated CRP and ESR now with raise in creatinine, autoimmune disorder with hemolysis was suspected. Coombs test was repeated which shows strong positive. Immunological profile showed positive for APLA (antiphospholipid antibody) and ANA (antinuclear antibody) with low complement (C3, C4) levels. Kidney biopsy is consistent with lupus nephritis pattern.

She was treated with steroids and cyclophosphamide (7 doses). Then clinically improved, became afebrile, Hb was corrected and creatinine normalized. LV function remained at 40 % and cardiac medications were being continued. No further episodes of hemolysis and she was being follow up for one year.

Discussion:

The present case highlights the importance of identifying the cause of myocardial injury as myocardial ischemia in a low-risk patient for myocardial infarction and in establishing the cause for Type 2 MI. In contrast to previous studies where Type 2 MI is more common in elderly our case is younger individual. Regarding the causes of Type 2 MI, anemia is the most common cause (3) similar to the present case. Patients with Type 2 MI less likely will show changes in ECG and hypokinesia on echocardiogram when compared to Type 1 MI (5). This is contrast to our case where ECG showing ST elevation and echocardiogram showing RWMA. With elevated troponin in any case the Type 1 MI should be ruled out by the absence of atherosclerotic plaque (2), which was done in our study by CTCA. In patients with suspected myocardial infarction but with no plaque in coronaries, myocarditis and cardiomyopathies has to be ruled by cardiac MRI (6,7) before labelling as Type 2 MI as the pattern of LGE varies between these conditions as shown in our case (8). Acute myocardial infarction can be a first manifestation of Antiphospholipid antibody syndrome in young

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adults (9). The pathology being the thrombotic occlusion of coronaries due to hypercoagulable state. But in the present case the coronaries were normal on CTCA, although the limitation was that we have not performed invasive coronary angiogram with intravascular imaging. So rather than thrombotic occlusion the myocardial injury is due to demand supply mismatch caused by acute hemolytic anemia due to autoimmune disorder casing Type 2 MI. Regarding the treatment of Type 2 MI the therapy should be individualized based on the specific conditions causing the myocardial injury like anemia and autoimmune disorder in our patient (10).

Conclusion:

Type 2 MI should be considered in young individuals with evidence of myocardial injury and normal coronaries. The precipitating cause should be sought and treatment should be tailored accordingly.

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