

Myocardial Infarction on the Background of Juvenile Idiopathic Arthritis

Akhtar S*, Khan A and Khan MI

Department of Cardiology, Khyber Teaching Hospital (KTH), Peshawar, Pakistan

*Corresponding author:

Saddaf Akhtar,
Department of Cardiology, Khyber Teaching
Hospital (KTH), Peshawar, Pakistan,
E-mail: saddafakhtar@ymail.com

Received: 05 Mar 2021

Accepted: 24 Mar 2021

Published: 30 Mar 2021

Copyright:

©2021 Akhtar S, Khan A, Khan MI. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Citation:

Akhtar S, Khan A, Khan MI. Myocardial Infarction on the Background of Juvenile Idiopathic Arthritis. *Ann Clin Med Case Rep.* 2021; V6(9): 1-3.

Keywords:

Juvenile Idiopathic Arthritis; Myocardial Infarction; Cardiovascular Diseases Risk

1. Abstract

Juvenile idiopathic arthritis (JIA) is the most common chronic inflammatory arthritis under 16 years of age but unlike rheumatoid arthritis, the risk of cardiovascular diseases including Myocardial Infarction (MI) in adulthood with JIA is not yet proven. This young lady with a background of JIA that progressed to adulthood was diagnosed with inferior wall MI despite no known cardiovascular risk factors. The patient was diagnosed well in time and was successfully thrombolysed with streptokinase.

2. Introduction

The most common autoimmune chronic inflammatory arthritis among children and adolescents under age 16 is Juvenile Idiopathic Arthritis (JIA) [1]. 40-50% of JIA progress to adulthood with an active disease [2].

Although increased risk of cardiovascular diseases (48%) has been demonstrated in adults with rheumatoid arthritis [3], a little has been known whether JIA predisposes adults to cardiovascular diseases despite the same pathogenesis of both the diseases⁴. No increase in cardiovascular diseases has been found in patients previously diagnosed with JIA when followed into adulthood till the age of 30 years [1].

Owing to the considerable interest in JIA predisposing adults to cardiovascular diseases, the case presented below is like a spark in the dark as this young lady had an MI without any known risk factors for MI.

3. Case History

This is a story of 24 years old girl who presented to CCU in the evening with 3 hours history of severe crushing central chest pain <http://acmcasereports.com/>

radiating to her left arm. The pain was associated with nausea, vomiting and profuse sweating. Serial ECGs were done that showed evolving ST elevation up to 5 mm in limb leads II, III and aVF (Figure 1). She was admitted to CCU with Acute Inferior wall myocardial infarction.

This young lady was previously diagnosed with juvenile idiopathic arthritis at the age of 12 years but was not on any regular treatment. For the last 1 year she was started on tapering dose of oral steroids as her arthritis got worsened. Besides that she had no significant past medical or surgical history. Her family history is also unremarkable.

On examination of the patient, she had developed cushingoid facies. Vitally she was stable with a BP of 130/80 mm Hg and pulse of 68 bpm. She was maintaining saturation of 96% at room air. There were no visible joint deformities and no signs of acute inflammation.

Baseline investigations showed a raised troponin level of 25 ng/ml (Normal: <0.6 ng/ml), raised ESR of 50 mm/1st hr. (Normal: 0-20 mm/1st hr.) and raised quantitative C - reactive protein (CRP) to 53.06 mg/L (Normal: <5.0 mg/L). Quantitative rheumatoid factor (RF) was 16 IU/ml (Normal: <14 IU/ml). Fasting Lipid profile, blood glucose level and liver and kidney function tests were all normal. 2D echocardiogram was obtained the report of which is given in (Figure 2).

She was loaded with 300 mg of aspirin and 300 mg clopidogrel per oral. 60 mg of low molecular weight heparin (LMWH) was given subcutaneously. After informed consent she was thrombolysed with 1.5 million IU of streptokinase (SK) diluted in 100 ml normal

saline over 45 min. Cardiac monitor was attached and she was observed closely for any complication. Post SK ECG showed successful thrombolysis and patient responded clinically as well. She was discharged after 24 hours of observation in CCU (Figure 3).

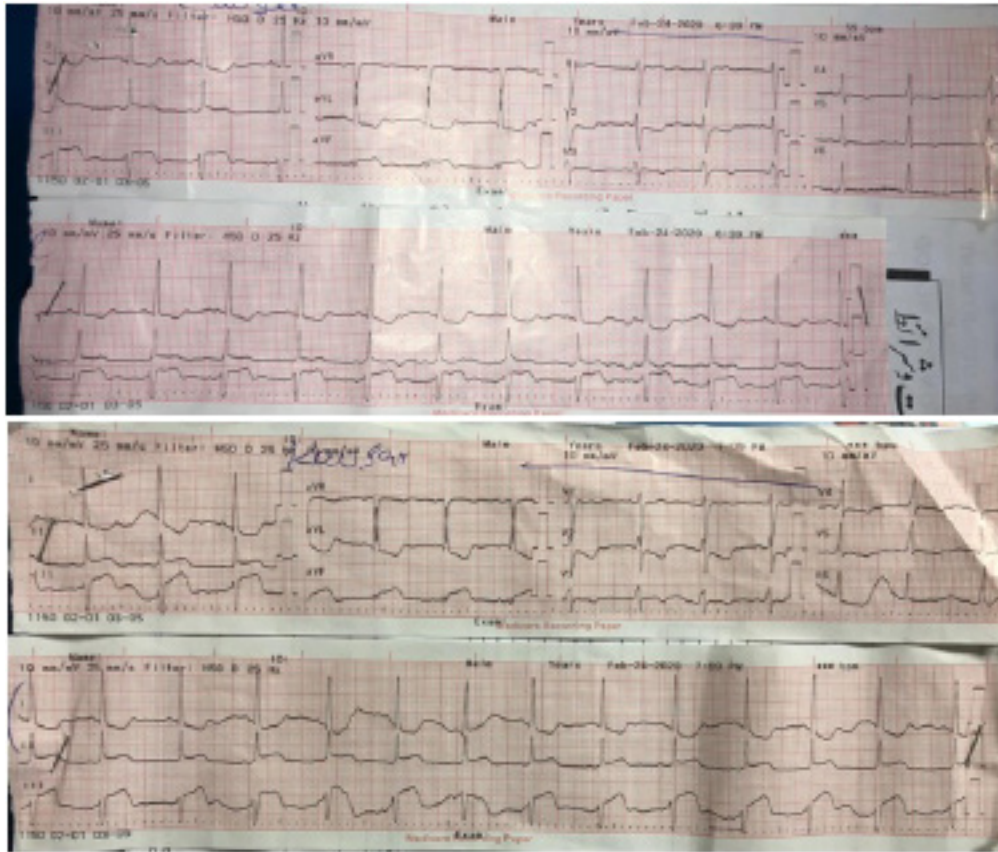


Figure 1

| DIMENSIONS | In cm | NORMAL | DIMENSIONS | In cm | NORMAL |
|----------------|-------|------------|-----------------------|-------|------------------------|
| Left Ventricle | | | Aortic Root (ED) | 3.0 | 2.0-3.7 cm |
| Diastole | 5.3 | 3.7-5.9cm | Left Atrium (ES) | 3.7 | 1.9-4.0 cm |
| Systole | 3.9 | 1.8-4.2 cm | Right Ventricle (Dia) | 2.0 | 0.7-2.8 cm |
| IVS (D) | 1.0 | 0.6-1.2 cm | | | |
| LVPW (D) | 0.9 | 0.6-1.2 cm | | | |
| FS | 26% | >28% | MVA (2D) | | 4.0-6.0cm ² |
| LVEF (est) | 51% | ≥51% | MVA (Doppler) | | |

2D& M-MODE Comments

- Aortic root is normal.
- Aortic valve is normal. The valve is tricuspid. There is normal mobility.
- Mitral valve is normal in mobility and thickness.
- There was no mitral annular calcification.
- Tricuspid valve is well visualized and is normal.
- Pulmonic valve is well visualized and is normal.
- Left ventricular dimensions show normal chamber size. LV wall thickness is normal.
- **There is mid and basal inferior akinesia with inferoseptal dyskinesia.**
- Right ventricular dimensions show normal chamber size. RV wall thickness is normal.
- There is normal right ventricular contractility.
- Left atrial size is normal. Right atrial size is normal.
- There is no pericardial effusion. No intra cardiac vegetation seen.
- IVC was normal with respiratory variation.
- No intra cardiac shunt seen. No intra cardiac mass seen.

COLOR FLOW AND DOPPLER WAVEFORM ANALYSIS

- Aortic systolic flow pattern was normal and there was no regurgitation noted.
- Mitral diastolic flow pattern was not reversed and there was trace regurgitation noted.
- Tricuspid diastolic flow pattern was normal and there was trace regurgitation noted.

IMPRESSION:

- REGIONAL WALL MOTION ABNORMALITY.
- FAIR LV SYSTOLIC FUNCTION.

Figure 2

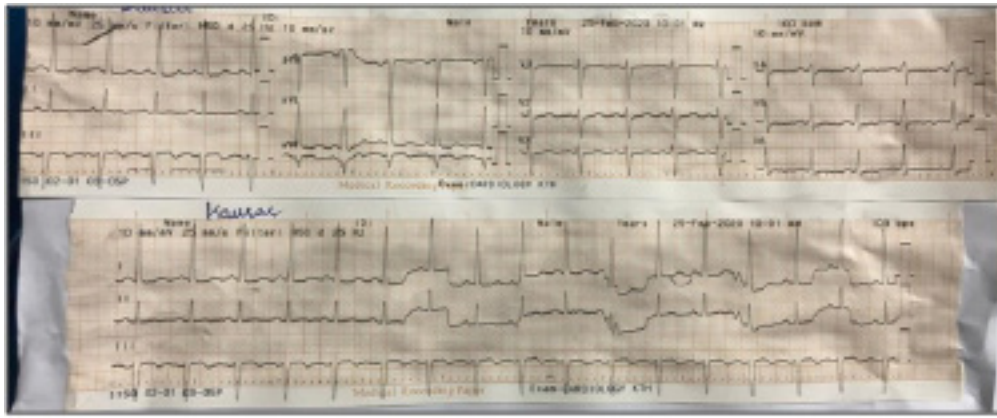


Figure 3

4. Discussion

The risk of MI in adulthood with JIA is the same as in general population [1]. This case is presented here because no cases has been reported till date to show increased risk of MI in patients with JIA who have no known cardiovascular risk factor. Multicenter studies have been conducted to see the relation which has remained statistically insignificant. Researchers are very keen to uncover this relationship between MI and JIA which offers a lot of work to be done in this area of medicine.

References

1. Jason H. Anderson, Katelyn R. Anderson, Hanne A. Aulie, et al. Juvenile Idiopathic Arthritis and Future Risk For Cardiovascular Disease: A Multicenter Study. *Scand J Rheumatol.* 2016 Jul; 45(4): 299-303.
2. Selvaag AM, Aulie HA, Lilleby V, Flato B. Disease progression into adulthood and predictors of long-term active disease in juvenile idiopathic arthritis. *Ann Rheum Dis.* 2016 Jan; 75(1): 190-5.
3. Avina-Zubieta JA, Thomas J, Sadatsafavi M, Lehman AJ, Lacaille D. Risk of incident of cardiovascular events in patients with rheumatoid arthritis: a meta-analysis of observational studies. *Ann Rheum Dis.* 2012; 71(9): 1524-9.
4. Coulson EJ, Ng WF, Goff I, Foster HE. Cardiovascular risk in juvenile idiopathic arthritis. *Rheumatology (Oxford).* 2013; 52(7): 1163-71.