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 diseases [4]. 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 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).
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