A 20 years old previously healthy male presented to our emergency department with acute chest pain of 4 hours duration. His past medical history was unremarkable and he was not a smoker. There was no family history of coronary heart disease. He had a history of recent respiratory infection for which he took over the counter medication. He reported that the pain radiated to his left shoulder, worsened with deep inspiration and lying supine and improved with leaning forward. No dyspnea or cough was reported. His vital signs were reported as blood pressure of 105/70 mmHg, heart rate of 90 beats per minute, body temperature of 37.4 ℃ and respiratory rate of 20 per minute. He was diaphoretic and in obvious discomfort. On cardiac auscultation a high-pitched scratchy friction rub was present best heard at left lower sternal border in end expiration. A 12 lead Electrocardiogram (ECG) showed normal sinus rhythm with diffuse concave ST segment elevation with ST depression in V1 and avR (Panel A). PR segment depression was also present best seen in V3 and V4. Ratio of ST-segment elevation to T-wave amplitude was 0.33 in lead V_e. Cardiac enzymes were negative. On routine lab tests C-reactive protein was 4 mg/L (reference level between 1-3 mg/L) and white blood cell count was 13500/mm³. Echocardiography in emergency department showed normal left ventricular function and there was no regional wall motion abnormality. Pericardial effusion was not present. ECG feature were typical of stage I of acute pericarditis. The patient was diagnosed with acute pericarditis based on clinical presentation and ECG findings and ibuprofen 400 mg QID was initiated which lead to marked improvement of symptoms. Electrocardiogram one week later showed marked resolution of ST segment elevations (Panel B). An electrocardiogram 3 weeks later showed T inversions in inferior leads and in V4-V6 (Panel C). ECG 3 months later showed normalization of T waves (Panel D).

Acute pericarditis is among the differential diagnoses of patients presenting with acute chest pain to emergency department and ECG remains and important diagnostic modality during the acute phase of clinical presentation. The evolution of electrocardiographic manifestations of acute pericarditis has been described through four phases based on ST-segment and T wave changes [1,2]. Stage I is most diagnostic and diffuse ST-segment elevation and PR-segment depression and can last a few hours to several days [3]. Stage II is normalization of the ST and PR segments, and sometimes one or more leads lead or lag the others. Stage III is widespread T-wave inversions and Stage IV is normalization of the T waves with usually complete resolution of ECG changes [1,2]. Orderly progress through stages has been regarded as characteristic and pathognomonic for acute pericarditis. Some stages may be absent if the process resolves too rapidly [2]. No reciprocal changes, Q waves or PR depression are found in the 12-lead ECG during acute pericarditis [3]. The most reliable distinguishing feature between acute pericarditis and ST elevation myocardial infarction may be the ratio of ST-segment elevation to T-wave amplitude in lead V_e. When this ratio exceeds 0.24, acute pericarditis is almost always present [1].

The patient had a benign course and recovered uneventfully. In view of the uncomplicated course of acute pericarditis following an upper respiratory tract infection which responded to NSAIDs the diagnosis of viral pericarditis was made.

References

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