Tuberculous Myocarditis

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38.1 Clinical History

Hypertension was detected in a young woman when she developed a cerebrovascular accident at the age of 38 years. She recovered and was on antihypertensives. Two years later, she developed sudden-onset headache followed by altered sensorium and was admitted in our emergency services department. The general condition was poor, the pulse rate was 76 per minute, and blood pressure was 170/100 mmHg. She was unconscious with nonreacting pupils and downgoing plantar reflexes. Rest of the systemic examination had been normal. Her routine hematological and biochemical investigations had been normal. The ECG (Fig. 38.1) showed abnormal inferior Q waves, borderline T wave abnormality, borderline prolonged QT interval, and baseline wander in leads V1, V3 and V4, while intraventricular hemorrhage was seen on computed tomographic scan. She was given antihypertensives, anticonvulsants, and intracranial tension lowering agents and antibiotics, but expired after 12 h.

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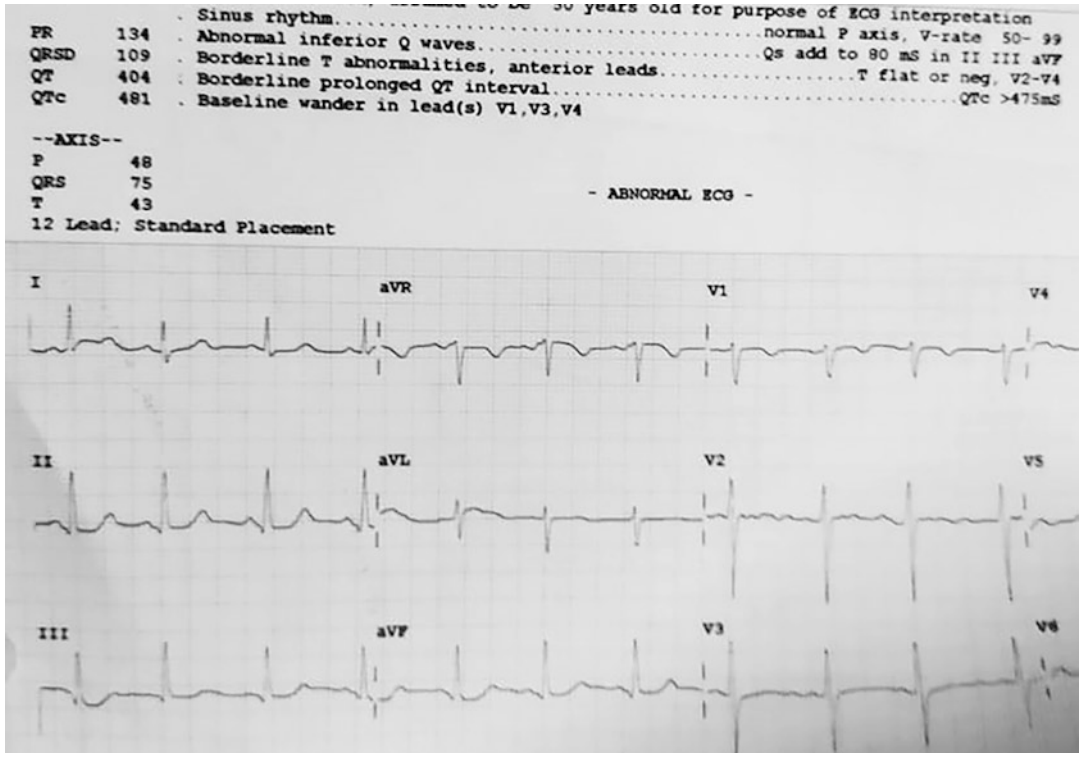

Fig. 38.1 Abnormal ECG tracing

38.2 Autopsy Findings

The heart (290 g) was moderately enlarged and globular due to biventricular enlargement. The heart also felt a little flabby and transverse sections revealed biventricular dilatation with myocardial thinning and few pale yellowish white areas in the subepicardial region (Fig. 38.2a). On histology, most of the sections of the right and left ventricular myocardial revealed caseating epithelioid granulomas in the interstitium (Fig. 38.2b, c); confluence of granulomas had produced the opalescent subepicardial foci (Fig. 38.2d, e). Lymphocytic myocarditis-like areas (Fig. 38.3a) were seen in the other parts of the myocardium with associated endocardial inflammation and fibrosis (Fig. 38.3a, b). All coronary arteries were patent. Rest of the chambers and valves were normal. Other findings were hypertensive intraventricular hemorrhage, Hashimoto's thyroiditis, mediastinal tuberculous lymphadenitis, pulmonary edema with bronchopneumonia, hepatic steatosis, and acute renal tubular necroses.

Cause of Death: Raised intracranial pressure due to intraventricular hemorrhage.

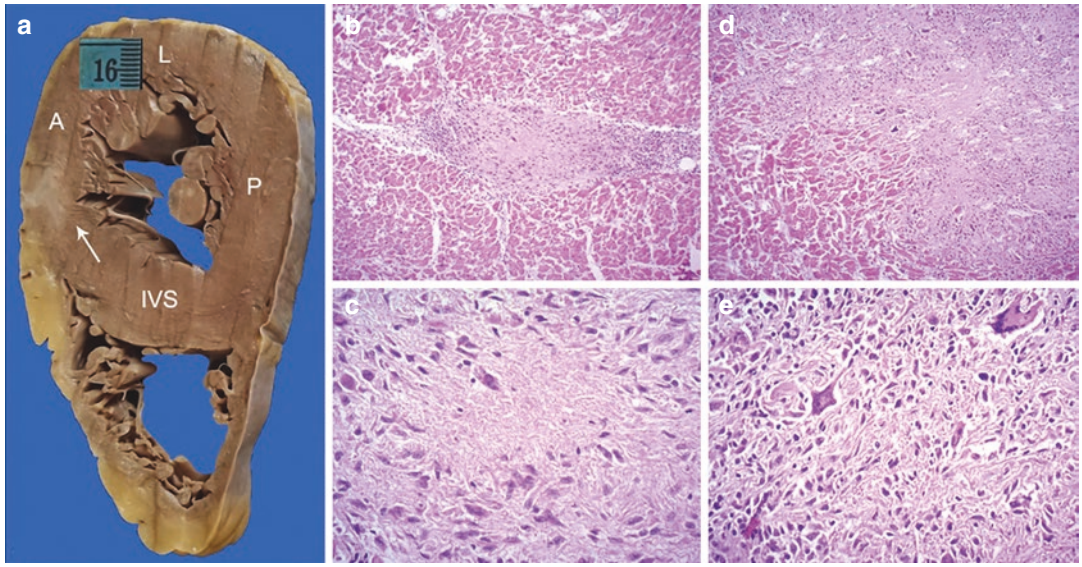

Fig. 38.2 (**a**) The transverse section forming the mid-portion of the ventricles shows a subepicardial irregular pale grey appearance of the myocardium. The interstitium shows a large caseating granuloma amidst the myofiber groups (**b**) H&E × 200 and (**c**) H&E × 400; The subepicardial focus was composed of confluent granulomas (**d**) H&E × 200 and (**e**) H&E × 400

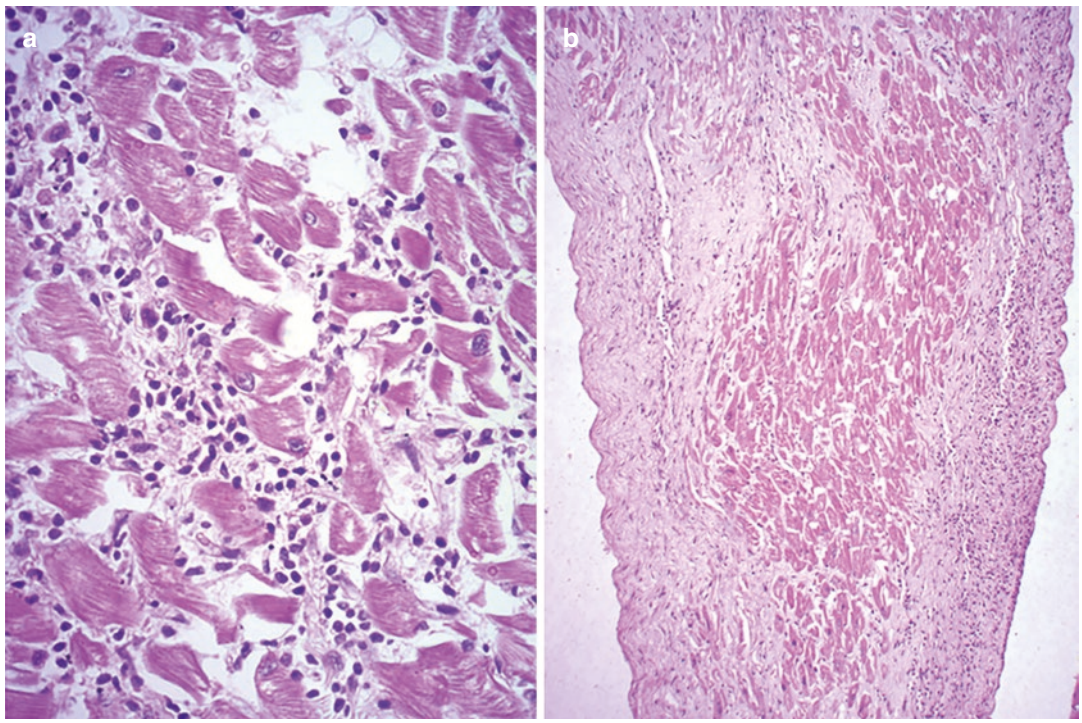

Fig. 38.3 (**a**) Dense lymphocytic clusters were seen in the interstitium (H&E × 400); (**b**) The endocardium over the trabecula was thickened with focal lymphocytic infiltration (H&E × 200)

38.3 Discussion

The ECG abnormalities recorded in this young woman were produced by tuberculous myocarditis, which is an uncommon manifestation of cardiovascular tuberculosis. Tuberculosis, which is still a major health problem in several countries, is generally said to spare the thyroid, heart, pancreas, and skeletal muscle. However, the heart is involved in around 1–2% of patients with tuberculosis and is primarily a disease of the pericardium (See Chap. 2). Affliction of the other layers of the heart (myocardium and endocardium), coronary arteries (See Chap. 66), or aorta (See Chap. 65) is exceedingly rare. The reported prevalence of tuberculous myocarditis ranges from 0.14 to 2% and affects the young population (<45 years of age), especially men. Many such patients have weak immune status produced by diseases, age, and even prevailing poor socioeconomic conditions. Active myocardial contraction and generation of lactic acid are said to be the factors for relative sparing of the myocardium. The inflammation generally occurs in conjunction with pericardial disease or may be associated with pulmonary or extrapulmonary tuberculous foci through retrograde lymphatic spread, hematogenous dissemination, or direct extension. Isolated myocardial disease is distinctly unusual. Though the right-sided chambers are collectively said to be the most vulnerable due to their proximity to the right-sided mediastinal lymph nodes (most frequently involved by tuberculosis), the left ventricle is commonly affected. Involvement of the mediastinal lymph nodes was present in the case presented; there was no active inflammation in any other organ at autopsy. Much of the granulomatous inflammation was present in the left ventricular myocardium.

Morphologically, the myocardial lesions may be nodular, miliary, or of the diffuse infiltrative type. The nodular pattern is the most common form, where the myocardium shows small or large (‘pea to egg’ sized) granulomatous reaction with caseation necrosis; the largest nodules (tuberculoma) are described in the right atrium. This is followed by the miliary lesions that usually develop in the context of miliary tuberculosis. The rarest is the diffuse pattern that is characterized by interstitial infiltration of lymphocytes and giant cells. Certain stringent criteria had been introduced for the diagnosis of tuberculous myocarditis, which included major [positive polymerase chain reaction (PCR), positive myocardial culture, or demonstration of acid-fast bacilli in the myocardial tissues] and minor [myocardial granulomas, myocardial imaging abnormalities, and involvement of extracardiac tissues] criteria. Diagnosis of myocardial tuberculosis is justified if there is presence of 2 or more major criteria or a major and 2 or more minor criteria. It should be noted that confirmatory tests, including the PCR, can often be inconclusive or may also not be available at all centers due to technical restraints and financial constraints. Hence, presence of the characteristic caseating granulomas in the myocardium should be considered as a major criterion. The acid-fast staining did not demonstrate mycobacteria in the myocardium, but was seen in the tuberculous lymphadenitis. Many patients, despite extensive involvement, can remain asymptomatic and the disease is diagnosed at autopsy, particularly with sudden death. Others present with arrhythmias (atrial fibrillation, paroxysmal ventricular tachycardia or long QT syndrome), atrioventricular block, valvular dysfunction, right-sided inflow or outflow tract obstructions, and congestive cardiac failure; the latter is seen with diffuse involvement and has decreased left ventricular ejection fraction. Magnetic resonance imaging is useful to recognize these lesions and should be coupled with other investigative procedures for confirmation of tuberculosis. Antituberculous, antiarrhythmic, and antifailure drugs result in significant amelioration. Though rare, one must be aware of this unusual presentation of tuberculosis, particularly in young patients with unexplained ECG abnormalities or cardiac failure, and making an early diagnosis will improve the prognosis and reduce mortality.

Further Reading

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