The supernormal period of conduction occurs at the end of the T wave. This period is related to the recovery of excitability of the Purkinje fibers, during which time there is a dip in their threshold of excitation. This phenomenon does not occur in ventricular muscular cells, but local nonpropagated potentials occurring in ventricular muscle may be conducted if they occur during the period of the dip in the strength-duration curve of the Purkinje fibers. Such local nonpropagated ventricular potentials have been shown to occur in response to subthreshold stimuli in canine myocardium.

Our patient was subjected to rapidly repetitive, low-intensity electrical stimuli which were manifested in ventricular premature beats developing into runs of ventricular tachycardia. The coupling interval varied slightly, and its relationship to the length of the preceding cycle suggested that this interval represented the period of supernormal conduction, increasing with increasing precycle length as the duration of the action potential increased but reaching a plateau at longer cycle lengths. This probably indicates the maximum duration of the action potential in the patient.

The episodes of ventricular tachycardia may represent facilitation of conduction from the ectopic source, due to an increasing number of available periods of supernormal conduction or to spontaneous changes in the threshold of ventricular activation. It is noteworthy that the rate varied during episodes of ventricular tachycardia. This may be due to spontaneous changes in the threshold of ventricular activation caused by partial fluctuating depolarizations of the myocardium adjacent to the rapidly stimulating electrode. Alternatively, there may be changes in exit block through the junction of ventricular muscle and Purkinje fiber, due to continuous varying local depolarizations from the ectopic focus, analogous to the mechanism of concealed conduction through the atrioventricular mode in patients with atrial fibrillation.

The tachycardia was probably not due to a mechanism for reentry, because the intensity of stimulation for the first beat was low. Pacemaker-induced ventricular reentry usually follows high-intensity stimulation. It is unlikely that the tachyarrhythmia was due to atrial fibrillation with aberrant ventricular response. The morphologic appearance of the tachycardia was identical to that of normally paced beats, both in previous ECGs and after replacement of the generator; furthermore, the arrhythmia disappeared abruptly after disconnection of the faulty generator.

References

Sarcoidosis Initially Occurring as Apical Infiltrate and Pleural Reaction*

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A 21-year-old black man had pleuritic pain as the initial symptom of his sarcoidosis. Chest roentgenographic examination showed an infiltrate in the left upper lobe and a left pleural thickening. Sarcoidosis was diagnosed by pulmonary biopsy. Thus, sarcoidosis may closely mimic tuberculosis by initially occurring as predominantly apical infiltrates and pleural reaction.

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Pleural involvement and pulmonary infiltrates involving the upper pulmonary fields in patients with sarcoidosis are rare. When these complications do occur, it is usually in association with late fibrotic parenchymal disease. In contrast, we recently saw a patient whose sarcoidosis initially occurred as a pleural reaction and apical infiltrate. Because of the likelihood of confusion with tuberculosis, it is important for the clinician to be aware of this manifestation of sarcoidosis.

Case Report

A 21-year-old black man was referred to our center in July 1975 with a presumptive diagnosis of pulmonary and pleural tuberculosis. His illness had begun five months earlier, with the acute onset of left-sided pleuritic pain. The patient had also noted low-grade fever with associated night sweats and a 9-kg (20-lb) loss of weight over the preceding two months. A chest x-ray film in May had shown a left apical infiltrate and pleural reaction at the left base. The patient's chest x-ray films from two years ago were normal.

On admission to our hospital, the patient underwent a physical examination, which yielded normal findings except for changes compatible with a left-sided pleural reaction. The chest x-ray film showed an infiltrative process involving primarily the left upper lobe (Fig 1). Pleural reaction was noted at the left base, but no free pleural fluid was demonstrated on chest x-ray films taken with the patient in the decubitus position.

The following laboratory studies yielded abnormal results: serum alkaline phosphatase level, 340 international units (IU) (normal, 9 to 35 IU); serum glutamic-oxaloacetic transaminase level, 61 IU (normal, less than 30 IU); and serum level of globulins, 5.1 gm/100 ml. The cutaneous tests with tuberculin were negative through second-strength purified protein derivative of tuberculin (250 tuberculin units), but the cutaneous tests with trichophyton showed 10 mm of induration. The cutaneous test with coccidioidin was also negative. The results of fungal serologic tests were normal, and multiple cultures of sputum for acid-fast bacilli and fungi showed no growth.

Multiple biopsies of pulmonary parenchyma were obtained through the fiberoptic bronchoscope. Sections of tissue revealed numerous noncaseating granulomas with multinucleated giant cells. Special stains for acid-fast and fungal organisms, as well as cultures of the pulmonary tissue, were negative. A pleural biopsy was considered but was not performed after the histologic diagnosis had been established by pulmonary biopsy. Therapy with corticosteroids was initiated, and by two months the symptoms had resolved, along with the biochemical abnormalities. A chest x-ray film at that time showed that the pleural abnormality and most of the infiltrate had resolved.

Discussion

Although both primary involvement of an upper lobe and pleural effusion have been reported in patients with sarcoidosis, the combination of the two abnormalities, as seen in this patient, must be exceedingly uncommon. Upper lobe involvement mimicking adult tuberculosis was noted to occur in 54 (9 percent) of a series of 616 patients with sarcoidosis. The changes were usually bilateral and were believed to represent a late stage of sarcoidosis, with upper retraction of the pulmonary roots resulting in migration of the densities from below to the upper zones. Earlier chest x-ray films in many of these patients showed the usual changes of hilar adenopathy and parenchymal infiltration, which were most marked in the middle and lower pulmonary fields. By contrast, our patient had unilateral upper lobe involvement and had had a completely normal chest x-ray film two years previously.

Pleural involvement in sarcoidosis has also been reported to be unusual, with Chusid and Siltzbach finding only seven patients (1 percent) in a series of 950 patients with this problem; however, in another retrospective review, 23 (10 percent) of 227 patients were found to have evidence of pleural reaction. Both studies emphasized that pleural sarcoidosis was almost always associated with the chronic stage of the disease, with either extrathoracic or extensive pulmonary parenchymal lesions being associated with the effusions. In contrast, our patient had roentgenographic evidence of pleural involvement at the symptomatic onset of his disease.

The importance of recognizing this unusual initial occurrence of sarcoidosis is obvious. Confusion with tuberculosis could easily occur if one does not persist in obtaining a definitive diagnosis in each case.

References


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