depends upon isolation of the organism in culture. Cultures for atypical mycobacteria may, however, be negative even when infection is present. Only about 50% of excised diseased lymph nodes will be culture positive[4]. With a mean time of about eight weeks for culture and twelve weeks for sensitivity results[5], initial diagnosis depends greatly on the clinical features.

Many forms of therapy for AMCA have been practised, including surgical excision, antituberculous chemotherapy and a combination of surgery and chemotherapy. In case 1, incomplete excision was complicated by chronic sinus formation and discharge. Needle aspiration and incision and drainage likewise tend to be complicated by sinus or fistula formation, as demonstrated by case 3. A sinus can persist for months or years[6], with serious cosmetic implications. In case 3, treated with rifampicin, healing was marked by fibrosis and scarring of the skin; excision of residual scar tissue may eventually be necessary for cosmetic reasons.

The role of chemotherapy in the treatment of AMCA is not entirely clear. Whilst atypical mycobacteria are generally resistant to first-line antituberculous agents there have been many well-documented cases of AMCA resolving during treatment with such medications[7]. More recent experience favours surgical excision over prolonged antituberculous therapy. For recurrent AMCA after surgery, treatment with newer chemotherapeutic agents such as the macrolides (e.g. clarithromycin) and fluoroquinolones (e.g. ciprofloxacin) has yielded encouraging results[8], but no controlled trials have yet been reported. The treatment of choice is complete surgical excision, with or without chemotherapy. It is both diagnostic and therapeutic.

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Cardiac memory mimicking myocardial ischaemia

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Cardiac memory describes the phenomenon whereby the T-wave abnormalities that result from a change in the direction of cardiac activation, as during ventricular pacing, persist for a while after the end of pacing.

CASE HISTORY

A woman of 85 was admitted after a syncope. For the past three months she had been experiencing dizziness and dyspnoea on exercise. Her heart rate was 60/min, blood pressure was 105/60 mmHg and there was a mitral systolic murmur. There was moderate cardiomegaly on the chest radiograph and echocardiography revealed biatrial dilatation and severe mitral valve insufficiency. A 12-lead electrocardiogram (ECG) showed atrial flutter with non-specific lateral repolarization abnormalities, and on a 24-hour recording the ventricular rate ranged from 37 to 170/min. During the hospital stay she converted spontaneously to bradycardic sinus rhythm, heart rate 40/min (Figure 1, A). The diagnosis was consistent with sick sinus syndrome; a dual chamber pacemaker was implanted and was seen to function (Figure 1, B). The next day, the patient reported acute chest pain and a 12-lead ECG showed new T-wave inversions with an intrinsic ventricular rhythm (Figure 1, C). The pain resolved rapidly and the T-wave abnormalities returned to baseline within 24 hours (Figure 1, D). The initial differential diagnosis in this episode included: acute myocardial ischaemia due to plaque rupture, or coronary embolism arising from the large left atrium; the mitral valve disease and the intermittent atrial arrhythmia; drug effects; penetration of the ventricular lead (which had passive fixation); and cardiac memory phenomenon with simultaneous non-cardiac pain. Acute myocardial necrosis was excluded by repeated analysis of heart enzymes, which never exceeded the normal range. Drugs that cause repolarization abnormalities, e.g. digitalis, were not being

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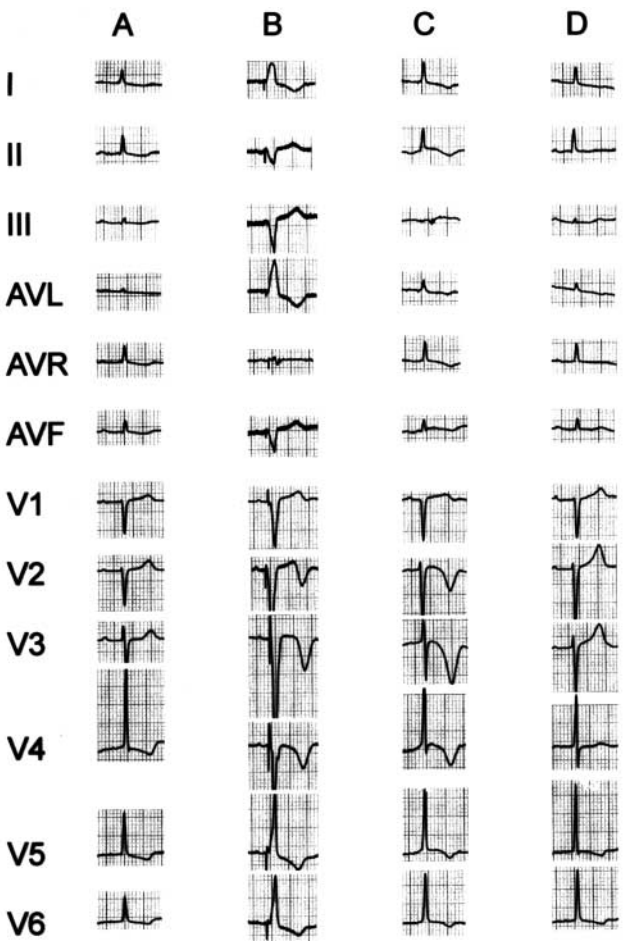

Figure 1 **Twelve-lead ECGs from patient with sick sinus syndrome.** A, sinus rhythm and nonspecific lateral repolarization abnormalities before pacemaker implantation; B, ventricular pacing one day after pacemaker implantation; C, new T wave inversions with intrinsic ventricular rhythm two days after pacemaker implantation; D, resolution of T wave inversion three days after pacemaker implantation

given at that time. Ventricular lead penetration was excluded by the absence of a pericardial effusion on repeat echocardiography. Therefore, we considered these transient repolarization abnormalities consistent with cardiac memory phenomenon.

COMMENT

Although the existence of cardiac memory has been known for many years, there is still limited information regarding its physiological significance and clinical implications. Clinicians do, however, need to be aware that this electrical curiosity, also termed Chatterjee phenomenon[1], can imitate acute myocardial ischaemia. Cardiac memory is characterized by persistent but reversible T-wave changes on the surface ECG induced by an abnormal electrical activation pattern: the reported stimuli include ventricular pacing[2], intermittent left bundle branch block[3], periods of pre-excitation observed in Wolff–Parkinson–White syndrome[4], and episodes of tachycardia[5]. The extent and the direction of T-wave deviation depend on the duration and the direction of the abnormal electrical activation; the phenomenon can persist for several weeks. The underlying cellular mechanisms are unclear, but existing data point to modification of specific potassium channels and changes in the phosphorylation status of the cAMP responsive element binding protein (CREB)[6].

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