Comment

Deaths from rhesus haemolytic disease of the newborn have been declining for many years, but since the introduction of anti-D the fall has been much steeper.[5]

It is not possible to draw definite conclusions from data based on only two years, but trends that would be expected if the prophylaxis was successful (assuming treatment of rhesus haemolytic disease of the newborn remains substantially unchanged) may be assessed.

Given the natural history of worsening of the disease in consecutive babies, the reduction in deaths is more likely to be seen initially in the liveborn babies. This is because mothers of stillborn babies will usually have been immunised for longer than mothers of liveborn babies—and many of them will still be in categories 1 and 2. On the other hand, the numbers in categories 3 and 4 should remain approximately constant since in neither group is prophylactic anti-D administered. Category 5 (failures) would probably be unaltered, but the number in category 6 should decrease perhaps first in the liveborn babies. In the non-rhesus cases there would be little change, since our earlier report could not yet have made an impact and the 8th revision of the ICD[3] was still in use. The table supports some of these predictions, particularly of the reduction in the number of deaths of liveborn babies in Category 6 and the similarities in non-rhesus cases.

We hope that the impact of our three reports, together with the introduction of the 9th revision of the ICD, which revised the hydrops coding, will result in both more accurate statistics and more efficient anti-D prophylaxis.

We are most grateful to Dr J A C Weatherall, Professor P L Mollison, Professor E D Acheson, and Dr G N Smith for their helpful comments.

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Pain in the thenar eminence: a rare case of atypical angina

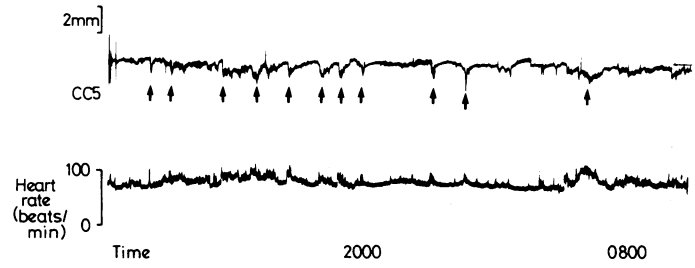
We report a case of atypical angina in which there was exercise-induced pain in the thenar eminence without any precordial symptoms or other presentation of cardiac pain.

Case report

A 64-year-old woman had had pain in the left thenar eminence since 1972. She had had no pain in the thumb or any other part of the hand, arm, or chest and it was strictly related to exertion. Referred to a neurologist in 1972, the patient underwent myelography, and in 1973 a rheumatologist performed electromyography; both results were normal. In 1975 she consulted an osteopath and finally visited four separate acupuncturists (in 1976 and 1977), who could not relieve her symptoms. By 1978, the pain was occurring on slight exertion and also spontaneously at night. The relation of the pain to exertion finally brought her under our care.

During a computer-assisted graded treadmill exercise test she developed pain in the left thenar eminence accompanied by notable ST segment depression in bipolar monitoring leads within 2·5 minutes. Continuous 24-hour ambulatory ST segment monitoring was performed using a frequency modulated tape recorder.[1] During this time she had 11 episodes of pain in the thenar eminence and each was associated with ST segment depression (figure). Thallium-201 scintigraphy showed normal perfusion at rest and multiple areas of relative hypoperfusion in the inferoposterior and anterior regions after exercise. Left ventricular and coronary angiography showed inferior wall akinesia and severe triple-vessel disease.

Treatment with propranolol 80 mg three times daily produced some improvement, but she continued to have spontaneous attacks of pain. Verapamil 120 mg three times daily produced further improvement, but she continued to have pain while resting so a bypass graft was considered. The patient refused surgery, and the dose of verapamil was increased to 120 mg four times daily. Over the next month her symptoms resolved, with an increase in treadmill exercise time to 5·2 minutes and only one episode of notable ST depression in 24 hours of continuous monitoring.



ST segment and heart rate trends from continuous ambulatory electrocardiogram monitoring. Arrows show time of attacks of pain in thenar eminence.

Comment

Since Heberden's first description of angina pectoris, many comprehensive accounts of the syndrome have been published.[2-4] Cardiac pain is transmitted via the visceral afferent fibres of the sympathetic nervous system which enter the spinal cord via the white rami communicantes and the dorsal roots from T1 to T4.[5]

Despite general agreement among anatomists regarding the autonomic nervous connections of the heart, however, cardiac pain is notoriously variable.[3] The radial side of the hand and the thumb is supplied by the C6 dermatome, which seems to be rarely affected. One out of 150 cases reported by Sampson[3] and one out of 160 cases reported by McKenzie[2] were of precordial pain which radiated to the thumb. Anginal pain may also radiate to a site of past injury,[4] but there was no history of trauma in this patient.

This patient had a rare atypical presentation of cardiac pain which led to considerable diagnostic difficulties. The absolute localisation of pain to the left thenar eminence together with the absence of any pain or discomfort in the chest during exertion or at rest seems to be a rare presentation of classical coronary artery disease which has not been described.

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Correction

Alcohol and the emergency service patient

An error occurred in this paper by Dr S Holt and others (6 September p 638). The third sentence of the Summary should have read "Clinical assessment of intoxication resulted in a false-negative diagnosis in 19% of inebriated patients. . . ."