AMOEBIIC PERICARDITIS

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IT HAS been reported that *Entamoeba histolytica* cysts are passed by 1.6-4.7% of the population in this country (Morton, Neal and Sage, 1951; Matthews and Smith, 1921). Clinical amoebiasis is not so rare either, and may at times develop in those who have never left Northern Europe or been resident in accepted endemic areas. (Manson Bahr, 1943; Paulley 1961; Conway and Watt, 1961).

Pleuro-pulmonary involvement not only commonly complicates hepatic amoebiasis but is often the presenting feature, especially of abscesses in the right lobe of the liver. This was true of a personal series. (Paulley, 1961). Of those seventeen patients pericarditis occurred but once, and was then associated rather unusually with a large right pleural effusion arising from a right hepatic abscess. The patient recovered on medical treatment.

Pericarditis as a complication of hepatic amoebiasis developed in 7.5% of 293 cases of pleuro-pulmonary, pericardial, and cerebral amoebiasis culled from the world literature over twenty years, (Takara and Bond, 1958). It carries a 55% mortality rate. Prior to that Norris and Beemer (1956), reporting a case and reviewing the literature, collected 47 examples of suppurative pericarditis with only two survivors, whereas Carter and Korones (1950) describing 14 instances of pericarditis in proven amoebic sufferers reported four survivors. In Lamont and Pooler's series (1958) of 250 patients with hepatic amoebiasis there were seven with pericarditis and five of these died.

Takara and Bond summarise the matter by observing that suppurative amoebic pericarditis usually results from rupture of an hepatic abscess of the left lobe of the liver, and that it is usually fatal. However an occasional patient who is treated early, or in whom a non-suppurative effusion develops, may survive. Rarely, they say, does an abscess of the right lobe of the liver give rise to pericarditis. Lamont and Pooler refer to three phases in amoebic pericarditis (1) Initial sympathetic slightly purulent effusion (2) Rupture of the hepatic abscess often causing death by tamponade (3) A phase of constriction developing over several weeks, and which could only be relieved by pericardectomy.

A possible example of hepatic amoebiasis of the left lobe of the liver occurring in this country and in which pericarditis was the main presenting feature seems sufficiently unusual to merit a report. The patient meets the diagnostic criteria of Lamont and Pooler (1958).

Case Report

A man aged 51 presented on 5.1.1962 with severe burning precordial pain, worse on breathing, a temperature 100°F., pulse 110, and B.P. 110/70 mm. Hg. Cardiac infarction was suspected because of ECG changes of an old posterior cardiac infarction, a diagnosis made clinically two years previously. The observation of a very loud widespread pericardial rub led to discontinuance of anticoagulants on review the day after admission. At the same time pleural pain followed by effusion developed in the left lower chest. Radiography confirmed this with elevation of the left diaphragm. It was assumed that pulmonary embolism had also occurred.

It was not until the fourth day that the following developments made the diagnosis of cardiac, combined with pulmonary, infarction suspect.

(1) The patient's increasingly intense precordial pain which was provoked by the least movement, leading to closer questioning about the mode of onset.

(2) A series of unimpressive lactic dehydrogenase figures: 187 units, 375 units, 287 units.

(3) Serial electrocardiograms did not support infarction, but showed developing pericarditis. Other investigations: Hb. 90%, WBC 11,600/cu. mm., polys 83% falling to 5,800 by 10.1.1962. LE Cells negative. Electrophoresis: increase in α 2. Virus studies were negative. Mantoux 1/10,000 negative. Barium swallow on 16.1.1962: no evidence of hiatus hernia.

Closer questioning now revealed that precordial pain had first arisen in the epigastrium when twisting to look out of the rear window of his car two days before admission. It recurred more severely (retrosterally) when he repeated the same movement twelve hours before admission. The fact that deep breathing hurt him over the precordium as well as the site of the pleural rub had been overlooked. He described this pain as "somewhere deep inside behind the breastbone like a lot of pins stuck on the outside of a balloon with the sharp ends pointing outwards."

At this stage pulmonary infection, possibly in an infarct, and involving the pericardium was considered, and he was given penicillin and chloramphenicol from 8.1.1962-16.1.1962. However the patient's fever, pain, and condition worsened. The physical signs showed no improvement after a week on antibiotics, and he now had left subcostal tenderness. Other causes of pericarditis were now considered. Of these neoplastic disease or a complicated hiatus hernia seemed more likely than tuberculosis or virus infection, and suspicion also turned to amoebic or non-specific abscess of the left lobe of the liver. There had been residence in India 1943-1945 but no history of amoebic dysentery. Daily the patient became more like a graven image, holding himself rigidly, bolt upright, terrified of the pain which occurred on the least movement. This feature, so significant of amoebic abscess, reached its climax on 6.1.1962 after a journey to the X-ray Department for a barium

swallow examination. So grave was his condition that evening (16.1.1962) with a fever of 102°, and pulse 120, that a trial of emetine hydrochloride was initiated.

Next morning he said "I feel 150% better. It is as though the balloon has gone down to half its size." Night sweats which he had had for a week ceased at once. He received injections of emetine gr. 1 for 4 days and later two further days, this was followed by a three week course of chloroquin. The pericardial rub disappeared after eight days of treatment, but the pleural rub was still present on deep inspiration in the left anterior axillary line on 6.3.1962, but the ECG showed no sign of pericarditis on that date. By May 1962 he said he felt better than he had done for many years. Retrospectively his wife said that she had heard his "scrapping" heart beat in bed for at least a week before admission.

To conclude the following is a passage taken from an article by Michon, Larcen, Grosdidier, Streiff and Frisch from Nancy (1959) with a description of amoebic pericarditis so graphic that it forbids translation.

"Tantôt le diagnostic d'amibiase hépatique est porté et le traitement mis en route lorsque la complication se produit, mais le plus souvent le diagnostic n'est pas établi quand, après une période prémonitoire qui associe altération de l'état général température, dyspnée, douleurs épigastriques,, s'installe brutalement un tableau d'asphyxie souvent mortel. Le malade, à l'occasion d'un changement de position, pris soudain d'une douleur très vive, tombe, pousse un cri et meurt en quelques minutes."

Summary

A patient with pericarditis and left sided pleurisy continued to deteriorate until he was given emetine. However, Synek and Synek (1964) have called in question the diagnostic specificity of emetine for amoebiasis.

Dressler's syndrome remains an alternative diagnosis, although the mode of onset, the intense pain on movement, and the patient's parlous state were, in the author's experience, not typical of that condition. Despite the diagnostic doubt

inevitable in patients with amoebic abscess who survive the report may be of use as a further reminder of pericarditis as a complication of amoebiasis, (Mullen and Williams, 1965) a fact, well known in the tropics but which may escape notice in this country.

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