AMOEBIC PERICARDITIS: A CASE REPORT

G. GORDON, M.B., CH.B. (CAPE TOWN)

Former Medical Registrar, King Edward VIII Hospital, Durban

Pericarditis is a rare complication of amoebic abscess of the liver. Reviewing the literature, Carter and Korones[1] reported a total of only 44 proved cases of suppurative amoebic pericarditis for the period 1885 to 1950. The mortality rate was very high, and diagnosis during life was made in only 2 cases, one of which recovered. In an unselected series of 2,000 autopsies performed at King Edward VIII Hospital during a 5-year period 1950-55, 8 cases of suppurative amoebic pericarditis were found. The number of autopsies performed was less than 10% of all deaths.

The condition may present in the following ways:

1. Cases of liver abscess with which is associated a pericardial friction-rub or electrocardiographic changes suggestive of pericarditis. There is no pericardial effusion or cardiac distress and the condition clears up as the liver abscess is treated. This is probably not very uncommon with abscesses of the left lobe of the liver.

2. As a serous (sympathetic) pericardial effusion without frank rupture of the abscess into the pericardium. When the diagnosis of liver abscess is not apparent this type may easily be confused with pericarditis from other causes, especially tuberculous pericarditis. The serous effusion may become purulent.

3. Sudden rupture of the liver abscess into the pericardium may occur, with suppurative pericarditis. This may present like the case described below, but sudden collapse and rapid death may occur.

The following case-history illustrates some typical clinical features of suppurative amoebic pericarditis.

CASE REPORT

An African male 29 years old was admitted to King Edward VIII Hospital, Durban, on 12 November 1954 complaining of severe pain in the left hypochondrium and slight non-productive cough, for 6 weeks. The pain had become progressively worse since its onset. There was no history of previous dysentery or other significant illness.

Examination. The patient, a well built and well nourished man. was acutely ill and distressed. His temperature was 103°F. The jugular venous pressure was not raised and there was no oedema. There was a very tender fluctuating swelling of the anterior abdominal wall in the left hypochondrium extending down from the costal margin and about 2½ inches in diameter. The surface of the swelling was smooth, with normal overlying skin.

The pulse was normal and the blood pressure 140/60 mm. Hg. The apex beat was easily palpable and not displaced. The heart sounds were of normal intensity and a pericardial friction-rub was heard at the apex.

Special investigations were as follows:

Blood: Hb. 5·6 g.% (38%). P.C.V. 20%. M.C.H.C. 28%. White blood-cells 23,000 per c. mm. polymorphs 90%, lymphocytes 9%, eosinophils 1% mononuclears 0%). ESR 76 mm./hr (Wintrobe).

Stool: Normal.

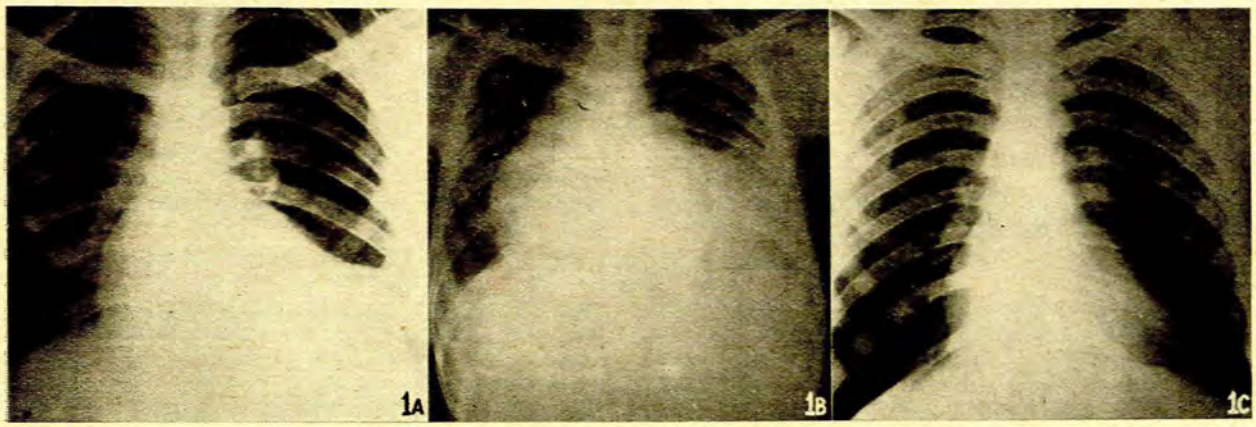

Fig. 1. A: Taken on the day of admission, showing elevation of the left dome of the diaphragm. B: 12 hours after rupture into the pericardium showing great increase in the size of the heart shadow. C: 1 month after discharge from hospital. The heart is now normal radiographically.

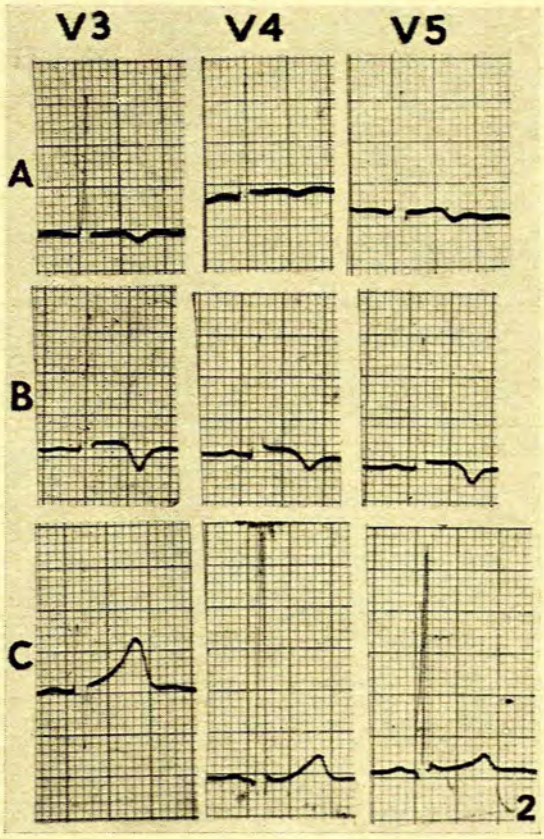

Fig. 2. A: 4 days after admission, showing inverted 'T' waves. B: 1 day after rupture into the pericardium. C: 1 month after discharge from hospital. The 'T' waves have become normally upright. Note also the increase in voltage of 'R'.

Radiograph of chest on admission (Fig. 1A): 'The left dome of the diaphragm is elevated, with some pleural reaction at the left base. A radiograph of the abdomen and fluroscopy of the chest indicated that the stomach with its normal air bubble was being displaced posteriorly by a large abscess in the left lobe of the liver.

An *electrocardiogram* taken 4 days after admission showed 'T' wave changes compatable with pericarditis (Fig. 2A).

Progress. Treatment with Camoquin[2] was started on the day of admission and a blood transfusion of 1 pint of whole blood was given on the 4th day. Three days after admission 500 c.c. of thin 'anchovy sauce' pus was aspirated from the abdominal swelling. Four days later annother 300 c.c. of pus of a similar nature was removed from the same site. The patient was now very much improved and his temperature was normal. This improvement continued until the evening of his 11th hospital day, when he developed a sudden severe lower substernal pain. His condition deteriorated rapidly and he became very dyspnoeic. The neck veins became engorged and there was pitting oedema over the sacrum. Pulsus paradoxus was present. There was now no palpable cardiac impulse and the heart sounds were faint and distant. Immediate aspiration from the original abdominal site yielded 300 c.c. of typical 'anchovy sauce' pus. The following day a radiograph of the chest showed a great increase in the size of the heart shadow (Fig. 1B). The pericardial sac was aspirated through the left 5th intercostal space 3/4ths of an inch lateral to the mid-clavicular line and 600 c.c. of thin 'anchovy sauce' pus was removed. The laboratory report on this fluid was as follows:

Macroscopic: Turbid blood-stained fluid with a large clot.

Microscopic: Pus ++. Erythrocytes +++. Organisms not detected (neither acid-fast bacilli nor amoebae were detected).

Protein 4·6%.

The patient improved almost immediately. The apex beat was now palpable in the mid-clavicular line and the heart sounds became louder.

Treatment with Camoquin was stopped and a course of emetine hydrochloride. 1 gr. daily for 10 days and procaine penicillin, 600,000 I.U. twice daily by injection, was started. He was also given 2 c.c. of mersalyl intramuscularly and this was continued twice weekly. The emetine was followed by a course of Chloroquin, 250 mg. twice daily by mouth for 20 days.

Five days later the pericardium was again aspirated and 150 c.c. of a light-chocolate-coloured pus was removed. The abdominal swelling was aspirated through the original site immediately afterwards and 800 c.c. of a similar chocolate-coloured pus was obtained. Aspiration of the pericardium and liver abscess was

attempted 7 days later, but no pus was obtained. The patient improved steadily and was discharged from hospital 40 days after admission symptom-free and with no signs except that the ECG was still abnormal, showing inverted 'T' waves in the limb leads and leads V3-V6.

He returned to hospital for review 1 month after discharge. He was feeling well and no abnormal signs were found. The electrocardiogram and radiograph of the chest were now both normal (Fig. 2C and Fig. 1C), as was the blood count. The ESR was 4 mm. per hour (Wintrobe).

When he was last seen 2 months after discharge from hospital he was quite well.

DISCUSSION

The patient presented with an amoebic abscess of the left lobe of the liver and a pericardial friction-rub. On the 11th day of admission the abscess ruptured into the pericardium.

The pericardial friction-rub was a significant finding, indicating that more serious pericardial complications were likely. This has also been the experience of other authors.[1,3]

The electrocardiographic findings shortly after admission confirmed that pericardial involvement had already occurred.

I have, however, seen cases with friction-rub and similar electrocardiographic changes that recovered uneventfully on conventional treatment without showing any evidence of serious cardiac involvement.

There are too few records of successfully treated cases to indicate clearly what are the best methods. It is clear, however, that emetine or Chloroquin must be given to eradicate the amoebae in the tissues. One might argue that there is cardiac embarrassment and for this reason emetine should be avoided, but I am not convinced that Chloroquin is as rapidly effective in tissue amoebiasis as emetine and would prefer the latter drug, at any rate up to a total dose of 6 gr. Chloroquin could be given simultaneously with, or after, the emetine.

Huard and Meyer-May[4] recommend surgical drainage of the liver abscess and pericardium but, unless aspiration was technically very difficult because of thick pus or inaccessability of the abscess, I should prefer to avoid surgery since the patient is gravely ill and the risk of secondary infection is greater than with aspiration. Both pericardium and liver abscess should be aspirated.

As with uncomplicated liver abscess it seems logical to give an intraluminal amoebicide such as diiodohydroxyquinoline to eliminate bowel parasites, when the patient has recovered from the acute episode.

In the case reported here recovery seemed complete, but the follow-up is short and eventual constrictive pericarditis may be a possibility. The remarkable absence of scarring in the liver following amoebic abscesses encourages one to think that the cure may be permanent.

REFERENCES

1. Carter, M. G. and Korones, S. B. (1950): New Engl. J. Med., **242**, 390.
2. Gordon, G., Wilmot, A. J. and Elsdon-Dew, R. (1955): Trop. Med. Hyg., **58**, 133.
3. Kern, F. (1955): Arch. Intern. Med., **76**, 88.
4. Huard, P. and Meyer-May, J. (1936): *Les abcès du foie*, pp. 114-119. Paris: Masson et Cie.