SUPPURATIVE AMŒBIC PERICARDITIS
A COMPLICATION OF AMŒBIC LIVER ABSCESS IN AN AFRICAN CHILD

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Introduction
This appears to be the first report from West Africa of suppurative amœbic pericarditis in a child. It is reported firstly because the condition is rare in children, and secondly to draw attention to the recent increase in reports of juvenile hepatic and pericardial amœbiasis, many of which have emphasized the importance of amœbic infection in endemic areas as a cause of death in children.

Case Report
A twenty-three-month-old male child of the Yoruba tribe was admitted to hospital in Kaduna, Northern Nigeria, on 26th September, 1960. He had been brought to the hospital out-patient department seven days before with a cough, discharging ears, vomiting and diarrhœa, for which penicillin had been given with some improvement. The day before admission he had stopped eating, developed swelling of the abdomen and had vomited three roundworms.

On examination the child was thin, undersized and obviously anaemic. He was weak and listless. The abdomen was distended. Temperature 103°F. (39.5°C.). Pulse 148 per minute. Respirations 34 per minute. The tongue was coated but the throat not inflamed. The skin was normal and there were no enlarged glands. The neck veins were not congested. Scattered crepitations were heard on both sides of the chest and the air entry was noted as poor. There was no neck rigidity. Pupils were equal and reacted to light.

Investigations

Blood
W.B.C. Total count: 17,750 per c.mm. Polymorphs 62 per cent, lymphocytes 26 per cent, monocytes 9 per cent, eosinophils 3 per cent.

Red blood cells. Marked hypochromia, occasional target cells, no sickled forms. Sickle cell test negative. No malarial parasites. Haemoglobin 6.6 g. per 100 ml.

Stools

Treatment and Progress
Tetracycline syrup 125 mg. stat. and 62.5 mg. six-hourly was prescribed. Later that evening there were more adventitious sounds in the chest, but the temperature had fallen to 99°F. (37.2°C.). The child was taking fluids by mouth and had passed one small stool containing mucus. The next morning the temperature was 98.6°F. (37°C.), but the patient still looked very ill and a tachycardia of 146 per minute persisted. It was now noticed that the apex beat was neither visible nor palpable and that the heart sounds were faint. A chest X-ray showed a gross enlargement of the cardiac shadow which almost filled the chest (cardio-thoracic ratio 84 per cent). The
child suddenly collapsed and died while being prepared for pericardial aspiration. Clinical diagnosis: pericardial effusion; ascariasis.

**Autopsy**

The general appearance was that of a poorly nourished male child with no external abnormalities. There was a large purulent pericardial effusion, which proved to be continuous with an abscess in the left lobe of the liver. The pericardial surface of the heart was covered by thick fibrin. The myocardium appeared pale. The heart valves were normal. The lungs were compressed but otherwise normal. Some mucoid exudate was present in the bronchi and trachea. The left lobe of the liver was replaced by a large abscess cavity, which contained pale yellow pus and was bounded by a thick, fibrinous wall. The right lobe of the liver was normal. An adult roundworm was lying free in the ileum and another in the lumen of the vermiform appendix. The spleen was normal in size but the Malpighian corpuscles were prominent. Adrenal glands, kidneys and urinary passages normal. Brain and meninges not examined. Amoebae were seen in the wall of the liver abscess and also in the connective tissue outside the pericardium.

Pathological diagnosis: suppurative amoebic pericarditis secondary to ruptured amoebic liver abscess; ascariasis.

**Discussion**

Amoebiasis in children has been considered rare, and therefore its complications are not often seriously considered in the differential diagnosis of puzzling abdominal or thoracic disease in young patients. Standard textbooks may not mention juvenile amoebiasis (Trowell and Jeliffe, 1958; Fairley, 1961) or may refer to it only very briefly (Manson-Bahr, 1961).

Gelfand (1957), on the other hand, does not agree that children are rarely affected, and refers to reports from South Africa and Nyasaland. Wilmot (1962) finds that patients between six and seventeen years of age seem to enjoy a greater immunity to ill-effects from *E. histolytica* than either older or younger patients.

It is of historical interest that the first description of pathogenic amoebae (Lambl, 1860) was based on examination of a stool specimen from a two-year-old girl. Also four of the earliest reports of amoebic liver abscess were of children under one year old (Brown, 1824; Niblock, 1911; Leroy des Barres, 1930; Biggam and Ghalioungui, 1933). Before 1957 reviews of amoebic liver abscess in children consisted of collections of papers most of which were single case reports. Good bibliographies are given by De La Maza and Guzman (1953), Lestrade and Gerineau (1956), Senecal et al. (1957) and Smith et al. (1955). In more recent years large personal series have been reported. Fifty-four cases have been described in Mexico and Venezuela (Torreolla et al., 1956; Salas et al., 1958; Biagi, 1958; and Gomez Malaret et al., 1960). Walt (1959) reported 16 cases encountered in Durban in a period of eight months, and claims personal knowledge of 72 other cases in the city. Scragg (1960) describes 53 cases from his own experience in Durban between 1951 and 1958; 48 (91 per cent) were under three years old and the youngest was an infant of eight weeks. McDougall (1960) in Nairobi met ten cases of liver abscess out of 17 cases of juvenile amoebiasis in a period of fourteen months. Scragg (1960) also mentions reports from the Philippines, Italy, Algiers, Egypt and North America.
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It seems likely that this apparent increase in the incidence of juvenile am\textaeic liver abscess in Africa is related to the modern increase in facilities for investigation of the African patient. In West Africa the fatalistic acceptance of death in young children common in many tribes prevents cases from coming to the doctor. The tendency of am\textaeic dysentery to remit spontaneously, and also the clinical response which may occur to tetracycline therapy, may cause am\textaeic infection in infants in endemic areas apparently to respond to antibacterial therapy and thus may not be investigated further.

A review of published reports of suppurative am\textaeic pericarditis between 1885 and 1950 was undertaken by Carter and Korones (1950). They reported one case and traced reports of 43 proved cases. Only two of these were diagnosed during life, and there is no mention of occurrence in children. Buri \textit{et al.} (1955) reported the condition in three adults, all diagnosed in life, of whom one survived, and Norris and Beemer (1956) in a further three adults, of whom two had been diagnosed in life. Two more surviving adults were reported by Gordon (1956) and Acevedo Olvera \textit{et al.} (1960). The following authors have reported suppurative am\textaeic pericarditis in children: Torreolla (1956), two patients under five years old; Salas (1958), two patients under four years old; Scragg (1960), one patient. Gordon (1956) quoted interesting post-mortem figures to the effect that in the King Edward VIII Hospital in Durban am\textaeic pericarditis was found eight times in an unselected series of 2,000 autopsies, and he remarks that less than 10 per cent of all deaths came to post-mortem. Accounts of large series of am\textaeic liver abscess in adults suggest that about 2 per cent rupture into the pericardium (Vergoz and Hermenjat-Gerin, 1932; DeBakey and Ochsner, 1951; Lamont and Pooler, 1958). Multiple abscesses are common in children, perhaps commoner than single ones, and the left lobe is more likely to be involved. Therefore an increased incidence of rupture into the pericardium in children may easily be understood.

There is little doubt that am\textaeiasis tends to be a more acute disease in children, with more frequent complications and higher mortality (Wilmot, 1962). Biagi (1958) considers the disease of major importance as a cause of death in young patients in Mexico City. Scragg (1960) writes that acute awareness of the condition in children is necessary, and McDougall (1960) states that it is evident that even in small infants am\textaeic infection must be considered as a cause of bloody diarrhoea. Certain features in the published reports of juvenile am\textaeiasis suggest that the diagnosis may not be too difficult to establish if the condition is borne in mind. Salas (1958) found that colitis was present in 40 out of 44 fatal infections in children, and Biagi (1958) that 12 out of 13 children with hepatic manifestations had diarrhoea.

Our patient was diagnosed as suffering from pericarditis only a short time before death. If he had died a little earlier a post-mortem examination would probably not have been done and death would have been attributed to bronchopneumonia. Conversely, if he had lived a little longer it may be that his life could have been saved by the use of anti-am\textaeic drugs and aspiration, as reported by Gordon (1956), Buri (1955) and Acevedo Olvera \textit{et al.} (1960).

Summary

The occurrence of fatal suppurative am\textaeic pericarditis in an African child aged
twenty-three months is reported from Kaduna, Northern Nigeria. Reports of six
other cases in children have been traced. The present patient may be the youngest,
is among the first in which pericarditis was diagnosed in life and is the first reported
from West Africa.

Pericarditis was diagnosed in our patient just before death. He might easily have
died an hour earlier under the diagnosis of bronchopneumonia. Recovery from
suppurative amebic pericarditis is now being recorded, and would be more common
if the diagnosis were made earlier.

A review of the literature emphasizes the abrupt increase in the reported incidence
of juvenile amebiasis from several endemic areas in recent years. It
is suggested that there may be a high incidence of amebic infection in infants and children as yet
unrecognized in other parts of the world. This is important because amebiasis
is virulent in infants, with frequent complications and a high mortality if untreated.

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