Amoebic Liver Abscess in Rhodesia in the Adult African

PART II

BY

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SITE OF ABSCESS

The site of the abscess is not always easy to determine with any exactitude. One may presume that if the right dome is elevated the abscess is in the right lobe, and should the tender mass be found in the inferior surface of the right lobe that this is where it is likely to be. Although an amoebic abscess of the liver is typically single, multiple abscesses are by no means uncommon; and whilst we may clearly refer to the site of the abscess, we cannot be certain that other abscesses are not present as well at the same time (Figs. 2, 3, 4 and 5).

In the male the main site for the abscess in the liver was the upper surface of the right lobe.

MAIN	SITE	OF	PRESENTATION
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Right upper aspect of liver	 	39
Inferior surface of the liver,	 	13
Left base (left lobe) of liver :	 	1
		53

My findings would agree with those of Wilmot, who reports that a left lobe abscess is rare, probably occurring in the proportion of about nine abscesses on the right side to one in the left lobe. Multiple abscesses occurred in about 24 per cent. of cases.

In the recognition of liver abscess radiological examination of the upper abdomen must be regarded as an essential part of the investigation. There are two aspects of the investigation which deserve comment. The first is fluoroscopic study of the two domes of the diaphragm, noting its movements and whether there is any restriction in their movement, although this disorder will almost invariably be found on the right side. It is a pity that this test is not done more often. but facilities for fluoroscopy are limited owing to the lack of radiologists. This investigation is of special value not so much in those in whom the outline of the right dome is abnormal and elevated, but in those in whom there is no altered position of the diaphragm, but there is limited movement of the affected dome (Figs. 4 and 5).

The main changes described by Rowland (1963) are an elevated right dome of the diaphragm and a localised bulging or "humping" effect. Besides this, a small pleural effusion or clouding of the right costophrenic angle is frequently encountered. Other significant findings include a basal atelectasis which may be either linear or from a compression collapse. Rowland (1963) records that the site for an amoebic abscess is in the anterior basal segment of the right lower lobe, an unusual site for other forms of lung abscess (Figs. 6 and 7).

Rowland groups the patients with liver abscess into three categories. In Type 1 abscesses occur without radiological changes. In Type 2 there are minor radiological changes such as pleural effusion, moderate elevation of restricted movement of the diaphragm. In Type 3 the radiological changes are gross, with a considerable elevation of the right dome with restricted movement. Elevation in the diaphragm was not an invariable finding, especially when the enlargement is downwards (Figs. 8 and 9).

The following findings were noted in 28 subjects in whom changes were recorded on X-ray:

Total elevation of right dome	11864	man.	18
Localised bulging in right dome			1
Patch of pneumonitis at right base			3
Pleural effusion at right base			3
Pleural effusion at left base			1
Abscess right base			2
			-
			28

COMPLICATIONS

There are a number of serious complications which may follow a liver abscess. Firstly, it may rupture or communicate by a leak with the peritoneal cavity, and Wilmot describes four clinical forms of this complication:

- (1) Sudden rupture of the abscess with a generalised peritonitis.
- (2) A more gradual leak, giving rise to a localised abscess.
- (3) The development of a chronic generalised peritonitis.
 - (Figs. 10 and 11.)

ASCITES

Ascites must be a rare complication of liver abscess and ordinarily is not a sign which one associates with it. The difficulty is to be sure that this is not due to an associated cirrhosis of the liver. In this series two patients showed an ascites. In one the fluid was typical of what one usually finds in ascites, being straw-coloured and clear and not resembling amoebic pus. The diagnosis can be established in these cases by finding the pus and by aspirating the abscess. The first patient, aged 42, was seen in May, 1959, because of abdominal swelling, ascites and pain. An abscess which was forming was noticed over the eleventh intercostal space on the right side behind. Pus was aspirated. The liver was not palpable. The serum was 5.5 g. per cent. (albumin 1.6 g. and globulin 3.9 g. per cent.).

PERICARDITIS

Pericarditis is another interesting complication of liver abscess. When it breaks it may leak into the pericardial sac. It is said to occur more often when the abscess is situated in the left rather than the right lobe (Wilmot, 1962).

AMOEBIC LIVER ABSCESS

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Fig. 2—Slight elevation of right dome of diaphragm with loss of translucency of the costo-phrenic angle.



Fig. 4—Patch of pneumonitis at right base due to abscess of liver which has been aspirated and can be recognised by the gas beneath the right dome.



Fig. 3-Greatly elevated right dome of diaphragm.



Fig. 5-Besides the raised right dome there is a linear atelectasis immediately above the dome.

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Fig. 6—Greatly elevated right dome with gas (due to aspiration of abscess).



Fig. 7—Patch of pneumonitis at right base. The dome on the affected side is not apparently elevated.



Fig. 8—Lateral view showing involvement of the middle segment of the right lower lobe bronchus.



Fig. 9—Amoebic abscess of liver with perforation into the lower zone of right lung, producing signs almost indistinguishable from a lung abscess.

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The first sign of a pericarditis may be a friction rub or signs associated with a pericardial effusion. In other cases there is a sudden rupture with the development of a true purulent pericarditis from the outset, in contrast to the serous effusion which may develop at first and continue for some weeks before it becomes purulent. The diagnosis of an amoebic pericarditis is usually made on recognising the underlying abscess of the liver. Usually, when there is an effusion in the pericardium, differentiation from tuberculous pericarditis is almost impossible. When the pus is of sufficient quantity the diagnosis may be established if this possibility is borne in mind.

In the series there was one patient, an African boy aged about 12 years, who developed a pericardial effusion which was probably secondary to a liver abscess. The boy came from Mtoko district of Mashonaland and was admitted to hospital on 28th November, 1962, because of a painful swelling on the posterior aspect of the right chest wall which had begun three weeks prior to admission. He was also said to have been feverish with loss of weight and some shortness of breath. He was eating well at the time



Fig. 10—Abscess in left lobe of liver connecting with the pleura and lung on the same side. (This case is not included in the present series.)

and there was no history of diarrhoea or of vomiting.

The boy looked thin and ill, with a striking pallor. A tender fluctuating mass 2 in. x 1 in. was present behind the right side of his chest two inches from the vertebral column. The right base was also dull, with marked diminution of the air at the right base. A few rhonchi were detected in this region. There were apparently no signs of heart failure and the heart sounds were normal. The patient was tender in the right hypochondrium. We considered a diagnosis of either liver abscess or of an empyema which was presenting posteriorly in the chest wall. On 30th November the fluctuating mass in the chest was aspirated. The first sample withdrawn was bloodstained, but after that a cloudy yellow fluid was obtained. It contained large numbers of polymorphonuclear leucocytes, but no organisms were cultured. On culture of the sputum, pneumococci were grown.

By then we became suspicious that the child might have a pericardial effusion. He was febrile, the temperature varying between 99.6° F. and 101.6° F. The right base was dull with a very poor air entry, and a few crepitations were audible at its upper limit.



Fig. 11—An abscess in the left lobe of the liver after aspiration.

On 5th December, 1962, the patient began to complain bitterly of pain in the right upper abdomen, which had become much distended. I decided to consult a surgeon, who advised an intravenous drip. Two days later (7th December) the abdomen was no longer distended or tender. The diet (5 per cent. dextrose) had apparently helped him a great deal. However, three days later the house physician reported that she thought the heart sounds were "rather muffled." An E.C.G. did not show any changes suggestive of a pericarditis. However, the patient remained unduly dyspnoeic, and so on 12th December 22 oz. of straw-coloured fluid was removed. The next day the boy was no longer dyspnoeic. As the boy was febrile as well, I decided to treat him for tuberculosis despite the fact that the Mantoux reaction was negative.

The next day the patient had improved dramatically. The breathlessness was much less. The boy was delighted with what was done for him. The right base, however, remained as dull as before and the patient continued to run a slight to moderate pyrexia (99-100° F.). By the 27th of the month he was again distended and this time I aspirated 36 oz. of a thick and slightly bloodstained fluid. The heart sounds continued to remain muffled. The aspiration was repeated on 5th January, 1963, and the patient appeared to be very tender over the right side of the liver, which could be felt about two inches below the right costal margin. We now suspected the presence of pus beneath the right Mr. Graham was consulted. After tapdome. ping one pint of purulent fluid from the pericardium he decided to drain the pericardium surgically. This Mr. Graham did and at the same time he incised the tender inflammatory mass in the chest wall. He traced it and proved that it was a fistula which passed through a hole in the diaphragm into the liver. He inserted a corrugated drain into the liver. Mr. Graham removed a small piece of the pericardium for histological examination. Microscopy showed rather dense fibrous tissue with a sparse chroniciinflammatory cellular infiltrate and with a small part of its' surface covered by a narrow zone of granulation The connective tissue was diffusely intissue. filtrated by acute and inflammatory cells (including large number of histiocytes). The pus from the abscess in the chest wall consisted largely of polymorphonuclear leucocytes, but on culture no growth was obtained. The pericardial fluid showed both polymorphonuclear and mononuclear cells, but the culture (aerobic and anaerobic) was sterile.

The tuberculous therapy was stopped and a course of chloroquin and emetine was substituted, and slowly the patient became sufficiently improved for him to leave hospital. The sinus in the back cleared up. Further follow-up was not possible.

TREATMENT

Wilmot et al. (1959) have reported favourably on the value of chloroquine when it is given alone for liver abscess, although the failure rate was over 25 per cent. Nevertheless this is a fairly satisfactory rate, as chloroquine is such a safe drug. Far better results are obtained with two courses of emetine. Wilmot favours a course of chloroquine followed by a single course of emetine, which he considers is as good as two courses of emetine. This authority does not apparently advocate that a liver abscess should be aspirated as a routine. He favours giving a course of chloroquine, followed by emetine, and noting the response. If, however, there is pointing, bulging or a markedly raised right dome of the diaphragm, some advantage would be gained by aspirating the abscess before starting drug therapy. Aspiration is preferable to open surgical drainage, which should be performed only after three or four aspirations have failed and only when it fails to achieve a diminution in the quantity of pus should surgery be contemplated (Wilmot et al., 1959).

REFERENCES

- ADI, F. C. (1965). W. Afr. med. J., 14, 181.
- BOYD, J. S. K. (1957). Trans. Roy. Soc. Med. & Hyg., 51, 47.
- ELSDON-DEW, R. (1950). S. Afr. med. J., 24, 160. FLSDON-DEW, R. (1957). Quoted from Problems Relating to Amoebiasis, by Prof. P. C. C. Garnham. Discussion by R. Elsdon-Dew, Amoebiasis Research Unit, Durban.
- FOSTER, W. (1965). A History of Parasitology. E. & S. Livingstone, Edinburgh.
- POWELL, S. J., WILMOT, A. J. & ELSDON-DEW, R. (1959). Trans. Roy. Soc. trop. Med. & Hyg., 53, 190.
- ROWLAND, H. A. K. (1963). J. trop. Med. & Hyg., 66, 113
- STAMM, W. P. (1957). Trans. Roy. Soc. trop. Med. and Hyg., 51, 306.
- WILMOT, A. J., POWELL, S. J. & ADAMS, E. B. (1959). Amer. J. trop. Med. & Hyg., 8, 623.
- WILMOT, A. J. (1962). Clinical Amoebiasis. Blackwell Scientific Publications, Oxford.