MATTERS ARISING

Solitary necrotic nodule

In 1983 we described a lesion of the liver which we called "solitary necrotic nodule"¹; we indicated that these lesions have characteristic morphological features and suggested several pathogenetic mechanisms. Consequently, Berry described "solitary necrotic nodule" as "non-existent"² and produced rather unconvincing evidence that these lesions were all sclerosed haemangiomas.³ The latter view seems to have become enshrined in a standard liver pathology textbook.⁴

Since 1983, we have identified a total of 10 more "solitary necrotic nodules." In one patient two lesions were present on the anterior surface of the liver. We believe that these lesions are relatively common. All of these cases have shown the same characteristic microscopical features as previously described but also have a very characteristic macroscopic appearance to which we would like to draw attention. The lesions have always been immediately subcapsular, slightly protruding from the surface of the liver with a sharply defined border and a depressed surrounding liver surface (figure). Thus we believe it may be possible to differentiate these lesions macroscopically from metastases, which are usually not so well defined, are flush with the liver surface, and often centrally umbilicated.

In all of the lesions we have seen we have never been able to demonstrate positively a pathogenetic mechanism. In none of our cases has there been a haemangiomatous element to the lesion nor have we been able to show feeder vessels as described by Berry. While we in no way dismiss the suggestion that haemangiomas may account for some of these lesions, we would reiterate our belief that other mechanisms may be causative, especially effete larval granulomas. The capsule of intrahepatic granulomas containing nematodes has very similar histological appearances to those that we have described in "solitary necrotic nodule".⁵ We suggest that several regressing benign lesions of the

liver may show appearances identical with the lesions we have described and that these nodules may on occasion be multiple. Perhaps the term "fibrosing necrotic nodule" of the liver might be more appropriate.

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- 1 Shepherd NA, Lee G. Solitary necrotic nodules of the liver simulating hepatic metastases. *J Clin Pathol* 1983;36:1181-3.
- 2 Berry CL. Solitary necrotic nodule of liver; a non-existent lesion. *J Pathol* 1985;146:263A.
- 3 Berry CL. Solitary "necrotic nodule" of the liver; a probable pathogenesis. *J Clin Pathol* 1985;38:1278-80.
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Professor Berry comments:

I am sorry that the picture of a feeder vessel

in my paper¹ did not convince you—the mounted version was thought to be convincing at the Pathological Society where it appeared as a poster.² It also seems to have convinced those colleagues who have sent me similar examples (with feeder vessels) (figs 1 and 2). Figure 1 is from such a lesion in the kidney (the structure to the right is a vessel) which was otherwise identical with the liver lesion and which contains a reticulin pattern in its centre rather like that seen in Shepherd and Lee's original report.³

Morphologically I have found these lesions to be slightly raised from the surface as they now describe (I do not agree that metastases are flush with the liver surface). I have found feeder vessels in all lesions I have examined and a pattern of "reticulin" in the body of the mass suggesting a collapsing vascular field.

In a survey of 1500 livers, sliced at 1 cm intervals, I found no parasitic lesion,⁴ although hamartomas, adenomas, diffuse hamartomatosis, ectopic haematopoiesis, haemangiomas, cysts, etc, were all represented. It is interesting to note Shepherd and Lee's adherence to larvae as a cause of these lesions; I am sure they look forward to finding some evidence to support their contention.

Figure 1 A 3 cm in diameter lesion was found in the kidney which was resected together with an adrenal adenoma. The feeder vessel is to the right. (Elastic van Gieson.)

Figure 2 Part of the wall of the feeder vessel "deeper" in the lesion. Elastic lamellae are seen to the left and right of small vascular channels. (Elastic van Gieson.)

Solitary necrotic nodule slightly protruding from liver surface.

- 1 Berry CL. Solitary "necrotic nodule" of the liver; a probable pathogenesis. *J Clin Pathol* 1985;38:1278-80.
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- 3 Shepherd NA, Lee G. Solitary necrotic nodules of the liver simulating hepatic metastases. *J Clin Pathol* 1983;36:1181-3.
- 4 Berry CL. Liver lesions in an autopsy population. *Hum Toxicol* 1987;6:209-14.
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Prevalence of HTLV-I in Zimbabwe

The letter by Emmanuel *et al* is of tantalising interest but requires definitive confirmation.¹ Seroprevalence studies for HTLV-I have been conducted in different parts of the world including the Caribbean,^{2,3} Papua New Guinea,⁴ and West Africa.⁵ An extensive survey from Southern Africa would have enlightened us further as to the global distribution of this pathogen. Dr Emmanuel found four (out of 900) samples to be reactive by ELISA (Dupont), "in patients sent for HIV exclusion". Samples from only two patients were also positive by Serodia-ATLA test. While the Serodia-HIV assay is used as a confirmatory anti-HIV test, the Serodia anti-ATLV or HTLV-I (-II) assay is indeed a sensitive screen test but is of low specificity, even without regard to the problems of some cross-reaction between HIV and HTLV-I⁶ or interference of ELISA reactivity from malaria and immune complexes.^{7,8}

In 1988, at the North London Blood Transfusion Centre, we screened over 6000 individual samples using the Serodia-ATLA kit. Out of 4136 routine blood donors, 49 (1 in 80) were screen test positive when tested in accordance with the manufacturer's criteria. Even higher Serodia-ATLA screen test positive rates of 1 in 50 (51:2376) were found in samples collected for malaria antibody testing—that is, donations from people who come from or have travelled to areas endemic for malaria or tropical areas. All but one, however, were reported as HTLV-I negative by the Virology Reference Laboratory at The Middlesex Hospital, London, indicating the high incidence of false positive results with this test at the recommended screening dilutions.

When reference laboratories are readily available, the indirect immunofluorescence assay may be used for confirmation.⁹ We recognise that confirmation of HTLV-I seropositivity is, at present, still a problem. Obviously PCR and RIPA would be outside the range of many laboratories, especially in developing countries, but we would like to reinforce the following point: as is the case with anti-HIV testing, samples reacting by ELISA or by particle agglutination screening tests, especially in relation to anti-HTLV, require extensive confirmatory testing. In the absence of such testing, the clinical importance of four samples reactive by ELISA in Zimbabwe remains unclear.

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breast carcinomas and concurrent chronic lymphocytic leukaemia.

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- 1 Misselevitch I, Fradis M, Podoshin L, Barel E, Boss JH. Giant cell arteritis of the tongue associated with squamous cell carcinoma. *J Clin Pathol* 1989;42:1001-5.
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Dr Boss *et al* comment:

The comments on our observation of giant cell arteritis of the tongue associated with squamous cell carcinoma are relevant at a time when fungal diseases in the immune compromised cancer patient are being recognised more frequently. Moreover, for as yet unknown reasons, fungi have a special affinity for blood vessels and we have previously encountered double fungal infestation of the pulmonary circulation.¹ The bizarrely distorted elastic lamellae of the arteries are a characteristic feature of, among others, mycotic vasculitis. In our laboratory it is routine custom to request special stains for acid fast bacteria and fungi whenever giant cell granulomatous lesions are found, whether affecting a vessel or otherwise. In the patient we described no micro-organisms were detected in the affected branch of the lingual artery.

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Arteritis of the tongue

We were interested to read the letter by Misselevitch *et al* on giant cell arteritis of the tongue associated with squamous cell carcinoma.¹ The factors they enumerate for elastic lamina injury are also important in the establishment of fungal infection in the mouth.² We have observed several cases of fungal arteritis of the head and neck in patients with cancer; such an arteritis can look deceptively like giant cell arteritis, especially if special stains for fungi are not used. The illustration shows an example of a Mucor arteritis of the head and neck in a patient with bilateral

(v) *Mucor* (arrows) and giant cells in inflamed vessel wall (Haematoxylin and eosin.)

(g) Elastica fragments in the giant cells (Elastica van Gieson.)

(f) *Mucor* hypha (arrow) emerging from a break in the elastica (Elastica van Gieson.)