



Ultrasound for the diagnosis of HIV-associated tuberculosis

To the Editor: A recent letter in the *Journal*¹ suggested that ultrasound of the abdomen might be an appropriate way to diagnose tuberculosis (TB) when sputum smears are repeatedly negative for TB and in febrile patients known or suspected to be HIV-positive. The authors describe ultrasound findings they regard as pathognomonic of TB but do not give the number of scans performed or the yield of helpful scans. We have presented such an analysis with regard to microbiological investigations for TB,² and provide data for abdominal ultrasound.

In our case series of 141 consecutive HIV-positive patients with proven TB, seen between October 1994 and September 1996 at Somerset Hospital, Cape Town, 35 (25%) had an abdominal ultrasound. This high figure relates in part to a concurrent study at Somerset Hospital of abdominal pain in HIV-infected patients.³ TB was found to be the leading cause of abdominal pain and our study focused on the diagnostic approach to TB, leading to overlap. However, while ultrasound was performed in 12 of 17 patients with TB and abdominal pain, the majority of scans were performed to look for disease processes other than TB that might have contributed to pyrexia. As an aside, all scans were normal or reflected the likely presence of TB — indicating that the use of ultrasound to exclude concomitant non-tuberculous pathology was not warranted.

To investigate whether ultrasound can be an appropriate tool in the diagnosis of TB we applied our previously published diagnostic algorithm to the 35 patients with ultrasound scans (Fig. 1, Table I). This approach is compatible with that of Emby and Hunter¹ who only advocate use of ultrasound if initial

microbiological investigations are negative. The algorithm we originally published has been modified here by adding ultrasound of the abdomen as the diagnostic investigation of choice in patients with negative sputum smears who do not have pleural effusion or who are negative on lymph node biopsy.

Scans were classified as normal, reporting intra-abdominal lymphadenopathy, or abnormalities of the liver or spleen according to the radiologist's report. When a pericardial effusion was suspected on clinical grounds the request form for the abdominal scan also recorded the need to examine the heart in the manner suggested by Emby and Hunter. In 2 of the 10 patients with intra-abdominal lymphadenopathy the radiologist examined the heart and both scans demonstrated a pericardial effusion, confirming the clinical assessment. Of the 9 patients with other abdominal abnormalities 2 requests for ultrasonographic assessment of the heart yielded 2 further pericardial effusions. However, owing to the directed nature of these assessments we cannot comment on whether all abdominal scans should be accompanied by an assessment of the heart, as implied by Emby and Hunter. Many radiographers might be reluctant to assess patients in this way routinely.

Table I shows for each category of abdominal scan the

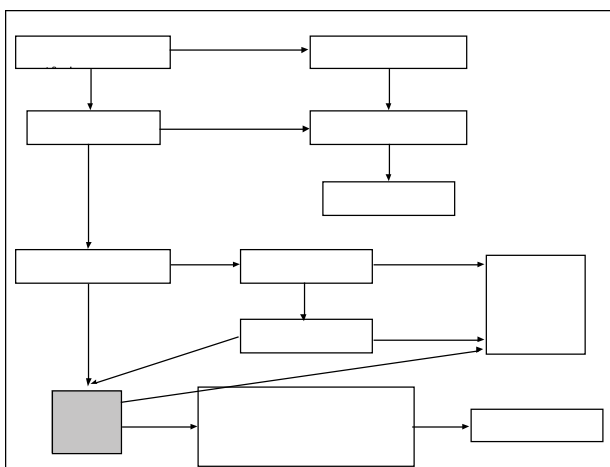


Fig. 1. Diagnostic algorithm for HIV-infected patients with suspected tuberculosis, modified from Hudson et al.² to include abdominal ultrasound.

Table I. Eligibility for scan had algorithm been followed in patients with HIV-associated tuberculosis, Somerset Hospital

	Abdominal ultrasound result		
	Normal (N = 16)	Intra-abdominal lymphadenopathy (N = 10)	Abnormal liver or spleen (N = 9)
Patient eligible (N)			
Diagnosed on urine culture	1	-	-
Diagnosed on sputum culture	-	1	1
Diagnosed on liver biopsy	-	-	1
Patient ineligible (N)			
Not anaemic (Hb > 11.0 g/dl)	2	-	-
Sputum smear positive	3	2	5
Peripheral nodes suitable for biopsy	10	7	1
Pleural effusion	-	-	1



number of patients for whom the scan was appropriate or not in terms of the diagnostic algorithm. It can be seen that the number of scans could have been reduced to 6. Two of the 16 negative scans could have been avoided if a policy was in force of only scanning patients with anaemia (a surrogate marker of dissemination).

In conclusion, there is a limited role for ultrasound in the diagnosis of TB. However, at a time of crisis in academic and rural medicine in South Africa, calls for use of existing investigations in new ways should be qualified by careful cost-benefit analysis. We should also point out that our study was performed in an HIV-positive population at very high risk of

TB.⁴ Experience elsewhere may differ, possibly resulting in ultrasound being even less useful.

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1. Emby DJ, Hunter M. The value of ultrasound in the HIV-infected patient with a fever of undetermined origin. *S Afr Med J* 2002; **92**: 566.
2. Hudson CP, Wood R, Maartens G. Diagnosing HIV-associated tuberculosis: reducing costs and diagnostic delay. *Int J Tuberc Lung Dis* 2000; **4**: 240-245.
3. O'Keefe EA, Wood R, Van Zyl A, Carlem AK. Human immunodeficiency virus-related abdominal pain in South Africa. Aetiology, diagnosis and survival. *Scand J Gastroenterol* 1998; **33**: 212-217.
4. Wood R, Maartens G, Lombard C. Risk factors for developing tuberculosis in HIV-1 infected adults from communities with a low or very high incidence of tuberculosis. *J AIDS* 2000; **23**: 75-80.

Massive hepatomegaly due to visceral leishmaniasis

To the Editor: In 1908 Leishman and Donovan¹ described the protozoan *Leishmania donovani* in splenic tissue. Almost a century later leishmaniasis has emerged in new regions and new settings.¹ Recent interest in the disease has been prompted by recognition of cases in returning US Gulf War veterans and people with HIV infection.² We describe a case of visceral leishmaniasis diagnosed on bone marrow aspirate in a patient presenting to a tertiary hospital in KwaZulu-Natal.

A 42-year-old Mozambican national, who works as a marine merchant, was referred from a local hospital with fever, hepatosplenomegaly and pancytopenia. He had a 6-month history of gradual weight loss of approximately 10 kg with intermittent fever and rigors. He was treated for hepatic tuberculosis in a Mozambican hospital. A liver biopsy was not done before commencement of antituberculosis therapy and the patient did not improve after completion of this treatment.

He had been treated for malaria several years previously while living in Mozambique. He travelled to Brazil, Argentina, Italy and Portugal between 1995 and 1998.

On examination, the patient was febrile (39.8°C). He was pale and had no lymphadenopathy. He had a hepatomegaly that extended 10 cm below the costal margin and a 3 cm splenomegaly.

The full blood count showed haemoglobin 8 g/dl (normochromic, normocytic anaemia), platelets $132 \times 10^9/l$ and white blood cell count $1.8 \times 10^9/l$. Liver function tests revealed a hyperglobulinaemia. He had an erythrocyte sedimentation rate (ESR) of 113 mm/h, and his urea and electrolytes were normal. HIV and hepatitis screens were negative. The chest radiograph was normal. Ultrasound of the abdomen detected no additional abnormalities. A bone marrow aspirate and trephine were done in the first instance to investigate the pancytopenia. A liver biopsy was scheduled, but this was deferred when the results of the bone marrow aspirate and

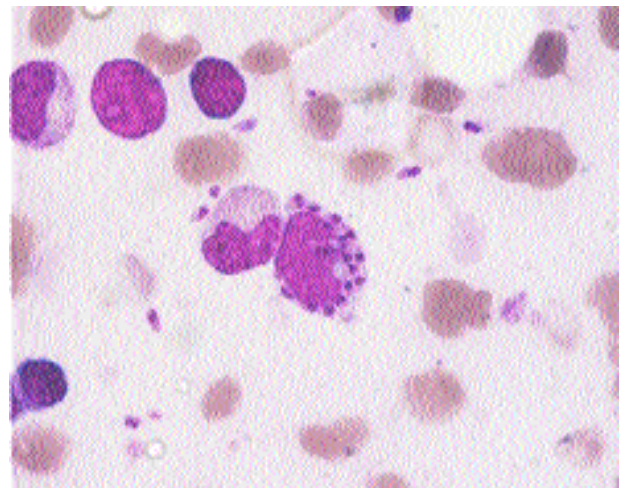


Fig. 1. Bone marrow aspirate — Gram stain showing intracellular 'dot-like' microorganisms (magnification 1 000). Courtesy of Dr P Sturm.

trephine were received. Fig. 1 is a photomicrograph of the marrow aspirate showing the characteristic intracellular amastigotes of leishmaniasis. This was confirmed on the trephine biopsy.

The patient was treated with intravenous amphotericin B (60 mg/kg/day) for 20 days. Renal impairment and thrombophlebitis complicated his therapy. The renal function improved when amphotericin B was stopped. At 3 months after treatment the hepatosplenomegaly had resolved and a repeat bone marrow aspirate was normal.

Discussion

This case highlights the importance of considering exotic diseases associated with common clinical presentations, given