

# A professional kitesurfer with multiple liver lesions

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## Clinical record

A 35-year-old British man was admitted with a 2-week history of abdominal discomfort, fatigue and intermittent high fevers associated with drenching night sweats. He reported having returned to Australia 2 months before presentation from a 12-year around-the-world trip, having travelled extensively for 6 months through South-East Asia and northern Australia. Before that, he had been to Argentina, the Maldives, Egypt, India and Cambodia, where he had worked as a professional kitesurfing instructor.

His past medical history was unremarkable and he was not on regular medication. His social history revealed intravenous heroin addiction in his early 20s. He had consumed more than 80 g of alcohol daily for a number of years, with occasional binges, until recently, when drinking alcohol provoked nausea and vomiting.

The patient appeared unwell with a tympanic temperature of 40°C. He was fair-skinned and of muscular build. Physical examination showed conjunctival jaundice but no evidence of needle tracks, rashes, finger clubbing, lymphadenopathy or suspicious cutaneous lesions. Tender hepatomegaly was noted, but no ascites or splenomegaly. Findings of cardiac, respiratory and neurological examinations were normal.

Abnormal laboratory findings on admission were: albumin concentration, 29 g/L (reference range [RR], 33–47 g/L); alkaline phosphatase titre, 164 U/L (RR, 30–115 U/L);  $\gamma$ -glutamyl transferase titre, 207 U/L (RR, 0–45 U/L); alanine aminotransferase titre, 76 U/L (RR, 0–40 U/L); aspartate aminotransferase titre, 279 U/L (RR, 0–40 U/L); lactate dehydrogenase titre, 4268 U/L (RR, 100–225 U/L); white cell count,  $13.4 \times 10^9/L$  (RR,  $4.0$ – $11.0 \times 10^9/L$ ); absolute neutrophil count,  $9.7 \times 10^9/L$  (RR,  $1.5$ – $6.0 \times 10^9/L$ ). There was no evidence of thrombocytopenia, renal dysfunction or coagulopathy. Findings on abdominal ultrasonography the day before admission were reported to be normal.

Despite there being few clinical features to suggest a source of sepsis apart from the hepatomegaly, because of his travel history and previous history of injecting drug use, a wide variety of infectious diseases, including subacute bacterial endocarditis, typhoid, malaria, and viral hepatitis, were all initially considered in the differential diagnosis. Multiple sets of blood cultures, urine and stool samples were sent for microscopy, culture and sensitivity. Three sets of thick and thin films for malaria as well as a test for *Plasmodium falciparum* antigen were negative.

In view of the clinical hepatomegaly, a computed tomography (CT) scan of the abdomen was performed, which was reported as strongly suggestive of microabscesses (Box). The presumptive diagnosis at this stage was a tropical pyogenic liver abscess, although military tuberculosis and candidiasis were also considered.

Intravenous therapy with flucloxacillin, ceftriaxone, ciprofloxacin and metronidazole was initiated. The ceftriaxone was subsequently changed to meropenem, and the ciprofloxacin ceased 5 days after there had been no growth on any cultures.

Serological tests for a wide variety of pathogens and diseases were also performed, including: HIV 1 and 2; hepatitis viruses A, B and C; cytomegalovirus; Epstein–Barr virus; Q fever; brucellosis; cryptococcosis; *Entamoeba histolytica*; Ross River virus; Dengue virus; flavivirus; leptospirosis; schistosomiasis; cysticercosis;

Coronal reconstruction computed tomography image of the abdomen showing hepatomegaly with numerous low-density lesions scattered throughout both lobes of the liver



and melioidosis. Results for all of these eventually returned negative.

Despite the antibiotics and fluids given intravenously, the patient's condition deteriorated over the course of 6 days and he developed hepatic encephalopathy, ascites, pleural effusions and peripheral oedema. He also continued to spike high temperatures daily, but repeated blood cultures were sterile. A CT scan performed a week after the initial CT scan suggested enlargement of the liver lesions.

Although hepatic abscesses were still considered most likely, given the markedly elevated lactate dehydrogenase titre and lack of clinical improvement with broad-spectrum antibiotic therapy, alternative diagnoses, in particular malignancy, were also considered. We therefore performed a core biopsy of the liver under ultrasound guidance. There was no evidence of pus, and the lesional tissue showing strong monoclonal antibody staining against Melan-A and HMB-45 confirmed liver infiltration by a poorly differentiated malignant melanoma.

Repeat physical examination included dilated pupil fundoscopy, which showed a brown, dome-shaped subretinal lesion just below the left optic disc, most suggestive of a primary choroidal melanoma. The patient said that he never used sunglasses while kitesurfing.

## Discussion

Exposure of the unprotected eye to sunlight or sunlamps is an important risk factor for the development of intraocular melanoma.<sup>1</sup> The incidence of ocular melanoma in dark-eyed individuals is lower, probably because they are less sensitive to solar radiation, or less is transmitted to the choroids.<sup>2</sup>

Uveal melanoma (affecting the iris, ciliary body, and choroids) is the most common primary intraocular malignancy in the Western world, affecting six to eight adults per million each year. Although fewer than 2% of patients show evidence of metastatic spread at presentation, over 40% will eventually die from widespread disease.<sup>3</sup>

Most intraocular melanomas are initially asymptomatic. Tumour enlargement may then cause distortion of the pupil (iris melanoma), blurred vision (ciliary body melanoma), or decreased visual acuity caused by either central growth close to the macula or secondary retinal detachment (choroidal melanoma). Because the uveal tract is a vascular structure without lymphatic channels, tumour spread occurs primarily by either local extension or by haematogenous dissemination. The first site of systemic metastases is the liver, although spread to other organs such as lung, bone, and subcutaneous sites have been described.<sup>3</sup>

Metastasis of melanoma to the liver, although rare, can produce a dramatic initial presentation with fulminant hepatitis, shock, and multisystem organ failure.<sup>4</sup> An elevated lactate dehydrogenase titre is one of the most predictive factors for metastatic spread and decreased survival in patients with malignant melanoma, with a sensitivity of 79% and specificity of 92% in detecting disease progression to stage IV melanoma.<sup>5</sup>

Extraocular extension and metastatic spread are associated with an extremely poor prognosis, and response rates with contemporary single-agent chemotherapy are generally below 10%. A recently published study investigated the use of chemotherapy with intra-arterial hepatic fotemustine.<sup>6</sup> Median survival rates were among the longest reported, with an overall response rate of 36%, a median overall survival of 15 months, and a 2-year survival rate of 29%.

Our patient was scheduled for three cycles of intravenous fotemustine therapy, but developed significant tumour lysis syndrome with intractable hyperkalaemia. He died shortly after the second cycle, only 28 days after being admitted to hospital. The

final diagnosis was hepatic failure secondary to metastatic melanoma from an intraocular primary melanoma. An autopsy was not performed.

### Competing interests

None identified.

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