The diving doctor's diary

Eosinophilic meningitis presenting as decompression illness

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Key words
Decompression illness, meningitis, eosinophilia, nematodes, case reports

Abstract
An experienced diver presented with suspected decompression illness (DCI) 17 days after an uncontrolled ascent from a 33 m dive carried out whilst on a tropical holiday. He had progressive symptoms of general malaise, headache, irritability, impaired mentation and facial, shoulder and leg pains and itchiness. A raised eosinophil count in blood and cerebrospinal fluid led to a diagnosis of eosinophilic meningitis rather than DCI, and this was confirmed by subsequent serology positive for the rat lung nematode worm *Angiostrongylus cantonensis*. Recovery was slow, with persistent symptoms still present at 18 months. This case illustrates the diagnostic difficulties where care givers in a traveller's home region are not versed in either diving or tropical medicine.

Introduction

Travel to tropical regions is never without health risks. When this is combined with diving activities, the development of symptoms in the traveller may present diagnostic problems, particularly where care givers in the patient's home region are not versed in either diving or tropical medicine. The following case illustrates these difficulties and the message that all post-diving symptoms are not due to decompression illness (DCI), even when the diving profile is a provocative one.

Case report

During a holiday in Tonga, a 48-year-old experienced diver undertook several dives. His final dive lasted 22 minutes to a maximum depth of 33 metres. He became low on air, panicked and made a rapid ascent to the surface despite the efforts of his buddy to restrain him. He developed a left-sided headache immediately after the dive, but was otherwise asymptomatic.

That evening he felt unwell and hot ("flu-like"). These symptoms persisted and he developed itchiness in the shoulders, right arm and both legs. He flew home to New Zealand three days after the dive, during which flight epigastric and central low thoracic back pain developed associated with bloating, anorexia and constipation unrelieved by ranitidine. He developed a rash thought to be due to this drug. A week later, he was admitted to the district hospital where he was described as anxious and depressed. Gastroscopy revealed a small gastric ulcer and he was discharged after five days on omeprazole.

Over the next few days, he developed worsening right leg pain. The severe headache persisted, associated with facial pain and tenderness and he had some photophobia. He became increasingly "jittery" and his wife reported slowed mentation and nocturnal hallucinations. He was readmitted to hospital, where neurological examination was normal, including normal fundi, and a CT scan of the brain was negative. The only abnormal investigations were GGT 102 IU.l^{-1} (normal range 0–50), ALT 91 IU.l^{-1} (normal range 0–50), eosinophilia 1.29 x 10^{9}.l^{-1} (normal range <0.51) and an abdominal ultrasound showing a "fatty liver".

At this stage, because of the provocative diving history, the patient's wife raised the possibility that his symptoms were diving related and he was referred to the regional hyperbaric centre 17 days post-dive. On admission, he had slow mentation with a Minnisota Mini-Mental Score of 26/30, mild neck stiffness, diminished pin-prick sensation over the anterior right thigh in the L2/3 distribution, a limping gait and sharpened Romberg's test of 15 seconds, though heel-toe walking was normal. There were no other neurological signs. The right knee and ankle and left wrist were slightly swollen and painful on movement, physical examination otherwise being unremarkable.

DCI was considered to be most unlikely but he was given a single short oxygen treatment (RAH Table 18.60.30) with no benefit. He was then referred to the Infectious Diseases service. MRI scan of the brain was unremarkable. Blood screen demonstrated a normal white count but with an eosinophilia of 1.8 x 10^{9} l^{-1}. A clinical diagnosis of eosinophilic meningitis was made. Lumbar puncture showed normal CSF pressure with a turbid aspirate. Globulins (CSF protein 1.63 g.l^{-1}; normal range 0.15–0.40) were moderately elevated, and white cell count was 493 x 10^{6}l^{-1} with 35% eosinophils. Gram stain was negative and there was no growth after six days.

Serology sent to the Institute of Clinical Pathology, New South Wales, was subsequently positive for *Angiostrongylus*

cantonensis, confirming the diagnosis of eosinophilic meningitis.

The patient continued to suffer severe chronic pain problems requiring referral to the regional pain centre. He gradually improved over the following year or so, but persistent upper limb girdle pain and recurrent headaches were still present at 18 months' follow up.

Discussion

Angiostrongylus cantonensis is a rat lung nematode worm with a complicated life cycle involving slugs or snails and the rodent central nervous system.[1] It may enter the food chain when the snails or rats are eaten by carnivores including fish or freshwater prawns. Human infection has been recorded throughout the western Pacific and parts of the Far East. Man is not a normal host but may become infected by eating third stage infective larvae in the normal intermediate host or other paratenic hosts that have not been correctly cooked. The incubation period ranges from 1 to 15 days.

Symptoms develop when the worm migrates across the blood-brain barrier, causing an inflammatory meningitis or meningo-encephalitis. Acute severe occipital or bitemporal headache, neck stiffness, paraesthesiae of the limbs and visual impairment are the typical symptoms. The pathological changes are caused by dead and degenerating worms and the inflammatory response to these.

This is a self-limiting infection lasting four to six weeks with a low mortality. Antihelminthic agents are of no clinical benefit and pain relief is difficult and may require opioids. Steroids are also of no proven benefit but often given in desperation as severe pain syndromes may occur requiring referral to specialist pain clinics as in this case.

DCI may be mimicked by a variety of conditions or by drug side effects. Mefloquine is not advised for use by divers travelling to malarial-risk areas because of its central nervous system side effects which may be confused with symptoms of DCI. In the past decade, we have seen a number of patients referred for suspected DCI in whom the diving activity was either coincidental or only contributory. Their diagnoses have included viral meningitis and myocarditis, atherosclerotic cerebrovascular disease, atypical or pseudoepilepsy, migraine with visual disturbances, inner ear barotrauma and musculo-skeletal injuries.

Whilst DCI cannot be excluded as a dual pathology in this case, it would seem unlikely given his subsequent course. Such diagnostic dilemmas reaffirm the need for thorough medical assessment of all divers with suspected DCI.

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References

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Obituary

Ichiro Nashimoto, 1935–2002

Dr Nashimoto is probably not well known to the majority of SPUMS members. However, he was a long-time researcher and active medical doctor in diving, tunnel and caisson work and hyperbaric medicine in Japan and the Far East. Even back in the 1960s he was running a clinical hyperbaric facility at the Tokyo Medical and Dental University. He contributed to many publications and articles, including co-editing with Ed Lanphier two Undersea Hyperbaric Medicine Society (UHMS) workshops on decompression illness in 1987 and 1991. He was a tireless worker in trying to improve the safety of caisson workers and harbour divers including the development of decompression tables.

He is recalled by several members of UHMS as a delightful host, renowned for his "night diving" pub tours, and as a kind and jovial man.

Professor Nashimoto died in May 2002 after a short fight with liver cancer.