

Angiostrongylus meningitis associated with intraparenchymal cerebral haemorrhage

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Abstract

Angiostrongylus cantonensis (*A. cantonensis*) is a nematode parasite found in Southeast Asia, Australia and the Pacific that is the most common cause of eosinophilic meningitis.

We report a case of intraparenchymal cerebral haemorrhage associated with *A. cantonensis* meningitis. This complication has not previously been reported in the literature.

Angiostrongylus cantonensis (*A. cantonensis*) is a nematode worm that is the most common cause of eosinophilic meningitis (see Figure 1). It is principally found in Southeast Asia, Australia and the Pacific.¹⁻³ Recent media attention regarding the difficulties in diagnosing *A. cantonensis* meningitis highlights the need for medical practitioners to be aware of the disease.⁴

Figure 1. *Angiostrongylus cantonensis*



Source: Wikimedia Commons. Lindo JF, Waugh C, Hall J, et al. Enzootic *Angiostrongylus cantonensis* in rats and snails after an outbreak of human eosinophilic meningitis, Jamaica. *Emerging Infectious Diseases* 2002;8(3):324–326.

Case report

A 51-year-old man of Tongan ethnicity presented to the Emergency Department with a 3-week history of right-sided headache and two episodes of nausea and vomiting on the day of presentation. The headache was constant with variable severity and exacerbations to 9 out of 10 primarily experienced in the right peri-auricular occipital and temporal areas. There was no history of trauma, hearing loss, ear discharge, photophobia, or rash.

Two weeks before presentation he had returned from Tuvalu where he had been living for the preceding 3 months. He had seen his family doctor three times for this illness and had been treated for a presumed viral illness with paracetamol.

His past medical history includes obesity, hypertension, type 2 diabetes mellitus, hyperlipidaemia and gout. His regular medications are metformin 1 g BD, Quinapril 20 mg BD, simvastatin 40 mg nocte and felodipine ER 10 mg mane.

On examination he was afebrile and other vital signs were normal. He was alert and orientated. Examination of the cardiovascular, respiratory and abdominal systems was normal. His neurological exam was normal including a normal cranial nerve exam and there were no signs of meningism. He had no skin lesions or rashes. There was no lymphadenopathy.

A CT of the head was performed to exclude an intracranial haemorrhage in the emergency department. There was no haematoma present but fluid in the right mastoid cavity was noted. He was referred to otorhinolaryngology (ORL) for presumed mastoiditis.

On ENT review his left ear appeared normal but the right tympanic membrane was moderately retracted with evidence of effusion. There was no erythema overlying the mastoid and no mastoid tenderness. He was treated with antibiotics for presumed acute otitis media. However he then proceeded to have a seizure-like episode and was referred to the general medicine department.

Relevant blood tests that were performed are shown in Table 1.

Table 1. Blood test results

Blood test	Result	Reference range
White blood count	14.53	4–11 (E+9/L)
Neutrophils	10.61	1.9–7.5 (E+9/L)
Eosinophils	0.78	0–0.5 (E+9/L)
CRP	16	0–5 (mg/L)
ESR	5	0–20 (mm/hour)
HbA1c	41	20–40 mmol/mol

A lumbar puncture was performed with an opening pressure of 54cm (n 10–20cm) and the CSF was clear and colourless. The CSF contained 270 WBC $\times 10^6/L$ (7%

polymorphs, 70% lymphocytes, 5% monocytes, 18% eosinophils) and a glucose of 3.2 mmol/L (normal 2.8–4.4 mmol/L) and protein of 0.73 g/L (normal 0.15–0.45 g/L).

No organisms were seen on Gram stain and there was no growth after 5 days incubation.

CSF histology and cytology revealed a lymphocytosis and eosinophilia with no evidence of malignant cells. *Mycobacterium tuberculosis* DNA was not detected on nucleic acid amplification assay nor on Ziehl-Neelsen staining.

The CSF was negative for *Streptococcus pneumoniae* on immunochromatographic test. The CSF was also negative for *Enterovirus* and *Cryptococcal antigen*. *A. cantonensis* serology is pending.

An LP was performed 7 days later. The CSF contained 140 WBC $\times 10^6$ /L (1% polymorphs, 60% lymphocytes, 4% monocytes, 35% eosinophils) and a glucose of 2.6 mmol/L (normal 2.8–4.4 mmol/L) and protein of 0.6 g/L (normal 0.15–0.45 g/L).

His symptoms improved immediately following both lumbar punctures. MRI showed no evidence of dural venous sinus thrombosis.

Based on the history, the elevated WBC with eosinophilia, the CSF showing a high WBC with eosinophilia and increased protein, a diagnosis of *Angiostrongylus cantonensis* eosinophilic meningitis was made.

Unfortunately he presented 3 days following discharge with decreased GCS and right arm weakness. CT and MRI revealed a 7 \times 2.5 \times 3cm strip of haemorrhage within the left temporal lobe. There was intraventricular blood with mild hydrocephalus and midline shift to the right of around 12 mm. There was no mass lesion, vascular malformation, or venous thrombosis identified as a cause for the haemorrhage (see Figure 2).

Figure 2. CT of patient's head showing acute left temporal haemorrhage with intraventricular blood



Discussion

This is the first reported case of *A. cantonensis* meningitis complicated by an intraparenchymal cerebral haemorrhage. In 1982, Kliks et al. reported one case of subarachnoid haemorrhage presumed to be due to *Angiostrongylus* meningitis, but case reports and reviews of *Angiostrongylus* meningitis have not revealed any other cases complicated by intracranial haemorrhage.^{5,6}

Diagnosis of *Angiostrongylus* meningitis is based on a history of possible exposure e.g. consumption of unwashed lettuce in an endemic region, clinical findings and CSF eosinophilia.¹ Definitive diagnosis, such as serologic tests, are available but are rarely used due to price and poor specificity. Direct identification of the *Angiostrongylus* parasite in humans has not been described in the literature.¹

The main differential diagnosis of parasitic eosinophilic meningitis is *Gnathostoma* meningitis. This usually presents with more severe disease and may affect the viscera and skin along with the CNS, elevated opening pressures are less common and imaging may reveal nodular lesions, CNS haemorrhage or hydrocephalus.^{1,6-8} However *Gnathostoma* meningitis is not found in the Pacific and therefore it would be very unlikely to be the cause of this patient's haemorrhage.¹

Another possibility is that consequences of the metabolic syndrome caused this patient's intracranial haemorrhage, however he does not have evidence of ischaemic heart disease or cerebrovascular disease.

This case highlights the difficulty of diagnosing this condition. The patient was reviewed a number of times prior to presentation and treated for a presumed viral illness. Furthermore, incidental findings on the first set of imaging distracted the consulting physician from the diagnosis. Therefore it is important that medical practitioners in the primary and hospital sectors have a broad differential diagnosis for any unwell returned traveller.

Learning points:

- Develop a broad differential diagnosis for unwell returned travellers
- Think of diagnoses outside of your specialty when assessing patients
- The commonest cause of eosinophilic meningitis is a parasitic infection

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