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Case Report

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Neuroangiostrongyliasis presenting as abdominal allodynia mimicking hepatobiliary disease in urban Australia

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Abstract

Introduction **Angiostrongylus cantonensis is a neurotropic helminth and the leading cause of eosinophilic meningitis in humans worldwide. Infection usually follows ingestion of larvae in contaminated produce or intermediate hosts.**

Case description **A patient in their 70s from urban Sydney, New South Wales presented to an Emergency Department four times in 10 days with right upper quadrant pain and bilateral lower limb pain. Abdominal ultrasound showed cholelithiasis, but inflammatory markers remained unremarkable and further abdominal investigations were non-diagnostic. Focal allodynia and fluctuating peripheral eosinophilia were present early. The patient subsequently developed severe headache, confusion and meningism. MRI demonstrated leptomeningeal enhancement and repeat cerebrospinal fluid analysis confirmed eosinophilic meningitis. Exposure history identified gardening and frequent consumption of home-grown leafy greens. CSF PCR and serology confirmed locally acquired *Angiostrongylus cantonensis*. The patient's allodynia improved after a short course of prednisolone, while neurocognitive function recovered gradually over 12 months.**

Discussion **This case highlights that atypical abdominal pain with focal allodynia and peripheral eosinophilia, in regions where *A. cantonensis* is established, should prompt early consideration of neuroangiostrongyliasis. History should assess gardening activity, snail and slug contact, and the ingestion of raw leafy greens.**

Learning points

- **Early neuropathic pain from neuroangiostrongyliasis may localize to the abdomen and mimic intra-abdominal pathology before meningitic features develop.**
- **A targeted exposure history that includes gardening, contact with snails and slugs, and consumption of home-grown raw leafy greens is critical to identifying plausible *A. cantonensis* exposure.**
- **Public health messaging in Australian regions where *A. cantonensis* is established should emphasize the importance of thoroughly washing leafy greens before consumption.**

Keywords Neuroangiostrongyliasis, Angiostrongylus cantonensis, Eosinophilic meningitis, Helminth infection, Peripheral eosinophilia, Rat lungworm

Background

Angiostrongylus cantonensis is a neurotropic nematode and the most common infectious cause of eosinophilic meningitis in humans [1]. In endemic settings, transmission is maintained by rats as definitive hosts and snails and other molluscs as intermediate hosts. Humans are incidental, dead-end hosts who typically acquire infection by ingesting infective third-stage larvae in intermediate or paratenic hosts, or on contaminated leafy greens [2]. After ingestion, larvae migrate into the central nervous system, triggering an inflammatory response that results in eosinophilic meningitis [3].

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Neuroangiostrongyliasis (colloquially known as Rat Lung Worm Disease) has been reported across multiple regions worldwide, including the Asia-Pacific, the Americas and parts of Africa and Europe [4]. More than 7,000 cases have been reported in the literature, but the true incidence remains uncertain and is likely underestimated [5]. Recognition is constrained in many settings by limited access to serological or molecular testing and by the absence of routine surveillance systems [4, 6]. As a result, the clinical and epidemiological spectrum remains incompletely characterized. In addition, clinician familiarity may be limited, especially where locally acquired infection is thought to be uncommon [7]. As such, diagnosis of neuroangiostrongyliasis can be difficult, particularly when early symptoms are non-specific or mimic more common conditions [8].

Case presentation

A patient in their 70s presented to a metropolitan emergency department in New South Wales, Australia on four occasions over 10 days with right upper quadrant abdominal pain and bilateral lower limb pain. Despite repeated evaluation, the underlying etiology became apparent only after the development of meningoencephalitis.

During the initial two presentations, the patient reported several days of intermittent right upper quadrant and bilateral lower limb pain associated with subjective fevers, fatigue, and one episode of vomiting. Their past medical history included hypertension and osteoporosis; they were a non-smoker with infrequent alcohol intake; they had no recent travel and no unwell contacts at work or home. Examination revealed right upper quadrant tenderness on palpation. No abnormalities were noted on blood tests, including full blood count, electrolytes, renal function, inflammatory markers and liver function tests. Abdominal ultrasound detected cholelithiasis, but no features of cholecystitis. Computed tomography (CT) of the abdomen and pelvis and CT pulmonary angiogram showed no acute findings. On both occasions they were discharged with symptomatic management after no acute pathology was identified although the patient's symptoms persisted.

On their third presentation, the patient again reported progressive abdominal and bilateral lower limb pain, along with new symptoms including lethargy, anorexia, frequent vomiting and mild headaches. They were admitted under the general surgery team for investigation of symptomatic cholelithiasis. Liver function remained normal; white cell count (WCC) and C-reactive protein (CRP) were not significantly elevated. Magnetic resonance cholangiopancreatography (MRCP) was negative for cholecystitis or cholangitis. The rheumatology team was consulted due to systemic symptoms and a non-specific pain pattern. On examination, light touch elicited a burning dysesthesia over the right upper quadrant and lower limbs, consistent with tactile allodynia. Antinuclear antibody, extractable nuclear antibody rheumatoid factor and creatine kinase were normal. After four days of admission, the patient improved clinically and was discharged home with a working diagnosis of a resolving viral illness with associated allodynia.

Four days later, the patient presented to the hospital for the fourth time. The patient had developed sudden severe headache associated with confusion and vomiting. They remained afebrile, but had meningism and altered mental status, with persistent allodynia over the right upper quadrant and both lower limbs. The patient was admitted under the neurology team with infectious diseases consultation.

Investigations

CT brain, angiogram, and perfusion were negative for ischemia, hemorrhage and large vessel occlusion. Contrast-enhanced Magnetic Resonance Imaging (MRI) of the brain revealed patchy leptomeningeal enhancement over the cerebral convexities and cerebellar hemispheres (see Fig. 1). MRI of the whole spine showed no spinal cord pathology.

Lumbar puncture was performed in the emergency department during the patient's fourth presentation; clear CSF was obtained with an opening pressure of 34 cmH_2O. CSF protein was elevated at 1.24 g/L, glucose was 4.2 mmol/L, WCC was 224×10^6/L with mononuclear predominance. CSF Gram stain and culture were negative. Limbic encephalitis panel was negative. CSF PCR testing for common bacterial and viral pathogens was negative, and cryptococcal antigen was negative in CSF and serum. CSF EBV PCR was positive; EBV serology was consistent with past infection (IgG positive, IgM negative).

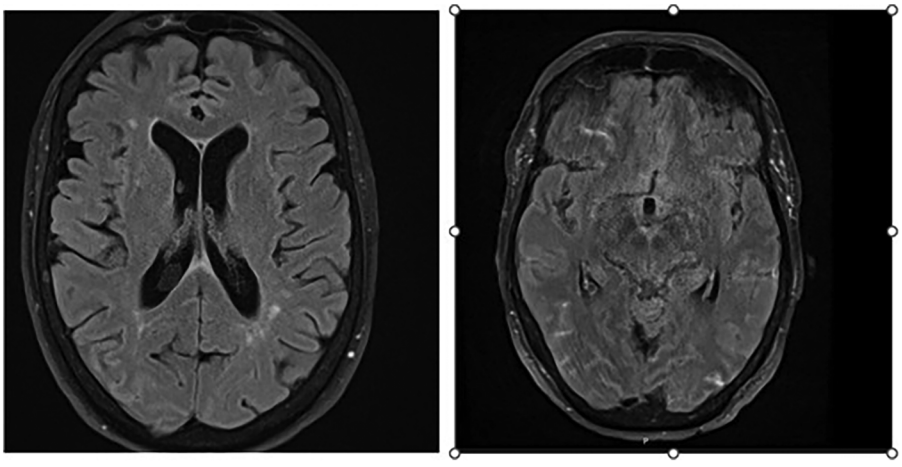

Figure 1 Brain MRI with leptomeningeal enhancement.

A repeat lumbar puncture was performed after five days due to persistence of symptoms. The CSF WCC had increased to 522×10^6/L with 24% eosinophils on cytology, consistent with eosinophilic meningitis. Peripheral eosinophilia was also observed, peaking at 1.79×10^9/L during the fourth presentation; mild eosinophilia (0.63×10^9/L) was present during the first presentation, resolved, and recurred transiently during the surgical admission.

Treatment

After presenting with altered mental status, the patient was treated empirically for infectious causes of meningoencephalitis. They were initiated on intravenous ceftriaxone, vancomycin (instead of penicillin due to a reported penicillin allergy) and aciclovir. Despite several days of empirical treatment, their symptoms persisted. Following the identification of eosinophilia in the CSF, the therapy was changed to prednisolone 50 mg daily for suspected neuroangiostrongyliasis.

Outcome

Once the CSF eosinophilia was identified, the history was revisited with a focus on epidemiological risk factors for eosinophilic meningitis. The patient's history identified a hobby of gardening and their regular consumption of home-grown lettuce without use of slug and snail control measures. This raised strong suspicion for neuroangiostrongyliasis, and prednisolone 50 mg daily was commenced for 5 days and ceased after clinical improvement. *Angiostrongylus cantonensis* infection was subsequently confirmed by positive serum serology and CSF PCR testing performed at a reference laboratory. Shortly after discharge, the patient reported a new symptom of blurred vision, prompting urgent review by an ophthalmologist; no evidence of ocular angiostrongyliasis was identified, and the symptoms were considered potentially steroid-related. At their 2-week follow-up, the patient reported the recurrence of mild headaches and difficulty multitasking, while their abdominal and lower limb allodynia had resolved. At their 6-week follow-up, the patient's headaches and cognition had improved. They reported persistent fatigue following the infection, which gradually improved over the subsequent 12 months before they returned to their premorbid level of physical and cognitive function.

Discussion

Diagnosis of *Angiostrongylus cantonensis* infection relies on a combination of epidemiological risk factors, characteristic neurological symptoms, CSF eosinophilia and, where available, molecular or serological confirmation [3, 6]. In our hospital, we utilize a local reference laboratory for confirmatory testing; results can take several weeks. Where confirmatory testing is unavailable or delayed, it is recommended that diagnosis using the above epidemiological, clinical and CSF criteria is presumptive [6].

Common presentations involve a returning traveler, or resident of an endemic region, who has ingested an intermediate or paratenic host, such as a snail, slug, prawn, frog or monitor lizard [5]. Clinically, early disease is often characterized by non-specific migratory sensory disturbances, with headache and meningism developing later as central nervous system involvement progresses [9]. Disease severity is thought to correlate with inoculum size and ranges from mild, transient sensory disturbance to severe meningoencephalitis and death [10]. Management generally focuses on controlling the eosinophilic inflammatory response with steroids, while the role of antihelminthic therapy remains uncertain because of concerns that rapid killing of larvae may worsen inflammation [11]. Recovery is often gradual over weeks to months, and prolonged symptoms, particularly neuropathic pain and fatigue, are well described [4, 12].

In our patient, the main early complaint was right upper quadrant abdominal pain. This led to a clinical suspicion of biliary pathology, supported by an ultrasound finding of cholelithiasis. Associated lower limb pain was documented but was not initially recognized as part of a unifying neurological syndrome. Initial peripheral eosinophilia was mild and transient, a pattern that is unlikely to prompt early consideration of parasitic infection in most clinical settings. Consequently, *A. cantonensis* infection was not considered during initial presentations and was only recognized when the patient developed severe meningoencephalitis. In retrospect, both abdominal and lower limb pain likely represented neuropathic hyperalgesia related to early larval migration within the nervous system. A similar pattern of diagnostic error is well recognized in herpes zoster, where pre-eruptive radicular neuralgia may be misinterpreted as an acute surgical abdomen until the characteristic vesicular rash appears [13, 14].

Abdominal pain at presentation poses a diagnostic challenge in *A. cantonensis* infection. It is likely that clinicians will consider common surgical causes, delaying consideration of neuroinfectious etiologies. Only a few case reports and small series have recorded abdominal pain in *A. cantonensis* infection, typically after substantial exposures, and usually as one feature of a broader systemic illness, rather than the main symptom [15, 16]. To our knowledge, our case of neuroangiostrongyliasis presenting with predominant abdominal pain leading to extensive surgical investigations has not previously been described.

In Australia, *Angiostrongylus cantonensis* is well established in wildlife, but the extent of associated human disease remains uncertain. Zoonotic surveys in New South Wales and Queensland have shown substantial infection burdens in urban rats, domestic dogs, native marsupials and captive wildlife [17–20], creating ongoing potential for human exposure. Human infection is not notifiable in Australia, so the true incidence is unknown [21]. Many of the most severe cases described in Australia have occurred in infants and children following deliberate or accidental ingestion of slugs or snails [21–23]. In adults, published cases include infections acquired overseas and sporadic locally acquired infections [24–26]. Overall, these data suggest that neuroangiostrongyliasis in the Australian population is either rare, or frequently under-recognized. Consequently, awareness of the condition among Australian clinicians is likely to be limited.

The suspected route of acquisition in this case has practical implications for clinical assessment and public health advice. Several outbreaks have linked neuroangiostrongyliasis to the ingestion of raw vegetables contaminated with infected snails

or slugs [27, 28]. However, some researchers have argued that larval loads in snail and slug mucus are generally low, so slime contamination of leafy vegetables may pose minimal inoculum risk [4, 29]. In our patient, daily consumption of lettuce from a home garden in a region where *A. cantonensis* is established provided a plausible exposure. However, it is unclear whether infection followed a single high-risk ingestion, or cumulative low-level exposures over time. Regardless of the exact mechanism, this indicates that clinicians should enquire about gardening activities and the consumption of home-grown leafy greens when assessing patients with possible *A. cantonensis* infection. Public health messaging in endemic areas should also highlight the risks associated with growing vegetables at home and emphasize snail and slug control and thorough washing of leafy greens before consumption.

As a single-patient observation, the findings are not generalizable and cannot define the frequency of abdominal allodynia in neuroangiostrongyliasis. Nonetheless, the case highlights a diagnostically important presentation and supports early consideration of *A. cantonensis* infection when focal allodynia and eosinophilia occur in an endemic region.

Conclusion

This case illustrates how early allodynia, when localized to the abdomen, can mimic surgical pathology and delay recognition of neuroangiostrongyliasis. In areas where *Angiostrongylus cantonensis* is established, an atypical pattern of abdominal pain, particularly when accompanied by eosinophilia, should prompt consideration of neuroangiostrongyliasis. A focused exposure history that includes gardening, contact with snails and slugs, and consumption of raw leafy greens, including home-grown produce, is crucial to early diagnosis of *A. cantonensis* infection. Recognizing this pattern may reduce unnecessary surgical investigations, shorten time to diagnosis, and limit long-term neurological morbidity from this under-recognized infection.

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M.L. (corresponding author) was directly involved in the clinical care of the patient, conducted the literature review, and led the final drafting, editing, and submission of the manuscript. J.L. prepared the initial draft of the Case presentation, Investigations, Treatment, and Outcome sections of the manuscript, liaised with the patient to obtain the written consent. G.L. contributed a social science perspective in writing up the case, and significant input to editing the manuscript. J.J.P. was the senior clinician involved in the patient's care and provided expert oversight of case management and critical review of the manuscript. All authors reviewed and approved the final version of the manuscript.

Author contributions

Maria Lean (Conceptualization [lead], Investigation [lead], Writing—original draft [lead], Writing—review & editing [equal]), James Loomes (Writing—original draft [supporting], Writing—review & editing [equal]), Garth Lean (Writing—review & editing [equal]), and Jeffrey J. Post (Conceptualization [lead], Supervision [lead], Writing—review & editing [equal])

Conflicts of interest

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Data availability

There are no new data associated with this article.

Patient consent statement

Written informed consent for publication of their clinical details and clinical images was obtained from the patient. A copy of the consent form is available for review by the Editor of this journal.

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