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CASE REPORT

Eosinophilic Meningitis and Pneumonitis due to Angiostrongyliasis: a Case Report

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INTRODUCTION

Angiostrongylus cantonensis infection is one of the most common causes of eosinophilic meningitis in Southeast Asia and the Pacific Basin.[1] The organism was first described in 1935 by Chinese parasitologist Hsin-tao Chen. It was first found in the cerebrospinal fluid (CSF) of a Japanese patient who died of eosinophilic meningoencephalitis in Taiwan in 1944.[2] Since then, there has been an increase in the global distribution of reported cases. Sporadic cases in travellers who have returned from endemic areas have been reported.

Infection with *A cantonensis* can arise from ingestion of food items contaminated by intermediate or definitive hosts. Neural tissue can be targeted after infection. Lung involvement is less commonly encountered clinically. To the best of our knowledge, this is the first reported local case of *A cantonensis* infection with both central nervous system and lung involvement.

CASE PRESENTATION

A 29-year-old Chinese man with a history of epileptic seizure since childhood was admitted to Tuen Mun Hospital with a 5-day history of severe cerebellar ataxia, preceded by a 3-day history of fever and mild dry cough after travelling to Japan. On admission, he was afebrile with a right-hand intentional tremor, pass pointing sign, and tandem walking instability. No neck rigidity was detected. His Glasgow Coma Scale score was 15. Computed tomography (CT) of the brain on admission was essentially normal.

Initial lumbar puncture revealed elevated opening pressure and a CSF white blood count of 257/μL, with 96% lymphocytes and 4% polymorphonuclear leucocytes. Cryptococcal antigen, bacterial culture and sensitivity test, CSF Japanese encephalitis virus antigen, *Mycobacterium tuberculosis* polymerase chain reaction (PCR), and CSF viral PCR test results were all negative. Subsequent magnetic resonance imaging (MRI) of the brain (Figure 1) showed diffusely increased leptomeningeal enhancement, suggestive of meningitis. MRI of the whole spine showed no abnormal signal along the spinal cord. A new lumbar puncture revealed escalating white cell count from 257 to 2049/μL. Repeat CSF viral, bacterial, and fungal test results were again all negative.

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Data Availability: All data generated or analysed during the present study are available from the corresponding author on reasonable request.

Ethics Approval: The study was approved by New Territories West Cluster Research Ethics Committee of Hospital Authority (Ref No.: NTWC/REC/20052). The patient was treated in accordance with the tenets of the Declaration of Helsinki and has provided written informed consent for all treatments and procedures. Patient consent for this study was waived by the Committee.

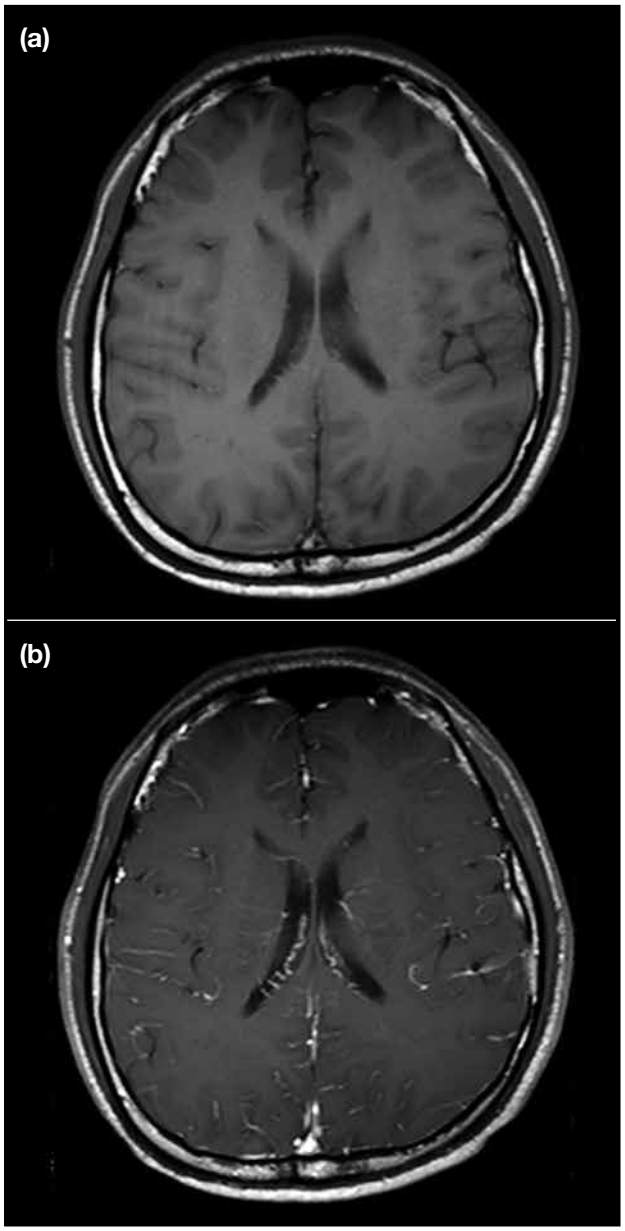

Figure 1. (a) T1-weighted and (b) T2-weighted post-contrast magnetic resonance images showing diffuse leptomeningeal enhancement over the brain, suggestive of meningitis.

However, peripheral blood eosinophil count progressively increased to 33.3% (normal <6%). A third lumbar puncture demonstrated eosinophilia, and a PCR test for *Angiostrongylus* spp. was positive. Plain chest radiograph on admission had revealed patchy infiltrates over peripheral lung fields (Figure 2). CT scan of the thorax showed bilateral subpleural consolidation and ground-glass opacities (Figure 3). Of note was the presence of the halo sign in some areas, that is ground-glass opacity surrounding some of the consolidative lesions.

The patient was prescribed empirical antibiotics, antiviral treatment, and analgesics with gradual clinical improvement. He was not immunocompromised after

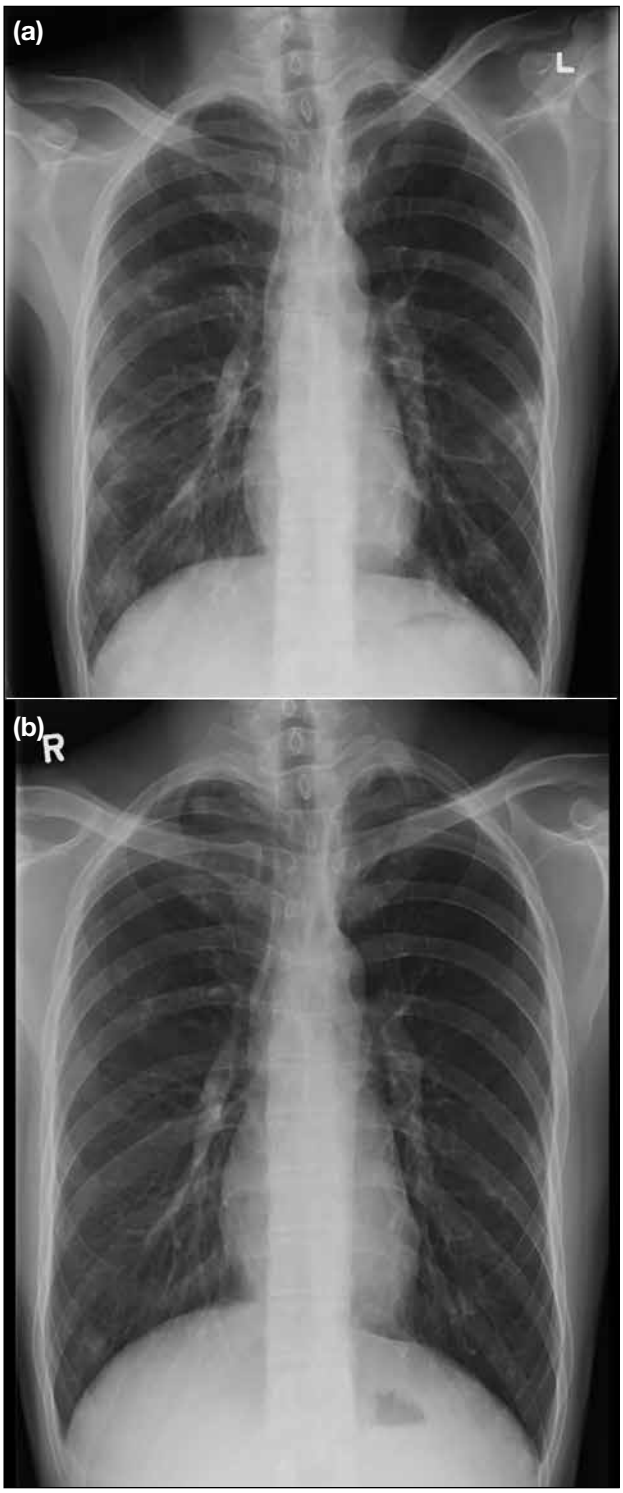

Figure 2. (a) Early plain radiograph of the chest revealed patchy air-space opacities over lung peripheries. (b) Plain radiograph of the chest taken 3 months later showed resolution of bilateral lung consolidation.

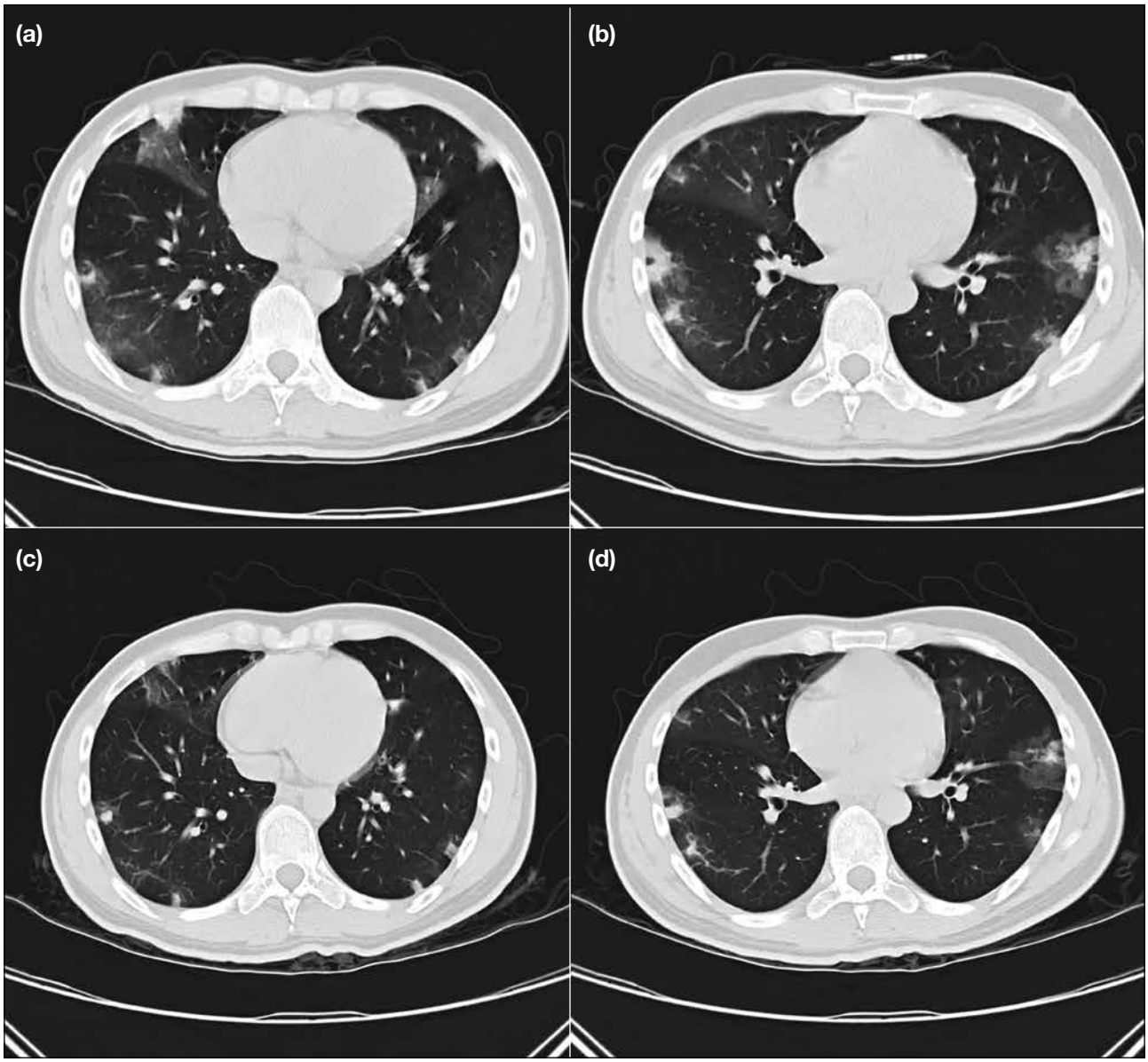

Figure 3. (a) & (b) Initial computed tomography scans of the thorax showed bilateral subpleural consolidation and ground-glass opacities. (c) & (d) New computed tomography scans of the thorax taken 4 days later showed a mild decrease in size of bilateral lung consolidative subpleural lesions.

work-up and was discharged after 4 weeks, with follow-up CT scan of the thorax during admission (Figure 3) and chest radiograph after discharge showing resolution of the lung lesions (Figure 2). His fever had resolved, and no headache or neurological signs were evident. Peripheral eosinophil count was decreasing.

DISCUSSION

A cantonensis primarily infects rat lungs. First-stage larvae migrate to the pharynx where they are swallowed and excreted via the faeces. Intermediate hosts such as snails ingest the faeces of rodents contaminated by these first-stage larvae that then moult twice within the mollusc to develop into second- and third-stage larvae. The third-stage larva is the infecting form of the parasite. Human infection can occur after ingestion of undercooked snails, or through ingestion of unwashed salads or raw vegetable juice contaminated with infective third-stage larvae.[3]

Once infective third-stage larvae are ingested, they can invade the gastrointestinal system and cause enteritis. The larvae then pass through the liver and lungs before reaching the nervous system. Symptoms that include

cough, sort throat, and fever develop as the nematode moves through the lungs. The neurological manifestations of *A cantonensis* infection include eosinophilic meningitis, encephalitis or encephalomyelitis, radiculitis, cranial nerve abnormalities, and ataxia.

Angiostrongyliasis should be considered in patients with high eosinophil count in blood and/or CSF. The diagnosis of angiostrongyliasis is confirmed by detection of *A cantonensis* antigen or antibody in blood or CSF. PCR for antigen detection in CSF has recently been developed and may help to confirm diagnosis at an earlier stage.[4] In our case, the patient had a high eosinophil count in blood and CSF. The CSF PCR confirmed the diagnosis.

MRI examination of the brain and spinal cord in our patient showed diffuse leptomeningeal enhancement of the brain only. There was no abnormal enhancement of the spinal cord on MRI study. In a case series with 17 MRI examinations of the brain and spinal cord in five patients with angiostrongyliasis, multiple enhancing nodules in the brain and linear enhancement in the leptomeninges were the main imaging findings.[5] Spinal cord involvement with enhancement of the nerve roots can also occur but is rare.[5] In a study of 27 patients who were clinically diagnosed with angiostrongyliasis, MRI findings included leptomeningeal enhancement, thickening of the meninges, intracranial nodules, and perimeningeal vascular thickening.[6]

The CT thorax in our patient showed diffuse bilateral subpleural consolidations with ground-glass halo sign. In another study, CT thorax in 15 patients revealed pulmonary nodular lesions and ground-glass opacity lesions located in the subpleural area, which are characteristic signs of the disease.[7] In a case series of 12 patients with lung involvement, CT findings included small nodules with or without a halo of ground-glass attenuation, patchy areas of ground-glass attenuation and focal thickening of bronchovascular bundles located mainly in the peripheral lungs.[8] A large series of 81 patients with *A cantonensis* infection showed concordant imaging findings of focal leptomeningeal enhancement on MRI brain (n = 36) and lung nodules and ground-glass opacity (n = 18).[9]

The diagnosis of angiostrongyliasis can be difficult and requires a high level of suspicion. Clinical history is very important, particularly a history of travel to endemic regions or ingestion of commonly infested host. The diagnosis should be suspected in patients with eosinophilic meningoencephalitis with elevated eosinophils in the blood and/or CSF. MRI brain finding of leptomeningeal enhancement is non-specific. *A cantonensis* can be associated with focal brain lesions on imaging but less commonly than with other helminthic infections of the central nervous system (gnathostomiasis or neurocysticercosis).[10-12] Lung findings of subpleural consolidation and ground-glass opacities, some with halo sign, are also non-specific and can be found in various diseases ranging from infection or neoplasia to inflammatory conditions. Although many cases are self-limiting, severe neurological sequelae and even death may occur.

In summary, we report a case of angiostrongyliasis involvement of the brain and lung. Early recognition and accurate detection of *A cantonensis* can improve patient outcomes. It is important to raise public awareness of disease risk factors in endemic areas and international travellers to prevent further transmission.

REFERENCES

1. Baheti NN, Sreedharan M, Krishnamoorthy T, Nair MD, Radhakrishnan K. Neurological picture. Eosinophilic meningitis and an ocular worm in a patient from Kerala, south India. J Neurol Neurosurg Psychiatry. 2008;79:271.
2. Nomura S, Lin H. First clinical case of Haemostrongylus ratti. Taiwan No Ikai. 1945;3:589-92.
3. Tsai HC, Lee SS, Huang CK, Yen CM, Chen ER, Liu YC. Outbreak of eosinophilic meningitis associated with drinking raw vegetable juice in southern Taiwan. Am J Trop Med Hyg. 2004;71:222-6.
4. Ansdell V, Wattanagoon Y. *Angiostrongylus cantonensis* in travelers: clinical manifestations, diagnosis, and treatment. Curr Opin Infect Dis. 2018;31:399-408.
5. Jin E, Ma D, Liang Y, Ji A, Gan S. MRI findings of eosinophilic myelomeningoencephalitis due to *Angiostrongylus cantonensis*. Clin Radiol. 2005;60:242-50.
6. Yang B, Yang L, Chen Y, Lu G. Magnetic resonance imaging findings and clinical manifestations in cerebral angiostrongyliasis from Dali, China. Brain Behav. 2019;9:e01361.
7. Cui Y, Shen M, Meng S. Lung CT findings of angiostrongyliasis cantonensis caused by *Angiostrongylus cantonensis*. Clin Imaging. 2011;35:180-3.
8. Cheng J, Huang H, Wang X, Wu A, Yu Z, Xu F, et al. *Angiostrongylus cantonensis*: chest CT findings [in Chinese]. Chin J Radiol. 1999;33:371-3.
9. Wang J, Zheng X, Yin Z, Qi H, Li X, Ji A, et al. A clinical analysis of 81 cases of *Angiostrongylus cantonensis* in Beijing [in Chinese]. Chin J Intern Med. 2008;1:50-1.
10. Diaz JH. Recognizing and reducing the risks of helminthic eosinophilic meningitis in travelers: differential diagnosis, disease management, prevention, and control. J Travel Med. 2009;16:267-75.
11. Wang QP, Lai DH, Zhu XQ, Chen XG, Lun ZR. Human angiostrongyliasis. Lancet Infect Dis. 2008;8:621-30.
12. Ramirez-Avila L, Slome S, Schuster FL, Gavali S, Schantz PM, Sejvar J, et al. Eosinophilic meningitis due to *Angiostrongylus* and *Gnathostoma* species. Clin Infect Dis. 2009;48:322-7.