Magnetic Resonance Imaging Findings of Eosinophilic Meningoencephalitis Caused by Angiostrongyliasis

TSYH-JYI HSIEH 1  GIN-CHUNG LIU 1  CHUAN-MIN YEN 2  YU-TING KUO  MIN-SHON CHOU  FENG-O SHEU

Departments of Radiology 1  and Parasitology 2, Kaohsiung Medical University, Kaohsiung, Taiwan

We report on magnetic resonance imaging (MRI) findings of eosinophilic meningoencephalitis caused by angiostrongyliasis in a 79-year-old Chinese woman. MR imaging in this case was compatible with the features of meningitis and angiostrongyliasis, including leptomeningeal thickening and microcavity formation with slight hemorrhagic and granulomatous inflammatory reaction caused by migration of worms. To our knowledge, this is the first report of MRI of eosinophilic meningoencephalitis caused by angiostrongyliasis in Taiwan.

Key words: Meningoencephalitis, angiostrongyliasis; Magnetic resonance (MR)

Eosinophilic meningoencephalitis is an entity of central nervous system (CNS) involvement associated with eosinophilic pleocytosis. The most important pathogen of human eosinophilic meningoencephalitis is believed to be the invasion of the CNS by the rat lungworm, Angiostrongylus cantonensis [1]. Definitive diagnosis of this parasitic disease can be made by the recovery of worms from cerebrospinal fluid (CSF) or other tissue. In addition, detection of circulating antigens in CSF of a patient by means of monoclonal antibodies by double-antibody sandwich ELISA has been established for the immunodiagnosis of angiostrongyliasis [2]. Imaging studies were not considered to be so important in previous reports [1, 3]. In previous articles, computed tomography (CT) showed brain edema, increased intracranial pressure, or a small space-occupying lesion. However, the most common findings are normal features [3]. MR imaging is thought to be of diagnostic value and helpful [4]. We report a case of angiostrongyliasis meningoencephalitis which was first observed by magnetic resonance (MR) imaging in Taiwan.

CASE REPORT

A 79-year-old female was referred to our hospital on 1 October 1999 for evaluation and treatment of eosinophilic meningoencephalitis. On 20 August 1999, she had eaten raw frogs and denied any discomfort until 30 August 1999, at which time she began to suffer from headaches, nausea, and vomiting. Mild fever (about 37.5°C), and marked eosinophilia (WBC: 6500/µl, eosinophils: 64.1%) were noted on 10 September 1999. The symptoms worsened on 22 September 1999. A high fever of up to 39.4°C associated
with delirium status and stool incontinence were encountered. Antibiotics (vancomycin + Cipnoxin) were given following blood culture results, and the symptoms improved. However, CSF study on 25 September 1999 showed WBC of 71/µl and eosinophils: 80%. Under the impression of eosinophilic meningitis, mebendazole was given. Brain CT was also performed with no definite abnormal findings.

On admission, she was suffering from mild fever and headache with no neurological problems. Antibodies to worm antigens of A. cantonensis in serum and CSF assayed by ELISA were positive, and the levels (1.02 and 0.73 ELISA, respectively) were significantly higher than those of control subjects. Worms were also identified in CSF. For evaluation of the infectious condition of eosinophilic meningoencephalitis, the first MR examination was performed on a 1.5-T MR scanner (Phillips ACS-NT, Netherlands) with parameters of spin echo T1-weighted images (WI) (451/12, TR/TE), turbo spin echo T2WI (3000/120/13, TR/TE/TSE factor), FLAIR images (6000/100/2000/19, TR/TE/TI/TSE factor), and post-Gd-DTPA-enhanced spin echo T1WI (451/12). A small spot with high signal intensities

![Figure 1](image1.png)

Figure 1. A 79-year-old woman with eosinophilic meningoencephalitis. a. T1-weighted axial image showing a small spot with high SI at the left caudate nucleus. b. FLAIR axial image revealing high SI in the corresponding area. c. T1-weighted axial images showing multiple small lesions with low SI in white matter, which has high SI on FLAIR d. and T2WI images e. f. Gadolinium-enhanced T1-weighted axial image showing multiple small lesions with central enhanced spots.
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(SIs) on T1WI, T2WI, and FLAIR images was identified at the left caudate nucleus; the perifocal zone showed moderately low SI on T1WI and moderately high SIs on T2WI and FLAIR images. This lesion had no enhancement (Figure 1a-b). The lesion was considered to consist of subacute hematoma with perifocal edema. There were multiple other lesions with isointensity to mildly low signal intensity on T1WI and high signal intensities on T2WI and FLAIR images at the medulla oblongata, cerebellum, and cerebrum including both white and gray matter (Figure 1c-e). The central spots or lines of intense enhancement were revealed after intravenous Gd-DTPA administration (Figure 1f). Granulomatous inflammatory reaction was suspected to be the cause. Abnormal enhancement of leptomeninges was also identified. Meningitis was correlated to this finding. After treatment by levamisole (300 mg/day) for 1 month, the symptoms improved, and laboratory data returned to normal limits. The second MR with the same parameters was performed 8 months after the first examination. The previous lesions with abnormal signal intensities were not seen in this study (Figure 2).

**DISCUSSION**

The neuropathology of human angiostrongyliasis was first described in the 1960s by autopsy [1]. Gross pathology revealed thickening of the leptomeninges and recovery of worms [1]. Vascular dilatations, both arterial and venous, in the subarachnoid space were also noted in autopsy [5]. Microscopically, migration of the worms is one characteristic cause of the neuropathology of angiostrongyloides [5]. Microcavities or tracts are produced by the passage of migrating worms. Disruption of brain tissue, debris, giant cells, and cellular infiltration were identified. Hemorrhage is sometimes noted in microscopic examination. Non-hemorrhagic tracts are usually smaller than 150 µm. Some of the tracts are filled with mononuclear cells, foreign-body giant cells, and macrophages. Perivascular cuffing and degenerated nerve cells can be found in the vicinity of these tracts. Except when worms are found in blood vessels, perivascular spaces, or CSF, the cellular reaction is identified around the worms, more so on dead than living ones. The granulomatous inflammatory reactions can be demonstrated with mononuclear cells and eosinophils. The nerve cells in adjacent areas to the worms may exhibit central chromatolysis and cytoplasmic axonal swelling. Similar pathological findings may be seen in the spinal cord. In our case, multiple enhanced spots with perifocal moderately high SIs on T2WI and FLAIR images and mildly low SI on T1WI were visualized in the brain stem, and cerebral and cerebellar parenchyma. No specific distribution was noticed. These lesions are considered to be due to the granulomatous inflammatory reaction caused by the worms [4]. The migration of worms may destroy adjacent

![Figure 2. Follow-up MR examination 8 months later. The FLAIR a. and gadolinium-enhanced T1-weighted b. axial images are free of abnormal SI lesions.](image)
BBB and induce engorgement of adjacent vessels. These situations may induce a high SI after Gd-DTPA administration. The perifocal edema also showed increased SIs on T2WI and FLAIR images (Figure 1d-e). Comparing the T2WI and FLAIR images, we can identify these lesions more easily on FLAIR images. However, we could not clearly detect the worms because their very small sizes are beyond the imaging resolution.

Intracranial hemorrhage can be easily identified and staged by MR images. We found a spot with high SIs on T1WI, T2WI, and FLAIR images, and no enhancement was appreciated (Figure 1a-b). Under high-field MR examination, abnormal SI is considered to be subacute hemorrhage. Perifocal edema was also noted. The lesion did not show up on CT images because MR is more sensitive in detecting slight hemorrhaging, especially subacute hemorrhaging. Leptomeningeal thickening can be noted on post-enhanced T1WI.

The patient was cured with medical treatment. All neurologic symptoms and signs disappeared. The previous lesions detected on MR images had resolved on the follow-up MR examination 8 months later (Figure 2). This is compatible with the clinical findings.

In conclusion, MR imaging of our case revealed the features of meningitis and angiostrongyliasis, including leptomeningeal thickening and microcavity formation with slight hemorrhaging and granulomatous inflammatory reaction caused by migration of worms. We think that MR imaging studies are useful for detecting and following CNS involvement by angiostrongyliasis. They are also correlated to the clinical symptoms.

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**REFERENCES**

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廣東住血線蟲引發腦膜腦炎之磁振造影表現

謝賜吉¹  劉金昌¹  顏全敏²  郭禹廷¹  周敏雄¹  許鳳娥¹

高雄醫學大學附設中和紀念醫院 放射科¹
高雄醫學大學 寄生蟲科²

我們報告一例七十九歲的中國女性感染廣東住血線蟲而併發腦膜腦炎，它在磁振造影(MRI)的影像有特殊的表現，除了腦膜炎引起的腦膜增厚，也可看到蟲體遷徙引起之微小腔洞所造成之腦實質變化。就我們所知，這是台灣的第一個報告。

關鍵詞：腦膜腦炎，廣東住血線蟲，磁振造影