CASE REPORT

MR spectroscopy and MR perfusion character of cerebral sparganosis: a case report

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ABSTRACT. The authors report the case of a 46-year-old woman with cerebral sparganosis resulting from infection with a larva of *Spirometra*. Computed tomography and magnetic resonance imaging revealed a mass lesion with prominent perifocal oedema in the left parietal lobe. Advanced imaging pulse sequences, including MR spectroscopy and MR perfusion, were performed. During surgery for the removal of a granuloma, the parasite was discovered and excised. Following treatment, the patient's neurological deficits markedly improved.

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Sparganosis is a rare incidental parasite infection in humans caused by infestation of a plerocercoid of the genus *Spirometra*. Cases of sparganosis infection in humans have been reported worldwide, although most cases occur in Southeast Asia, China, Japan and Korea, and less commonly in the United States and Europe [1]. We describe a case of cerebral sparganosis in a 46-year-old woman and discuss the characteristic findings of computed tomography (CT), classical magnetic resonance (MR) imaging, MR spectroscopy and MR perfusion imaging.

1.8 ppm (Figure 2c). MR perfusion imaging showed no apparent increased cerebral perfusion of the lesion and surrounding area (Figure 3). The MR perfusion imaging was performed on a 1.5 tesla machine (Signa Excite, General Electric, Medical Systems, Milwaukee, WI, USA). The specific imaging parameters were as follows: TR/TE 1500/40; field of view 280 × 280 mm; section thickness 5 mm; matrix 128 × 128; flip angle 60°. The contrast medium (gadopentetate dimeglumine; 0.2 mmol kg⁻¹) was injected with the power injector at a flow rate of

Case report

The 46-year-old woman suffered headaches, nausea and vomiting for two weeks. Imbalance gait and right hemiparesis were noted for one day before transferring to our hospital. Routine laboratory findings including complete blood count and differential count, erythrocyte sedimentation rate, urinalysis results and chest X-ray studies revealed no definite abnormality.

An unenhanced CT scan of the brain (Figure 1) showed a hypodense lesion in the left parietal lobe with extensive perifocal oedema and midline shift. MRI revealed a mixed iso-to-hyposignal lesion on T₂ weighted imaging in the left high parietal lobe with extensive surrounding oedema (Figure 2a). On diffusion weighted (DW) imaging and apparent diffusion coefficient (ADC) mapping imaging (Figure 2b), the lesion showed increased signal intensity indicating vasogenic oedema. Single-voxel MR spectroscopy (TE=144 ms) revealed a significantly increased choline (Cho) peak and decreased creatine (Cre) and N-acetylaspartate (NAA) peaks. There was a doubling peak at 1.3 ppm and another elevated peak between 1.4 to

Figure 1. Pre-contrast CT scan shows a patchy area of hypoattenuation in the white matter of the left parietal lobe with prominent mass effect.

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(a)

(b)

(c)

Figure 2. (a) Axial T_2 weighted MRI shows a mixed iso-to-hypointense lesion in the left high parietal lobe with extensive surrounding oedema. (b) Diffusion weighted imaging (left) and apparent diffusion coefficient mapping imaging (right), the lesion shows increased signal intensity. (c) MR spectroscopy (TE=144 ms) reveals an increased choline peak and decreased creatine and *N*-acetylaspartate peaks. There is a lipid or lactate doubling peak at 1.3 ppm. An elevated peak between 1.4 to 1.8 ppm is suspected to be alanine.

3 ml s^{-1} through the intravenous catheter. Heterogeneous enhancement of the parietal lesion was noted and a small rim-enhanced tunnel lesion in the gyrus rectus of the right frontal lobe was depicted on post-contrast enhanced T_1 weighted imaging (Figure 4).

Operation and pathological findings

About one week after admission, under the impression of unusual inflammatory process, an operation was performed for resection of the parietal lesion.

Pathology reported granulomatous inflammation in the brain lesion with dead parasite body noted. Inside the parasite, calcareous bodies were evident; the feature was compatible with the larva of the genus *Spirometra* (Figure 5). At the follow-up examination one month post-operation, the patient had no neurological deficit.

Discussion

Cerebral sparganosis is a rare parasitic infection in humans caused by infestation of the pleocercoid of

Figure 3. MR perfusion imaging shows no apparent increased cerebral perfusion of the lesion and surrounding area.

Spargametra mansoni [2]. The common route of infection in humans is through ingestion of fresh or improperly cooked meat of the infected host, such as pigs, snakes and frogs. Other infection routes include drinking contaminated water or applying the flesh or skin of an infected host as a poultice on a skin wound [3]. Reviewing our patient's history, the patient was a Taiwanese aborigine farmer with a history of drinking uncooked stream water; she also ate some raw snails and boar meat and, during childhood, experienced using frog skin as wound dressing.

Song T et al [4] described three important characteristic findings of cerebral sparganosis. The first important manifestation was the tunnel sign, which was caused by the moving tract of a migrating worm, corresponded to inflammatory granulomas according to the pathological examination. On MRI, the tunnel sign usually presents as a 4×0.8 cm columnar or fusiform-shaped hyposignal on T_1 weighted images, slight hypersignal on T_2

Figure 5. Photomicrograph of a section of the lesion reveals an inflammatory granuloma with a dead parasite body. Inside the parasite, calcareous bodies are evident. Haematoxylin and eosin (H&E) stain.

weighted images and as an obvious enhanced lesion. In our patient, the right frontal base lesion was compatible with the feature of a tunnel sign. The second most common feature of cerebral sparganosis on MRI was the conglomerated ring-like enhancement, which was compatible with the left parietal lesion in our patient. Other common findings of cerebral sparganosis corresponded to the varying stages of the inflammatory process: in the acute stage extensive perifocal oedema and mass effect were presented, whereas in the chronic stage cortical atrophy, dilated ipsilateral ventricle and punctate calcification were apparent. Bo and Xuejian [5] presented the MR spectroscopy and DW imaging findings for a child with cerebral sparganosis. In their study, iso-signal intense lesion on DW imaging was noted and increased Cho and decreased NAA peaks were observed; the findings were consistent with the spectral features of cerebral neoplasm or granuloma, which was similar to our patient's DW imaging and MR spectroscopy features. Moreover, MR spectroscopy of our patient revealed some additional peaks; the doubling peak at 1.3 ppm is likely to be lipid or lactate, implying necrosis of the lesion. Another peak was observed between 1.4 and 1.8 ppm indicating something not presented in brain

Figure 4. Post-contrast T_1 weighted images show a heterogeneous enhanced parietal lobe lesion and a small rim-enhanced tunnel lesion in the gyrus rectus of the right frontal lobe.

tissue, such as alanine, which had been reported in the MR spectroscopy findings of neurocysticercosis [6]. However, it was still difficult to differentiate the lesion from cerebral malignancy or granuloma. MR perfusion imaging in our patient presented no definite increased cerebral perfusion, which could indicate a benign lesion rather than a cerebral malignancy. To the best of our knowledge, this is the first report in which the MR perfusion imaging of cerebral sparganosis has been described.

Cerebral sparganosis requires surgical excision of the parasite; praziquantel has no effect on adult worms in the central nervous system. Complete surgical resection of granuloma together with worms was the choice of treatment [7]. There were at least two lesions in our patient, the left parietal lobe lesion and the right frontal lobe lesion. The surgeon had removed the left parietal lesion. After anti-inflammatory treatment with corticosteroids, the frontal lesion and evident left parietal perifocal oedema were diminished on follow-up imaging and the patient had no definite neurological deficit.

Conclusion

Cerebral sparganosis is difficult to correctly diagnose initially. MRI is the most helpful tool in this regard, as it identifies some characteristic features. Some advanced

sequences such as MR spectroscopy and MR perfusion provide more clues for differentiation from benign or malignant lesions. Combined with the clinical history, a correct pre-operative diagnosis of cerebral sparganosis could be established.

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