

Case report

MRI findings of VIIIth cranial nerve involvement in sarcoidosis

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Abstract. Right facial nerve palsy in a 58-year-old woman was due to sarcoidosis demonstrated by Gd-DTPA enhanced MRI. Abnormal enhancement of the right VIIIth cranial nerve in the distal internal acoustic canal was seen on MRI. The enhancing lesion was smaller after 1 month of prednisolone 50 mg day⁻¹. This is the first report on facial nerve involvement in neurosarcoidosis examined by Gd-DTPA enhanced MRI. The use of Gd-DTPA enhanced MRI with thin slicing, *e.g.* 3 mm slice thickness and 1 mm interslice gap, is effective in detecting small extramedullary lesions.

Neurosarcoidosis, which occurs in 5–10% of sarcoidosis patients [1, 2], commonly includes cranial nerve palsy, especially facial nerve palsy [1, 2]. However, there has been only one report of gadolinium-diethylenetriamine pentaacetic acid (Gd-DTPA) enhancement of cranial nerves (VIII) on MRI in neurosarcoidosis [3]; although MRI findings in central nervous system (CNS) sarcoidosis have often been described [4–6]. We report a case in which facial nerve (VII) involvement in neurosarcoidosis was detected by Gd-DTPA enhanced MRI.

Case report

A 58-year-old woman presented with a 3 day history of right facial nerve palsy. Sarcoidosis had been diagnosed by transbronchial lung biopsy 4 years previously. She could not close her mouth when she drank tea. There was no history of vertigo, hearing loss, tinnitus, ear pain or loss of taste. The only abnormalities on full physical examination were right facial nerve palsy and brown plaques on both knees. The right forehead wrinkles and nasolabial fold were shallow, and the right eye could not be completely closed. Skin biopsy on the knee showed sarcoid-type granuloma. Laboratory results were: WBC, 5800/mm³ (69.0% neutrophils and bands, 21.2% lymphocytes); AST, 90 IU l⁻¹; ALT, 81 IU l⁻¹. Serum angiotensin-converting enzyme (ACE) was elevated at 40.6 IU l⁻¹. Serological findings were negative for varicella-zoster virus, herpes simplex virus, echo virus or Coxsackie virus. The chest radiograph was normal. Axial T₁ weighted images pre- (Figure 1) and post-Gd-DTPA enhanced MRI (1.5 T

superconducting magnet MRI; Philips Gyroscan ACS-NT, The Netherlands) were obtained at the level of the internal acoustic canals with a 3 mm slice thickness and a 0.9 mm interslice gap. 3 days after the onset of facial nerve palsy, abnormal enhancement of the right VIIIth cranial nerve in the distal internal acoustic canal was seen (Figure 2). After 1 month of treatment with prednisolone 50 mg day⁻¹, the enhancing lesion was reduced in size (Figure 3). Serum ACE level had returned to the normal range (14.8 IU l⁻¹).

Discussion

MRI findings of CNS sarcoidosis, including white matter, periventricular, periaqueductal or leptomeningeal lesions, have often been reported



Figure 1. Axial image (500/13) demonstrating an extraaxial iso-signal intensity mass simulating sarcoid granuloma involving the VIIth cranial nerve (arrow) at the right internal acoustic canal.

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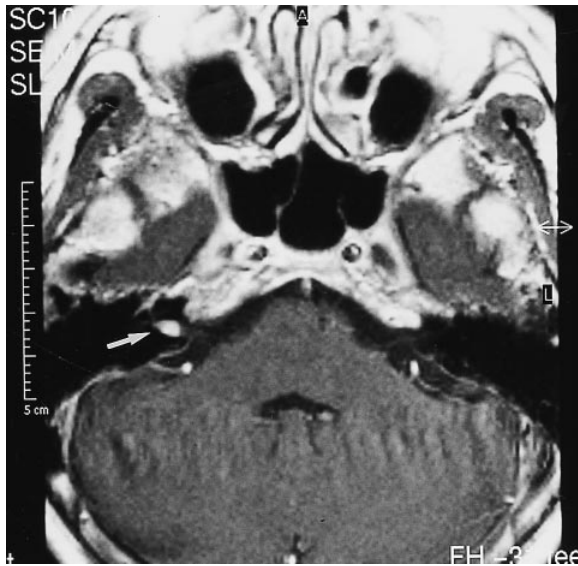


Figure 2. Post-gadolinium T_1 weighted (500/13) axial image showing markedly enhanced granulomatous lesion (arrow) in the right acoustic canal.



Figure 3. Post-contrast T_1 weighted (500/13) axial image after steroid therapy, showing a reduction of the size (arrow).

[5, 6]. These lesions partially or completely resolve on steroid treatment. However, there is only one report of MRI of extramedullary sarcoidosis involving the VIIIth cranial nerve [3], and none concerning the VIIth cranial nerve, although the VIIth cranial nerve is frequently involved in neurosarcoidosis. The reasons why extramedullary sarcoidosis has generally not been found by MRI so far may include large slice thickness, failure to use Gd-DTPA and unavailability of high-resolution MRI. It is recommended that patients with extramedullary sarcoidosis should be checked by Gd-DTPA-enhanced MRI, which should be set up with a 3 mm or less slice thickness and a 1 mm

interslice gap, to avoid the partial volume effect of fatty tissues surrounding these cranial nerves.

Only the internal meatal segment of the right facial nerve was enhanced and enlarged in this patient. The size of this lesion was reduced by steroid treatment over 1 month, although there was no distinct change in its intensity. O'Reily et al [3] reported that a high signal intensity sarcoid mass enhanced by Gd-DTPA in the VIIIth cranial nerve virtually resolved 1 year after steroid treatment, although a residual high signal lesion remained on the affected side. In contrast the facial nerve in Bell's palsy is not usually enlarged and the intensity of enhanced lesions is unaltered months after steroid treatment [7]. Ramsay-Hunt syndrome, which involves the acoustic nerve as well as the facial nerve, still shows labyrinthine enhancement by Gd-DTPA 6 months after onset [8]. Thus, the different effectiveness of steroid treatment between sarcoidosis and Bell's palsy may reflect different underlying pathological conditions. Histopathological findings in most patients with neurosarcoidosis indicate epineural and perineural granuloma as well as periangitis and panangitis [9]. The enhancement seen on MRI would therefore be expected to resolve with steroid treatment. On the other hand, in acute peripheral inflammatory facial nerve palsy, long-lasting intense contrast enhancement is seen in the facial nerve and may be explained by two factors [10]: the blood-nerve barrier is damaged by macrophage infiltration and Wallerian degeneration; while the perineurium is damaged by increased transfer of metabolic substrates to support regeneration. These conditions are unlikely to respond rapidly to steroid treatment.

In conclusion, Gd-DTPA enhancement MRI can be useful to show the response, in terms of size reduction, to steroid in cranial nerve palsy caused by granulomatous conditions such as sarcoidosis.

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