

MR Appearance of Hypertrophic Olivary Degeneration After Contralateral Cerebellar Hemorrhage

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Hypertrophic olivary degeneration is not a primary lesion but rather develops as a result of a contralateral lesion of either the dentate nucleus or the central tegmental tract. Pathologically, hypertrophic olivary degeneration is marked by myelin loss, vacuolar necrosis of neurons, and gliosis. These histological changes are consistent with an increase in mobile water protons and may be responsible for the MR signal abnormalities seen in the case reported here.

Case Report

A 68-year-old man with arterial hypertension experienced an acute onset of headache and vertigo. Examination at that time demonstrated left cerebellar ataxia, multidirectional nystagmus, bilateral ptosis, and meningeal irritative signs.

A noncontrast CT scan disclosed a left hemispheric cerebellar hematoma with compression of the left middle cerebellar peduncle and the fourth ventricle.

Although the patient improved progressively, at the time of discharge 1 month later there was still a major cerebellar ataxia, left-sided hypotonia, bilateral gaze nystagmus, and upbeat nystagmus on vertical gaze.

At follow-up examination 8 months later, these signs and symptoms had further improved but additional signs of a typical palatal myoclonus and intermittent abnormal fast movements of the left fingers and of the proximal portion of the left arm were found.

An MR study was performed on a 0.5-T unit (Magniscan, CGR). Spin-echo (SE) T1- and T2-weighted images were obtained in the axial plane. The sequelae of the hematoma were visible, and the MR features of the medulla oblongata appeared abnormal. An ovoid area of high signal intensity was present in the vicinity of the right olivary nucleus on proton-density and T2-weighted images.

Four months later, the clinical status was unchanged and the palatal myoclonus was still present.

The patient underwent a second MR study on a 1.5-T MR unit (Magnetom, Siemens).

The pre- and postolivary sulci, usually easily visible on MR images (Fig. 1), could not be recognized, and the olivary nucleus appeared hypertrophied. The slightly high signal intensity of the olivary nucleus was better detected on proton-density than on T2-weighted images (Fig. 2).

Discussion

The most characteristic and striking nuclear structure in the medulla oblongata is the olivary nuclear complex. This complex consists of two accessory nuclei and one main inferior nucleus, also called the principal inferior olive. The fibers emerging from the principal olive terminate within the lateral part of the contralateral cerebellar hemisphere and dentate nucleus [1, 2]. These nuclei are not distinguishable on MR images, but the relief of the olive is visible on normal axial views (Fig. 2).

It is well known from pathologic data that a lesion involving the dentate nucleus or the central tegmental tract may be associated with secondary hypertrophic degeneration of the contralateral olivary nuclear complex [3, 4]. This olivary lesion is often but not always associated with palatal myoclonus [3], which in fact was present in our case.

Two previous reports [5, 6] have described the MR appearance of olivary degeneration. In [5], signal abnormalities were inconstantly found in one of two patients examined, and in [6] signal abnormalities were seen in three of four patients. Similar features were seen in our case.

Pathologically, hypertrophic olivary degeneration is characterized by neuronal loss, vacuolar necrosis of neurons, loss of myelin, and gliosis [4]. In our case, MR demonstrated the two sites of abnormality: a hemorrhage within the left dentate nucleus and a contralateral olivary abnormality.

Correlation between the abnormal MR signal and the histological changes of olivary degeneration remains a matter of discussion, since no pathologic correlation was available in our case. However, similar histological components of this lesion have already been correlated with MR data by means of clinical and/or experimental work.

Loss of myelin and neuronal vacuolar necrosis are associated with an increase in water content and may be responsible for high signal intensity on long TR sequences [7]. It has also been demonstrated that gliosis in temporal lobe epilepsy can produce areas of high signal intensity on long TR sequences

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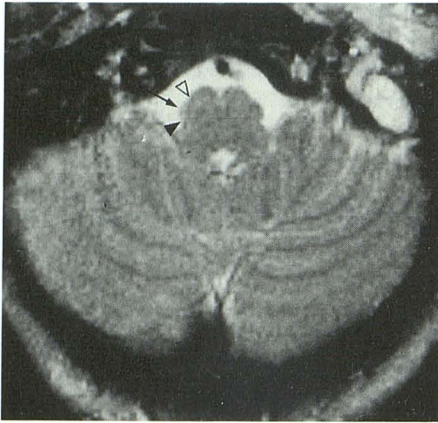
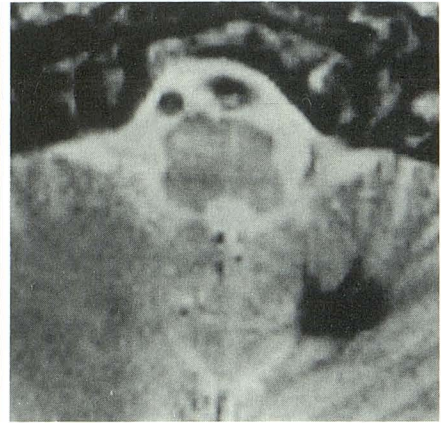


Fig. 1.—Axial T2-weighted, 2000/90/1, spin-echo MR image shows normal shape of upper medulla oblongata. *Straight arrow* = olivary nucleus, *open arrowhead* = preolivary sulcus, *closed arrowhead* = retroolivary sulcus.



A



B

Fig. 2.—MR study obtained 12 months after initial hemorrhage. *A and B*, Axial T2-weighted, 2000/22/1 (*A*) and 2000/90/1 (*B*), spin-echo images show hypertrophic olivary degeneration as area of high signal intensity with changes in the normal shape of the medulla oblongata: the two hemimedullae are asymmetric, and the pre- and postolivary sulci have disappeared.

[8]. Another possible cause of the high T2 signal intensity of the olivary nucleus is a focal area of small vessel infarction. However, we think that this diagnosis can be excluded, since the lesion is hypertrophic and modifies the normal shape of the brainstem. Furthermore, no shrinkage of the lesion was demonstrated on the second MR examination performed 4 months after the first one.

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