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Pontine Hemorrhages Presenting as Trigeminal Neuropathy

—Report of Three Cases—

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Abstract

Three patients with pontine hemorrhages presenting clinically as trigeminal neuropathy are described. No patient was hypertensive and angiograms were normal. Magnetic resonance (MR) imaging showed heterogeneous lesions, suggesting cavernous malformation, in one patient, but no definite diagnosis could be made in the other two patients. Such cases are rare and should be evaluated using serial MR imaging to differentiate angiographically occult vascular malformation from spontaneous hemorrhage.

Key words: pontine hemorrhage, trigeminal neuropathy, cavernous malformation, magnetic resonance imaging

Introduction

Pontine hemorrhage can manifest as a variety of symptoms,[3,4,9,11,12,21] but clinical presentation as trigeminal neuropathy due to small pontine hemorrhages in the lateral or tegmental areas is rare.[1,2,7,20] Serial magnetic resonance (MR) imaging is extremely useful to visualize chronological changes and to detect angiographically occult vascular malformations or other lesions.[8] Here, we report three patients with pontine hemorrhage presenting clinically as trigeminal neuropathy.

Materials and Methods

We retrospectively reviewed the clinical and radiological data from three of 77 patients with pontine hemorrhages demonstrated by computed tomography (CT) and/or MR imaging treated at our clinic over the past 8 years, who presented mainly with trigeminal symptomatology. They were normotensive and had no hematological abnormalities, trauma, or other systemic disorder.

CT scans, MR images, and cerebral angiograms were examined. Both T_1- and T_2-weighted SE pulse sequence MR images were obtained with 0.5 Tesla. T_1-weighted images were scanned with TE 40/TR 600–800 msec and T_2-weighted images with TE 100–120/TR 1800 msec. We performed serial MR studies to examine changes in the signal intensity of the hemorrhage.

Case Reports

Case 1: A 49-year-old female complained of sudden onset of left retro-orbital pain, followed by sensory disturbances primarily in the ophthalmic division. The pain subsided but the sensory disturbances persisted. Neurologically, she was alert with decreased pain and touch sensation over the left trigeminal distribution (greater in the first than second and third divisions). There were no other significant findings. CT scans demonstrated a lesion within the pons, near the trigeminal root entry zone. Both T_1- and T_2-weighted MR images demonstrated a small high-intensity lesion at the same location (Fig. 1 *left*).

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Cerebral angiograms showed no abnormalities. Follow-up T_1- and T_2-weighted MR images 1 and 3 months later revealed a very small low-intensity lesion at the same location (Fig. 1 *right*). She showed some improvement over the 1-year follow-up period.

Case 2: A 32-year-old male noted numbness in the tips of his left hand fingers followed about 10 days later by perioral numbness, also on the left, simulating the cheiro-oral syndrome. He had no history of hypalgesia and hypesthesia of the left hand or perioral region. CT scans showed a small, moderately high-density lesion in the right lateral tegmental area. Both T_1- and T_2-weighted MR images revealed a heterogeneous lesion in the same area (Fig. 2). Cerebral angiograms showed no abnormalities. Within 3 months his deficits completely disappeared, and a follow-up examination 3 years later found no deficit. He refused follow-up MR imaging due to claustrophobia, so the etiology of the hemorrhage could not be definitely identified.

Case 3: A 59-year-old male complained of sudden onset of facial numbness and left-sided ataxia. Neurological examination found he was alert with left hemifacial hypesthesia. The left corneal reflex was absent and the masseter was weak. Mild left cerebellar signs and mild hearing loss were also noted. CT scans showed a left dorsolateral pontine hemorrhage extending into the middle cerebellar peduncle. T_1- and T_2-weighted MR images demonstrated a heterogeneous mass with surrounding low intensity at a similar location (Fig. 3 *upper*). Cerebral angiograms showed no abnormalities. Two weeks later, the cerebellar signs became more prominent. Repeat CT scans showed an additional hematoma around the same area. He refused surgical removal of a suspected cavernous malformation. Follow-up T_1- and T_2-weighted MR images 2.5 years later showed a slightly different, but essentially heterogeneous lesion in the same location (Fig. 3

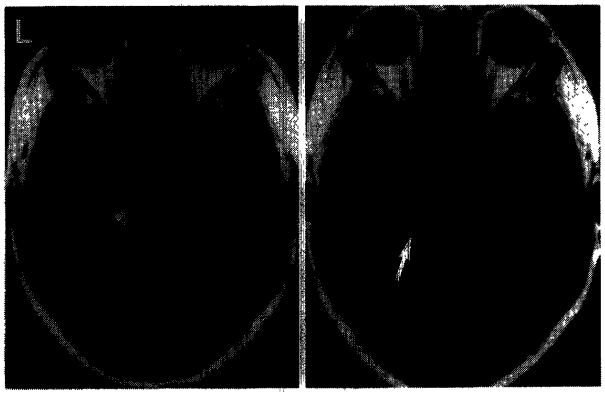

Fig. 1 Case 1. *left*: T_1-weighted SE MR image, showing a high-intensity lesion, representing the hematoma, near the root entry zone of the left trigeminal nerve. *right*: T_1-weighted SE MR image 1 month later, showing a small low-intensity lesion at the same location (*arrow*).

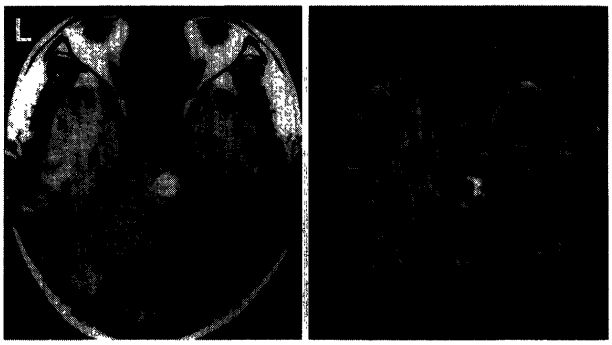

Fig. 2 Case 2. T_1- (*left*) and T_2-weighted (*right*) SE MR images, showing a heterogeneous lesion at the right tegmentum. He presented with the cheiro-oral syndrome.

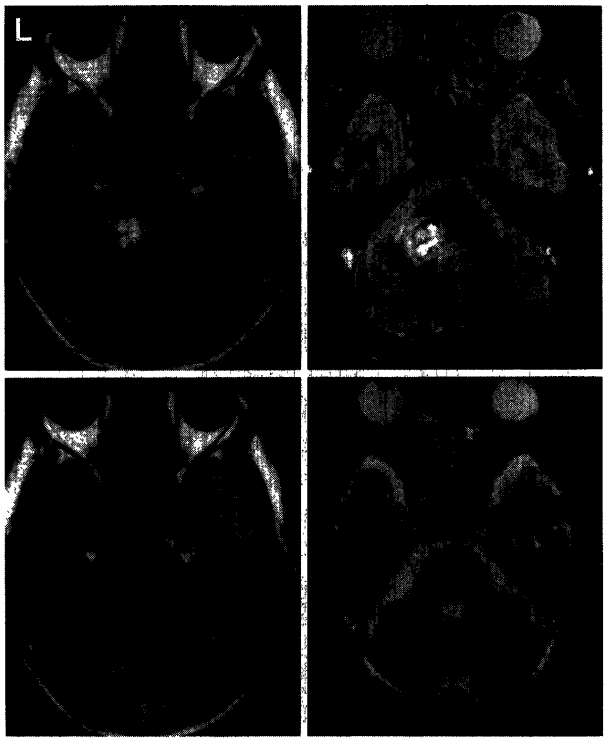

Fig. 3 Case 3. *upper*: T_1- (*left*) and T_2-weighted (*right*) SE MR images, showing a heterogeneous lesion with low-intensity rim in the left lateral tegmental area extending into the middle cerebellar peduncle. *lower*: T_1- (*left*) and T_2-weighted (*right*) SE MR images 2.5 years later, showing a slightly different, but essentially heterogeneous lesion at the same location.

lower). Follow-up examination after 4 years found improved cerebellar signs but the facial numbness remained. He has returned to full employment.

Discussion

Primary pontine hemorrhage in patients above 40 years of age is commonly due to hypertension. Other causes include systemic disorders such as hematological disease and vascular malformations, and secondary hemorrhages following tentorial herniations. The present cases presenting mainly as trigeminal neuropathy are unusual.

Pontine hemorrhages can originate from the paramedian and long and short circumferential arteries.[10] These hemorrhages vary widely in size, producing great diversity in clinical presentation. Trigeminal symptoms, as in our cases, could be due to direct damage or edema of the trigeminal nuclei/tracts resulting from the hematoma. Sensory dissociation does not usually occur since the trigeminal lemniscus and the trigeminothalamic tracts lie close together.

The cheiro-oral syndrome, first described by Sittig in 1914, can be caused by lesions of the parietal lobe, thalamus, midbrain, or the pons,[6,14,18,19] and manifests as sensory disturbances involving the distal portion of the hand and ipsilateral half of the perioral area. Lesions limited to the spinothalamic and trigeminothalamic tracts in the pons and the midbrain, the posterolateral and posteromedial ventral nuclei in the thalamus, the radiation fibers from the thalamus to the cortical sensory area in the parietal lobe, and the cortical sensory area itself on the contralateral side can also cause this syndrome.

CT has facilitated the diagnosis of pontine hemorrhage, except for small hematomas which may be masked by streak artifacts caused by surrounding bone. Blood in the fourth ventricle and surrounding brainstem cisterns may suggest rupture of an aneurysm or arteriovenous malformation,[21] but primary pontine hemorrhage can also produce a similar appearance.[9]

Non-hypertensive pontine hemorrhages presenting primarily as trigeminal neuropathy could be the result of bleeding from an angiographically occult vascular malformation. CT would usually demonstrate a hyperdense irregular or nodular lesion without significant mass effect, and varying degrees of contrast enhancement.[16,17] These findings, however, are not specific for angiographically occult vascular malformations. Angiography may not provide diagnostic information. T_2-weighted MR imaging shows a lenticular core of varying signal intensity suggesting a cavernous malformation.[5,15] A heterogeneous intensity signal indicates a mixture of old and new blood, *i.e.* hemosiderin and methemoglobin. Hemosiderin produces local magnetic heterogeneity resulting in greater magnetic susceptibility. This phenomenon is better seen with a high (1.5 Tesla) than a low (0.5 Tesla) magnetic field.[5] Gradient-echo MR images, which are very sensitive to inhomogeneous local magnetic fields, can differentiate the blood flow in the arteriovenous malformation from hemosiderin deposit.[13] Hemosiderin appears as a markedly low-intensity area while the blood flow is shown as a high-intensity area. Serial MR imaging would clearly demonstrate the changing intensity of the hemorrhage and be of great value.[8]

Pontine hemorrhage presenting primarily as trigeminal neuropathy is rare, and could result from a spontaneous hemorrhage or an angiographically occult vascular malformation. Serial MR imaging is the best available diagnostic modality for differentiation.

References

1) Ambrosetto P: Ataxic hemiparesis with contralateral trigeminal nerve impairment due to pontine hemorrhage. *Stroke* 18: 244–245, 1987
2) Berlit P: Trigeminal neuropathy in pontine hemorrhage. *Eur Neurol* 29: 169–170, 1989
3) Del-Brutto OH, Noboa CA, Barinagarrementeria F: Lateral pontine hemorrhage: Reappraisal of benign cases. *Stroke* 18: 954–956, 1987
4) Epstein AW: Primary massive pontine hemorrhage. A clinico-pathological study. *J Neuropath Exp Neurol* 10: 426–448, 1951
5) Gomori JM, Grossman RI, Goldberg HI, Hackney DB, Zimmerman RA, Bilaniuk LT: Occult cerebral vascular malformations: High-field MR imaging. *Radiology* 158: 707–713, 1986
6) Hashiguchi K, Igata A: A case of primary pontine hemorrhage with cheiro-oral syndrome (palm-oral sensory disturbance). *Shinkei Naika* 5: 79–80, 1976 (in Japanese)
7) Holtzman RNN, Zablozki V, Yang WC, Leeds NE: Lateral pontine tegmental hemorrhage presenting as isolated trigeminal sensory neuropathy. *Neurology* 37: 704–706, 1987
8) Komiyama M, Baba M, Hakuba A, Nishimura S, Inoue Y: MR imaging of brainstem hemorrhage. *AJNR* 9: 261–268, 1988
9) Komiyama M, Boo YE, Yagura H, Yasui T, Baba M, Hakuba A, Nishimura S: A clinical analysis of 32 brainstem haemorrhages: With special reference to surviving but severely disabled cases. *Acta Neurochir (Wien)* 101: 46–51, 1989

10) Komiyama M, Yasui T, Yagura H, Fu Y: Computerized tomographic evaluation of the bleeding sites in primary pontine hemorrhage. *Stroke* 22: 1309–1311, 1991
11) Mashiyama S, Niizuma H, Suzuki J: Pontine haemorrhage: A clinical analysis of 26 cases. *J Neurol Neurosurg Psychiatry* 48: 658–662, 1985
12) Nakajima K: Clinicopathological study of pontine hemorrhage. *Stroke* 14: 485–493, 1983
13) Needell WM, Maravilla KR: MR flow imaging in vascular malformations using gradient recalled acquisition. *AJNR* 9: 637–642, 1988
14) Ono S, Inoue K: Cheiro-oral syndrome following midbrain haemorrhage. *J Neurol* 232: 304–306, 1985
15) Rigamonti D, Drayer BP, Johnson PC, Hadley MN, Zabramski J, Spetzler RF: The MRI appearance of cavernous malformations (angiomas). *J Neurosurg* 67: 518–524, 1987
16) Savoiardo M, Strada L, Passerini A: Intracranial cavernous hemangiomas: Neuroradiological review of 36 operated cases. *AJNR* 4: 945–950, 1983
17) Simard JM, Garcia-Bengochea F, Ballinger WE, Mickle JP, Quisling RG: Cavernous angioma: A review of 126 collected and 12 new clinical cases. *Neurosurgery* 18: 162–172, 1986
18) Sittig O: Klinische Beiträge zur Lehre von der Lokalisation der sensiblen Rindenzentren. *Prager Med Wochenschr* 45: 548–550, 1914
19) Strauss H: Über Sensibiltätsströrungen an Hand und Gesicht, Geschmacksströrungen und ihre lokalisatorische Bedeutung. *Monatsschr Psychiat Neurol* 58: 265–276, 1925
20) Veerapen R: Spontaneous lateral pontine hemorrhage with associated trigeminal nerve root hematoma. *Neurosurgery* 25: 451–454, 1989
21) Weisberg LA: Primary pontine haemorrhage: Clinical and computed tomographic correlations. *J Neurol Neurosurg Psychiatry* 49: 346–352, 1986

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