Gasperini Syndrome, A Report of Two Cases

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Abstract

We report 2 cases of Gasperini syndrome and consider them with the 11 previously reported cases to describe the clinical characteristics of this rare syndrome: Core neurological signs are peripheral facial nerve palsy and abducens nerve palsy of the affected side: Among all cases, imaging demonstrated a small lesion in the mediolateral tegmental pons (10/13 cases of microinfarction; 2/13 cases of microbleeding). We found that the responsible artery in ischemic Gasperini syndrome is mainly the long circumferential branch of the anterior inferior cerebellar artery; Case 1 is the first case thought to be caused by infarction of the basilar artery’s paramedian branch.

Key words: abducens palsy, cerebral infarction, facial palsy, Gasperini syndrome, pons, tegmentum

Case Report

Case 1

A 62-year-old man with a history of diabetes and hyperlipidemia felt left facial weakness, diplopia, and right-sided numbness including his face. His condition continued the following day and he visited our hospital. On the next day, his condition had not improved and he was admitted. On admission, his general physical examination was unremarkable. The initial neurological examination showed left ocular abduction resulting from left abducens palsy and left gaze palsy on his voluntary left gaze. His left gaze palsy persisted on his eye tracking test. His convergence reflex was normal, and his vertical eye movement was full. He had right hemihypalgesia, right hemithermohypesthesia including his face, and left peripheral facial palsy. His masseter muscle strength and touch sensation and other cranial nerve functions were normal. Neither motor paresis of his extremities nor cerebellar ataxia was seen. Total cholesterol was 178 mg/dL, LDL-cholesterol was 130 mg/dL, HDL-cholesterol was 39 mg/dL, glucose was 129 mg/dL, and HbA1c was 8.2%. Other laboratory data were normal (Table 1). Diffusion-weighted imaging (DWI) and T2-weighted imaging (T2WI) on magnetic resonance imaging (MRI) re-
Figure 1. (A) Brain magnetic resonance imaging of Case 1. T2-weighted imaging (T2WI) shows a high-intensity area in the left mediolateral tegmental pons. (B) Brain MRI of Case 2. T2WI shows a high-intensity area in the right mediolateral tegmental pons. (C) Affected areas in Cases 1 and 2 as suspected by neurological examination. (D) Dominant arterial territories of the paramedian branch of the basilar artery and that of the long circumferential branch of AICA (11): ① nucleus of nerve VI ② abducens nerve ③ nucleus of nerve VII ④ facial nerve ⑤ ventral trigeminothalamic tract ⑥ medial lemniscus ⑦ anterior spinothalamic tract ⑧ lateral spinothalamic tract ⑨ spinal tract and nucleus of nerve V ⑩ medial longitudinal fasciculus ⑪ inferior cerebellar peduncle. (E) Lesion maps of the nine cases depicted by MRI. Remaining four cases (Cases 1, 2, 3 and 11) had no neuroimaging performed.

Case 1

A 68-year-old right-handed man with a history of acute stroke revealed a high-intensity area in the mediolateral tegmentum of his left pons (Fig. 1A). Magnetic resonance angiography (MRA) disclosed no abnormalities except for mild bilateral distal internal carotid artery stenosis. In his blink reflex, R1 and R2 waves had disappeared on the affected side, indicating a profound left peripheral facial palsy pattern. Auditory brainstem response (ABR) and short-latency somatosensory evoked potential (SSEP) were normal.

Case 2

A 51-year-old man with a history of diabetes and hyperlipidemia felt right facial weakness, diplopia, and right facial numbness. On admission to our hospital, his general physical examination was unremarkable. His initial neurological examination showed abducens palsy, facial hypalgesia, facial thermohypesthesia and peripheral facial palsy on the right side. His convergence reflex was normal, and his vertical
both men were treated with an anti-platelet agent. in patient 1, left peripheral facial palsy and right hemihypalgesia remained as neurological sequela. patient 2 fully recovered with no remaining neurological deficits.

**discussion**

in gasperini syndrome, abducens palsy, peripheral facial palsy, and deafness of the affected side are thought to occur as a result of VIth, VIIth, and VIIIth nerve nuclei or intrapontine peripheral nerve involvement (1). lateral gaze palsy to the affected side is thought to be caused by in-
volvement of the parapontine reticular formation (PPRF). Facial hypalgesia of the affected side is thought to be caused by involvement of the spinal tract or spinal tract nucleus of the trigeminal nerve. Facial hypalgesia, hemihypalgesia, and deafness of the unaffected side are thought to be caused by involvement of the ventral trigeminothalamic tract, lateral spinothalamic tract, and lateral lemniscus. Patient 1 fulfilled all signs of Gasperini’s original report except deafness on the unaffected side (Table 2). The lesion in patient 1 was thought to include the facial nerve nucleus, abducens nerve nucleus, PPRF, lateral spinothalamic tract, and ventral trigeminothalamic tract (Fig. 1C). On the other hand, patient 2 had abducens palsy, peripheral facial palsy, and facial hypalgesia on the affected side (Table 2). The lesion in patient 2 was thought to include the facial nerve nucleus, abducens nerve nucleus, and spinal tract or spinal tract nuclei of the trigeminal nerve (Fig. 1C). These lesions should anatomically be located in the pontine tegmentum, and both patients’ lesions were identified exactly there by MRI.

Gasperini syndrome is clearly rare. Only 11 cases appear to have been reported in the literature to date, including Gasperini’s original report. Of these 11 cases, 8 cases were reported in Japanese (3-8). We reported an additional 2 cases (11). The remaining 3 ischemic cases have no cardioembolic source or no abnormality of the vertebral and basilar artery, and they are thought to be caused by small vessel disease associated with diabetes mellitus, hyperlipidemia or hypertension, as reported in cranial nerve palsies due to small brainstem lesions (9, 10).

The long circumferential branch from the anterior inferior cerebellar artery (AICA) and the paramedian branch from the basilar artery are candidates for feeding arteries of the VIth and VIIth nuclei and surrounding areas (11). The long circumferential branch from the AICA supplies the spinal tract or spinal tract nucleus of the trigeminal nerve (Fig. 1D) (12). The paramedian branch from the basilar artery supplies the ventral trigeminothalamic tract. In 10 cases of ischemic Gasperini syndrome including our 2 cases, 6 patients had facial hemihypalgesia only on the affected side (Cases 3, 4, 6, 7, 9 and 13), indicating involvement of the spinal tract or spinal tract nucleus of the trigeminal nerve supplied by the long circumferential branch of AICA. Furthermore, 4 of 10 ischemic cases (cases 2, 6, 7 and 11) presented with cerebellar ataxia of the affected side, a finding that Gasperini did not mention. These cases were reported as AICA infarction presenting as Gasperini syndrome (2, 6). Finally, the long circumferential branch of AICA seemed to be the responsible artery in 8 of 10 patients with ischemic Gasperini syndrome (Cases 2-4, 6, 7, 9, 11 and 13). In contrast, only our Case 1 had facial hemihypalgesia only on the unaffected side, indicating involvement of the ventral trigeminothalamic tract supplied by the paramedian branch from the basilar artery. To our knowledge our case is the first reported case of ischemic Gasperini syndrome caused by involvement of the paramedian branch from the basilar artery.

We predicted a lesion in the pontine tegmentum based on these symptoms. Of the 13 cases including ours, 12 were examined by CT or MRI, and 11 of the 12 demonstrated foci in the mediolateral tegmental pons (Cases 2 and 4-13, additionally the cerebellar peduncle was partially involved in cases 6-8 and 11). These images are in accord with the lesion location estimated by clinical observation (Fig. 1E). These small lesions consist of 10 pontine infarctions and 2 instances of pontine bleeding. In the 10 ischemic stroke patients, 8 cases (Cases 2-4, 6-7, and 11-13) had conventional or MRA. Among these 8 cases, Cases 4, 6 and 7 were reported to have venous angioma, vertebral artery occlusion, and basilar artery stenosis, respectively. Case 3 had used oral contraceptive and was pregnant at the time of her stroke. The remaining 4 ischemic cases have no cardioembolic source or no abnormality of the vertebral and basilar artery, and they are thought to be caused by small vessel disease associated with diabetes mellitus, hyperlipidemia or hypertension, as reported in cranial nerve palsies due to small brainstem lesions (9, 10).

The core neurological signs of Gasperini syndrome are paramedian branch from the anterior inferior cerebellar artery (AICA) and the paramedian branch from the basilar artery are candidates for feeding arteries of the VIth and VIIth nuclei and surrounding areas (11). The long circumferential branch from the AICA supplies the spinal tract or spinal tract nucleus of the trigeminal nerve (Fig. 1D) (12). The paramedian branch from the basilar artery supplies the ventral trigeminothalamic tract. In 10 cases of ischemic Gasperini syndrome including our 2 cases, 6 patients had facial hemihypalgesia only on the affected side (Cases 3, 4, 6, 7, 9 and 13), indicating involvement of the spinal tract or spinal tract nucleus of the trigeminal nerve supplied by the long circumferential branch of AICA. Furthermore, 4 of 10 ischemic cases (cases 2, 6, 7 and 11) presented with cerebellar ataxia of the affected side, a finding that Gasperini did not mention. These cases were reported as AICA infarction presenting as Gasperini syndrome (2, 6). Finally, the long circumferential branch of AICA seemed to be the responsible artery in 8 of 10 patients with ischemic Gasperini syndrome (Cases 2-4, 6, 7, 9, 11 and 13). In contrast, only our Case 1 had facial hemihypalgesia only on the unaffected side, indicating involvement of the ventral trigeminothalamic tract supplied by the paramedian branch from the basilar artery. To our knowledge our case is the first reported case of ischemic Gasperini syndrome caused by involvement of the paramedian branch from the basilar artery.

### References


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