Hemiparesis caused by vertebral artery compression of the medulla oblongata

Case report

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The case is reported of a patient with progressive left hemiparesis due to vascular compression of the medulla oblongata. Metrizamide computerized tomography cisternography revealed that the left vertebral artery was compressing and distorting the left lateral surface of the medulla. Compression was surgically relieved and symptoms improved postoperatively. Neurological and symptomatic considerations are discussed in relation to the topographical anatomy of the lateral corticospinal tract.

KEY WORDS • vertebral artery • hemiparesis • microvascular decompression • metrizamide CT cisternography • corticospinal tract • medulla oblongata compression

The concept of neurovascular compression of cranial nerves as a cause of cranial nerve dysfunction has recently gained wide acceptance. It is well known to be a cause of trigeminal neuralgia and hemifacial spasm. There are some reports of eighth nerve dysfunction and glossopharyngeal neuralgia due to neurovascular compression. For hemifacial spasm and trigeminal neuralgia, microvascular decompression surgery is currently the treatment of choice, and there seems to be little question that this operation yields better results with fewer complications than any previous method. Although several reports have described hypertension as related to direct arterial compression of the medulla oblongata, to the best of our knowledge there have been no reports of hemiparesis due to vascular compression of the brain stem.

This report describes a patient with longstanding and gradually progressive hemiparesis, in whom metrizamide computerized tomography (CT) cisternography revealed vertebral artery compression and distortion of the medulla oblongata. Surgical vascular decompression was achieved, and the symptoms improved satisfactorily. It is believed that this is the first reported case with hemiparesis caused by neurovascular compression.

Case Report

This 53-year-old man was admitted to the neurological service of Tokyo Metropolitan Neurological Hospital on May 18, 1983, for evaluation of a progressive left hemiparesis. In July, 1976, he had urinary frequency and subsequently developed urinary retention. He also experienced an abnormal cold sensation in his left leg. In late July, he was admitted to a regional hospital and mild lumbar disc protrusion at L4-5 and L5-S1 was noted. Although he received no specific treatment, the abnormal cold sensation and urinary retention subsided within 1 month. During that period he experienced transient left peripheral facial palsy lasting for 1 week. Two months later, he noticed a weakness in his left leg. A neurological examination at a university hospital in September, 1978, revealed weakness in his left arm and leg. Myelography of the entire spinal canal was performed, but the only significant finding was slight protrusion of the lumbar discs L4-5 and L5-S1. The weakness progressed rather slowly, and after 5 years the patient could no longer walk.

Examination. On admission, neurological examination disclosed left hemiparesis with associated hyperreflexia. On the left side, the deep tendon reflexes of the biceps, brachioradialis, triceps, quadriceps, and gas-
trocnemius muscles were all remarkably exaggerated, and ankle clonus was observed. Pathological plantar reflex was elicited on the left side. The muscles of the left arm and leg, especially in the distal portion, were atrophic. No cranial nerve involvement was observed other than deviation of the tongue toward the left. No sensory disturbance was detected at that time.

Electromyography revealed a disuse atrophy pattern. Conventional enhanced CT showed a thick vertebral artery located close to the midline in the posterior fossa (Fig. 1 left). Vertebral angiography disclosed that the left vertebral artery was thick and tortuous, and seemingly described a loop as it ascended in the posterior fossa (Fig. 1 right). Vertebral artery compression of the medulla oblongata was suspected upon neurosurgical consultation and, on August 11, the patient was transferred to the neurosurgical service for further evaluation and possible surgical treatment.

To define the positional relationship between the artery and the brain stem, we performed metrizamide CT cisternography,1,3,7,18 together with intravenous injection of 65% meglumine diatrizoate (Angiographin) (Figs. 2 and 3). This procedure revealed severe vascular compression by the vertebral artery of the left side of the medulla oblongata as it ascended from the foramen magnum. The medulla was displaced to the right and was kidney-shaped, with a deep indentation on the left side (Fig. 2A-E). The CT density of the vascular shadow increased following intravenous injection of Angiographin. The slice at the cerebellopontine angle level showed that the artery was in contact with the seventh and/or eighth nerve (Fig. 2F).

Operation. The patient was operated on in mid-September, 1983. A left lateral suboccipital craniectomy and C-1 laminectomy was performed with the patient in a lateral recumbent position. The foramen magnum was opened, and the craniectomy was extended laterally as far as the mastoid air cells. The left tonsil of the cerebellum was retracted to expose the lateral aspect of the medulla oblongata, the 11th and 12th cranial nerves, as well as the left vertebral artery (Fig. 4). The vertebral artery was compressing the left side of the medulla in its ascending course after penetrating the dura. Consequently, the medulla was distorted, with the left lateral surface deeply indented. The artery then turned laterally and further ascended toward the cerebellopontine angle. The 12th cranial nerve was displaced dorsally by the left vertebral artery (Fig. 4 upper). The vertebral artery was gently separated from the medulla with a microsurgical probe. There was no perforating branch in the 10-mm length along the vertebral artery between the first dentate ligament and the branching point of the posterior inferior cerebellar artery. Two small Dacron slings were placed around this part of the artery and their ends were anchored with stitches to the dura mater at the edge of the foramen magnum, using the technique described by Fukushima,19 so that the vessel was separated from the lateral
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FIG. 2. Preoperative metrizamide computerized tomography cisternograms demonstrating the offending left vertebral artery (arrows) and the distorted medulla oblongata (A–E). In the left cerebellopontine angle, the artery is in contact with the seventh and/or the eighth nerve (F).

FIG. 3. Reconstructed image of the computerized tomography scans delineating the incurving left vertebral artery distorting the medulla oblongata (arrows').

surface of the medulla. To ensure repositioning of the artery and decompression of the medulla, a piece of Teflon felt was inserted between the artery and the left lateral surface of the medulla (Fig. 4 lower). The patient tolerated the procedure well.

Postoperative Course. The patient’s hemiparesis gradually improved following the operation. He was able to extend his left fingers and flex his left ankle dorsally, which he had been unable to do preoperatively. The strength of the flexor and extensor muscles of the left knee and of the hip improved remarkably. Hypertension was not observed. Postoperative metrizamide CT cisternography (Fig. 5) showed that the left vertebral artery was repositioned and well separated from the medulla. A high-density shadow was seen, which corresponded to the Teflon felt inserted between the vessel and the medulla. At his 5-month postoperative check, the patient was able to walk without assistance, and ankle clonus was no longer observed.

Discussion

This patient presented with weakness on the left side, ipsilateral to the vascular compression of the medulla oblongata. The left vertebral artery was thought to be compressing the corticospinal tract immediately below the pyramidal decussation. The metrizamide CT cisternogram showed the vascular compression of the lateral surface of the medulla most clearly on the cut below the anterior pyramid. In the lateral corticospinal tract, after its decussation, the fibers that innervate the lower extremities are located laterally and those innervating the upper extremities are located medially. The fact that paresis began and was more pronounced in the left leg corresponds well to the topographical distribution of these nerve fibers. The symptom of 12th nerve palsy can also be explained by the direct vascular compression of the left 12th nerve, which was observed during the surgery. Retrospectively, the episode of left facial palsy, which lasted only 1 week and was assumed to be of the
Upper: Operative exposure of the left lateral aspect of the medulla oblongata. The offending left vertebral artery (V-A) and the indented medulla are visible. The hypoglossal nerve (XII) is displaced dorsally by the artery. PICA = posterior inferior cerebellar artery. Lower: View after decompression. The left vertebral artery is suspended by Dacron slings anchored to the dura mater. A piece of Teflon felt is inserted between the artery and the medulla.

Peripheral type from the patient’s description, might be related to the left vertebral artery being in contact with the seventh nerve in the left cerebellopontine angle, which was clearly demonstrated in the metrizamide CT cisternogram (Fig. 2F).

Though mild protrusion of two lumbar discs (L4-5 and L5-S1) was also present in this case, the weakness in the lower extremity was of the upper-motor-neuron type with associated hyperreflexia. Moreover, electromyographic examination revealed a disuse atrophy pattern without any neurogenic pattern abnormalities. Therefore, any contribution of the lumbar disc lesion to the symptoms was considered to be insignificant. Urinary retention, recorded in the initial stage of the history, might have been related to the disc lesion.

Other nerve fiber tracts adjacent to the lateral corticospinal tract in the lower medulla oblongata, which might well be involved in the pathological state of the present case, are the spinocerebellar tract and the lateral spinothalamic tract. No symptom of these tracts was
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observed except for the transient episode of an abnormal cold sensation in the medial side of the patient's left calf. The compression may have involved the more ventral part of the medulla more than the course of the spinothalamic tract, and only a small number of fibers of the spinocerebellar tract might have been affected.

To date, there have been no previous reports of weakness caused by vascular compression of the medulla oblongata. The present case was initially suspected of being some degenerative disease, but metrizamide CT cisternography revealed vascular compression in the posterior fossa. We found metrizamide CT cisternography to be indispensable in detecting the abnormal anatomical relationship of the vessels and the brain stem. As this diagnostic tool becomes more frequently used in neurological services, we believe that cases similar to the one presented here will be found, and accumulation of such experiences may indicate a distinct clinical entity.

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References


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