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Clinical note

Hemibody pain relieved by microvascular decompression of the contralateral caudal medulla: Case report

Brian A. Simpson [a,*], Anthony Amato-Watkins [a], Margaret D. Hourihan [b]

[a] Department of Neurosurgery, University Hospital of Wales, Cardiff, UK
[b] Department of Neuroradiology, University Hospital of Wales, Cardiff, UK

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ABSTRACT

Microvascular decompression (MVD) of cranial nerves has become an established treatment for trigeminal and (vago)glossopharyngeal neuralgia and for hemifacial spasm. The authors present the case of a 64-year-old man who had a 3.5-year history of severe, drug-resistant hemibody pain with sensory and autonomic disturbance. The ipsilateral trigeminal, cochlear, and glossopharyngeal function also was affected. The contralateral posterior inferior cerebellar artery was seen on magnetic resonance imaging to be indenting the caudal medulla anterolaterally, causing displacement. After MVD of the medulla, there was an immediate and complete resolution of the pain and almost complete resolution of the sensory and autonomic disturbances. The pain later recurred mildly and transiently. The residual symptoms had resolved by 2 years.

1. Introduction

Dandy first described arterial contact with the trigeminal nerve in 1929 [5,6], and vascular compression of the facial nerve was first linked with hemifacial spasm (HFS) in 1947 [4] and 1948 [24]. Relief of trigeminal neuralgia (TN) by decompressive procedures was reported in 1952 [46] and 1959 [11]. Gardner and Sava [12] reported decompression of the facial nerve for HFS in 1962, and Jannetta subsequently developed and promulgated microvascular decompression (MVD) for TN and HFS [2,15,16,30]. MVD of cranial nerves is now an established treatment for TN [1,45,49] HFS [31], and (vago)glossopharyngeal neuralgia [21,23,39,41]. It also has been reported to relieve tinnitus [9,32,37]; disabling positional vertigo [18,33]; spasmodic torticollis [38,50]; nervus intermedius (geniculate) neuralgia [3,28]; a hyperactive gag reflex [40]; oculomotor [34], trochlear [42,44] and abducent [7] nerve dysfunction; and hypertension [19]. Vascular compression of the medulla has been reported to cause motor [22,29,43,47,48] and sensory [22,29,47] impairment, including decreased pain and temperature sensation [14], and paresthesia [43]. We present what we believe to be the first reported case of pain associated with vascular compression of the caudal medulla and relieved by MVD.

2. Case report and results

2.1. Presentation and examination

A retired police officer, then age 58 years, noticed altered sensation and pain throughout the right side of his face with hypersensitivity in the external auditory meatus and throat and watering of the right eye immediately after a bout of intense coughing. He had a respiratory infection at the time. Over the next few weeks, the sensory disturbance spread down the entire right side of his trunk and lower limb to the toes and then the upper limb, finally affecting the hand and its digits. The entire right side felt unpleasantly different from the left and was continuously painful, both aching and burning, with usual intensity 7 of 10 (4 of 10 when distracted, peaking at 8 to 9 of 10). The pain was always worst in his face, head, and upper quadrant. There were no paroxysmal, pulsatile, or postural elements. The right side of his head and neck felt subjectively hot, prompting the use of ice packs to aid sleep. Temperature perception was abnormally enhanced to both hot and cold on the right. The entire right side sweated excessively; the left sweated normally. There was no abnormal blushing. His right eye watered almost continuously, and more when the pain was

* Corresponding author. Address: Department of Neurosurgery, University Hospital of Wales, Cardiff CF14 4XW, UK. Tel.: +44 (0) 29 2074 2708; fax: +44 (0) 29 2074 2560.
E-mail address: briansimpson48@btinternet.com (B.A. Simpson).

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particularly severe, but with no other triggers. Hyperacusis developed (but no tinnitus), plus a constant feeling of a fish bone being lodged in the right side of his throat, along with an intermittent but persistent cough. Focal pressure on the right knee, elbow, or front or back of the right chest wall exacerbated the whole pain. Tramadol, gabapentin, and fluoxetine were of limited benefit. Tramadol was discontinued. He had stopped driving and playing his saxophone, and rarely left the house. He described the pain as "disabling," and occasionally expressed suicidal ideation. Once established, his condition remained stable for more than 3 years. There was no motor or bladder involvement. After nearly 3 years he began to have several diarrheal episodes every day, which responded to codeine phosphate 30 to 60 mg/d (started 6 months before the operation). Thorough investigation revealed no cause for the diarrhea, and it was attributed to "an autonomic disturbance." Hypertension was diagnosed 4 years before the onset of the pain and was controlled with a beta-blocker and an angiotensin-converting enzyme (ACE) inhibitor. The latter was started more than 2 years before the onset of the diarrhea. At presentation, the only abnormal neurological examination finding was of enhanced temperature sensation throughout the right side compared with the left, but not thermal allodynia. He was tall, and of normal, muscular build.

A reaction to contrast medium during magnetic resonance (MR) imaging 9 months before surgery manifested initially as an increased feeling of heat on the right side of his face and head with ipsilateral erythema, before spreading throughout the right side and then generalizing after several minutes, accompanied by pruritus. MR imaging was performed on a 1.5-T magnet. The T2-weighted axial (Fig. 1A–D), coronal (Fig. 2A, B), and sagittal (Fig. 2C, D) imaging demonstrated indentation and displacement of the left side of the lower lateral medulla by a loop of posterior inferior cerebellar artery (PICA) originating from the left vertebral artery. A contrast-enhanced MR angiogram (Fig. 3) demonstrated that the left vertebral artery was markedly dominant and the right vertebral artery was small and ended in the right PICA. No other abnormality was demonstrated.

2.2. Surgery

The surgery took place 3.5 years after the onset of symptoms. The patient accepted the speculative nature of the surgery and freely gave his informed consent. Using the "park bench" position, a low left retromastoid craniectomy was extended into the foramen magnum. Upward retraction of the cerebellar tonsil revealed a loop of PICA indenting the anterolateral aspect of the caudal medulla. Two delicate branches were long enough to allow the artery to be lifted away from the medulla safely, revealing a distinct, transverse, grayish-colored groove. The intra-arterial pressure tended to keep the PICA in the groove. A pad of Teflon felt was placed between the vessel and the medulla.

2.3. Postoperative course

On waking from the anesthetic, the patient reported that his right side now felt normal and that all the pain had gone, except for his (medial) right foot. His right side felt identical to the left. There were now no trigger points and no hypersensitivity. The effect was sustained and not related simply to postoperative analgesic medication. The hyperacusis also was relieved, and neurological examination was normal. Six to eight weeks postoperatively, a partial and gradual recurrence of the pain began in the shoulder,

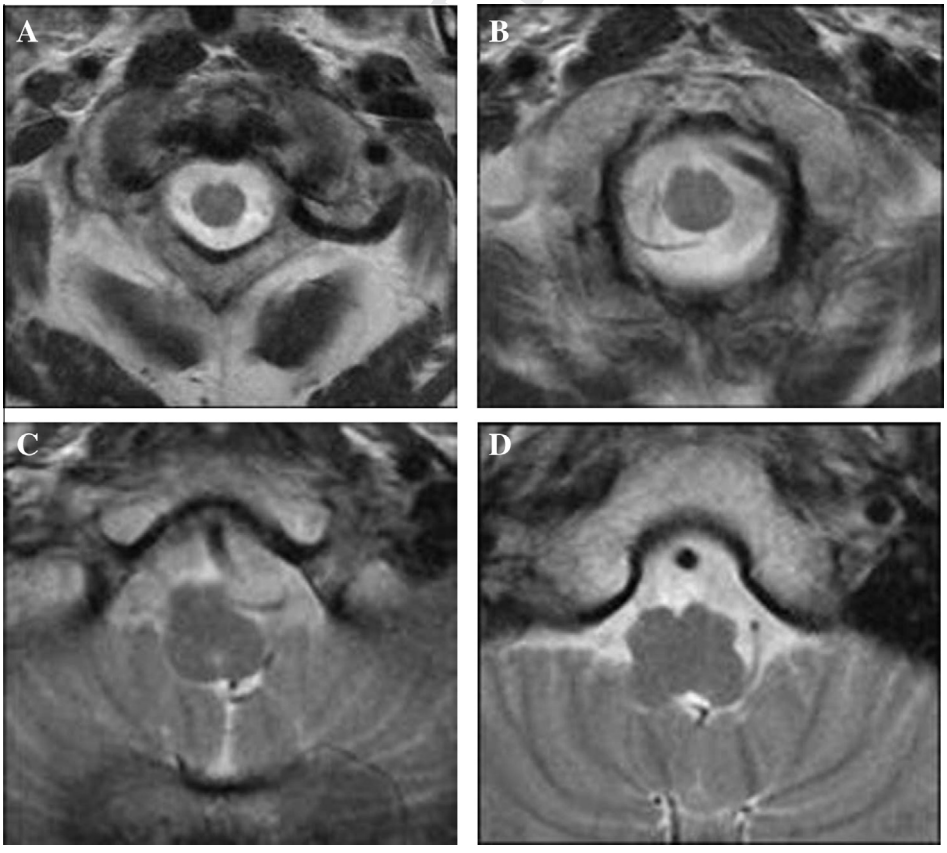

Fig. 1. (A–D) Axial T2-weighted magnetic resonance images (consecutive 5-mm slices) demonstrating indentation and lateral displacement of the left side of the lower anterolateral medulla by a loop of posterior inferior cerebellar artery, arising from the dominant left vertebral artery.

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