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Clinical Study

Microvascular decompression for hemifacial spasm secondary to vertebrobasilar dolichoectasia: Surgical strategies, technical nuances and clinical outcomes

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ABSTRACT

Hemifacial spasm (HFS) due to direct compression of the facial nerve by a dolichoectatic vertebrobasilar artery is rare. Vessels are often non-compliant and tethered by critical brainstem perforators. We set out to determine surgical strategies and outcomes for this challenging disease. All patients undergoing surgery for HFS secondary to vertebrobasilar dolichoectasia were reviewed. Hospital records, clinic notes and radiographic imaging were collected for outcome measures. Seventeen patients (eight males, nine females) were identified. Sixteen patients (94%) were treated with Teflon pledgets (DuPont, Wilmington, DE, USA) and one (6%) patient had a vascular sling placed around a severely diseased vertebral artery. All patients had significant reduction in symptoms and 82% of patients had complete resolution of symptoms (average follow-up: 41.4 months). One patient suffered persistent facial nerve paresis and swallowing difficulty. Two other patients suffered a 1 point decrease in the House–Brackmann facial nerve grading scale. Four patients (23%) required re-operation (infection, cerebrospinal fluid leak, and two patients with delayed recurrence of HFS). Of the latter, one patient required repositioning of a Teflon pledget and another patient underwent a sling decompression. There were no perioperative strokes or death. Excellent relief of symptoms with acceptable preoperative morbidity can be achieved using Teflon pledgets alone in most cases. In recalcitrant cases, sling transposition can be used to further augment the decompression. Careful attention must be paid to prevent vascular kinking and preserve brainstem perforators.

1. Introduction

Hemifacial spasm (HFS) is a disfiguring condition characterized by episodic, rhythmic, myotonic contraction of muscles innervated by the seventh cranial nerve. In the majority of patients, symptoms are secondary to arterial compression from loops of normal posterior circulation vasculature. Rarely, an elongated, enlarged and atherosclerotic vertebrobasilar junction in a crowded posterior fossa compartment can result in aberrant compression of the lower cranial nerves and brainstem. Microvascular decompression in cases of vertebrobasilar dolichoectasia (VBD) are surgically challenging for many reasons: a partially calcified, fixed, and tortuous vertebrobasilar junction tethered by critical brainstem perforators is often difficult to mobilize, and transposition using pledgets or vascular slings may result in vascular kinking or occlusion, increasing the risk for ischemic injury [1–3]. Limited data are available in the neurosurgical literature on the ideal surgical approach and long term efficacy of this complex subset of patients. We report our institutional experience with HFS secondary to VBD, and discuss the technical nuances in treating this challenging pathology. To our knowledge, this represents the largest surgical series in the literature to date.

2. Methods

All patients undergoing microvascular decompression of the facial nerve for HFS at the Barrow Neurological Institute were reviewed from 2007–2014. Preoperative vascular imaging and operative reports were used to identify those patients with VBD as the source of nerve compression. Computed tomography angiography, magnetic resonance angiography, MRI or formal posterior circulation angiography were analyzed for the vascular configuration of the posterior circulation in these patients. Those

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with VBD were identified using the criteria previously established by Smoker et al. [4]. The basilar artery was considered elongated if during its intracranial course it (a) was lateral to the margin of the clivus or dorsum sellae, or (b) bifurcated above the plane of the suprasellar cistern. Additionally, the basilar artery was considered ectatic if its diameter was greater than 4.5 mm [4–6]. Hospital records and clinic notes were reviewed to collect demographic data, medical history, hospital stay and relevant clinical history including initial symptoms, symptom duration, preoperative medical management, diseased vessels involved, and pre and post-operative House–Brackmann facial nerve grading scores. Clinic notes were used to document patient pain relief, functional outcome, and long term complications.

2.1. Surgical approach

All patients in this series underwent a retrosigmoid craniotomy to access the diseased vessel in the following manner: patients are secured to the operating room table in a supine position with rotation of the head contralateral to the side of approach and subtle lateral extension of the neck. In patients without supple necks, a park bench position is utilized. Preoperative Stealth navigation (Medtronic Sofamor Danek, Inc., Memphis, TN, USA) is used to tailor a linear vertical incision. All patients underwent standard monitoring of motor evoked potentials, brainstem auditory evoked potentials, sensory evoked potentials, and facial nerve monitoring. After dural exposure, the inferior edge of the transverse sinus and medial edge of the sigmoid sinus are identified, and either a cruciate or a diagonal "T" shaped dural opening is made in the direction of the sigmoid–transverse sinus junction to maximize exposure. Using the operating microscope, the arachnoid is sharply dissected and the cisterns are opened widely with early release of cerebrospinal fluid to obviate the need for preoperative lumbar drain placement in all cases.

Surgical dissection is carried out to the cerebellopontine angle, and early manipulation of dolichoectatic posterior circulation vasculature is avoided until the facial, acoustic, and vestibular nerves are clearly identified in order to prevent avulsion or further compression of tethered cranial nerves and brainstem in a crowded posterior fossa with distorted anatomy. The facial nerve course from the brainstem root exit zone into the cerebellopontine angle cistern and out through the internal auditory canal is identified. Bed rotation and/or microscope angulation are often necessary to visualize the length of the facial nerve and properly identify areas of vascular compression. Blunt microinstruments are used to peel the offending vessel from the facial nerve. The mobility of the vessel is assessed while care is taken not to place significant tension on basilar perforators. For short segment compression, Teflon felt pledgets (DuPont, Wilmington, DE, USA) are placed to cushion the nerve from pulsatile compression of the diseased vessel (Fig. 1, 2). For long segment compression due to severely diseased vessels, sling decompression is considered to translocate the diseased vessel away from the facial nerve. In such cases, a Durasis (Cook Biotech, West Lafayette, IN, USA) or Gore-Tex (W.L. Gore, Newark, DE, USA) sling is looped around the offending vessel and sutured to the overlying dura, or around a screw fixed into the occipital condyle (Fig. 3). Special attention is paid towards minimizing kinking of the vessel or it perforators after mobilization.

In addition to an institutional review, an extensive literature search was performed. A systematic review of the MEDLINE, PubMed, Google Scholar, and OvidSP-Wolters Kluwer Heath search engines was conducted. All articles with the keywords "vertebrobasilar dolichoectasia" and "hemifacial spasm" from 1972–2014 were reviewed. All clinical reports or surgical series were reviewed. Additionally, surgical series for "hemifacial spasm" were reviewed to identify those patients with VBD who were part of a larger series of HFS.

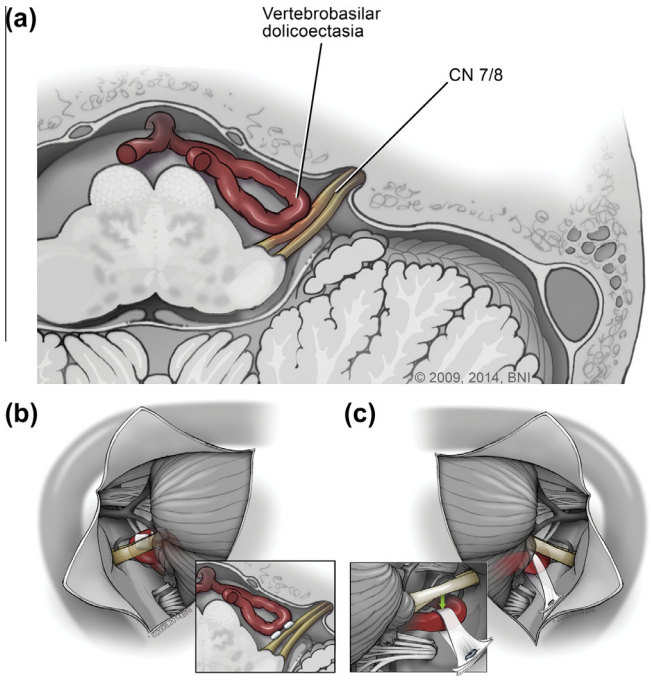

Fig. 1. (a) Diagram illustrating compression of the facial nerve by a large vertebrobasilar junction. Surgical decompression can be performed using either (b) Teflon interposition graft (DuPont, Wilmington, DE, USA) or (c) a Gore-Tex sling (W.L. Gore, Newark, DE, USA) secured to the petrous bone using a screw. CN = cranial nerve. *Used with permission from Barrow Neurological Institute* (This figure is available in colour at http://www.sciencedirect.com/).

3. Results

Seventeen patients (eight males, nine females) with VBD-induced HFS underwent microvascular decompression at our institution between the years 2007 and 2014. Their relevant clinical data are summarized in Table 1. At the index procedure, 16 patients (94%) underwent pledget placement (two cotton, 14 Teflon) decompression (Fig. 1, 2) and one (6%) patient with a severely diseased vertebral artery received a vascular sling (Fig. 1, 3). The majority of cases were left sided (71%), and 13 (76%) patients were managed medically for at least 6 months prior to surgical intervention. Average symptom duration prior to surgical intervention was 76 months. In addition to compression of the facial nerve, seven (41%) patients had evidence of brainstem compression on preoperative imaging. All patients had intraoperative evidence of VBD which correlated with preoperative imaging findings. In addition to HFS, one patient had concurrent trigeminal neuralgia, one patient had concurrent glossopharyngeal neuralgia, and three patients had significant hearing loss prior to surgery. Nine (52.9%) patients had hypertension, six (35.3%) had hypercholesterolemia, and two (11.8%) had diabetes. All patients underwent a retrosigmoid craniotomy, and the average preoperative House–Brackmann score improved from 1.5 to 1.2 at last follow-up, with an average follow-up period of 41.4 months. Two patients suffered a 1 point decrease in the House–Brackmann facial nerve grading scale. One patient suffered from postoperative deafness on the same side of the surgery. There were two postoperative complications (intracranial abscess, cerebrospinal fluid leak) both of which necessitated re-operation. Two patients, both treated with Teflon pledget interposition, had delayed recurrence of symptoms at 30 months and 120 months. The former was found to have a