Intracranial Meningioma at the Foramen Magnum Presenting as Bow Hunter's Syndrome via V4 Segment Compression

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Introduction

BACKGROUND

Bow Hunter's syndrome (BHS) is vertebrobasilar insufficiency resulting from dynamic vertebral artery compression during physiologic head rotation or extension. Most reported cases arise from degenerative changes affecting the V2–V3 segments of the vertebral artery within the cervical spine. Tumor-related BHS is exceedingly uncommon, and compression of the intradural V4 segment by an intracranial lesion has not been previously described.

OBJECTIVE

To describe the first reported case of Bow Hunter's syndrome caused by an intracranial foramen magnum meningioma producing dynamic compression of the intradural V4 vertebral artery, and to highlight the diagnostic considerations and surgical management of this rare etiology.

Case Description

A 77-year-old man presented with reproducible episodes of dizziness, right-sided weakness, and hemisensory loss triggered by leftward head rotation and resolving in neutral position. MRI demonstrated a homogeneously enhancing intradural extramedullary mass at the left foramen magnum, consistent with meningioma, causing compression of the cervicomedullary junction and intradural V4 segment of the vertebral artery. The patient underwent midline suboccipital craniotomy with C1 laminectomy and partial occipital condylectomy for microsurgical resection. Gross-total tumor removal achieved direct vertebral artery decompression, resulting in complete postoperative resolution of positional vertebrobasilar symptoms.

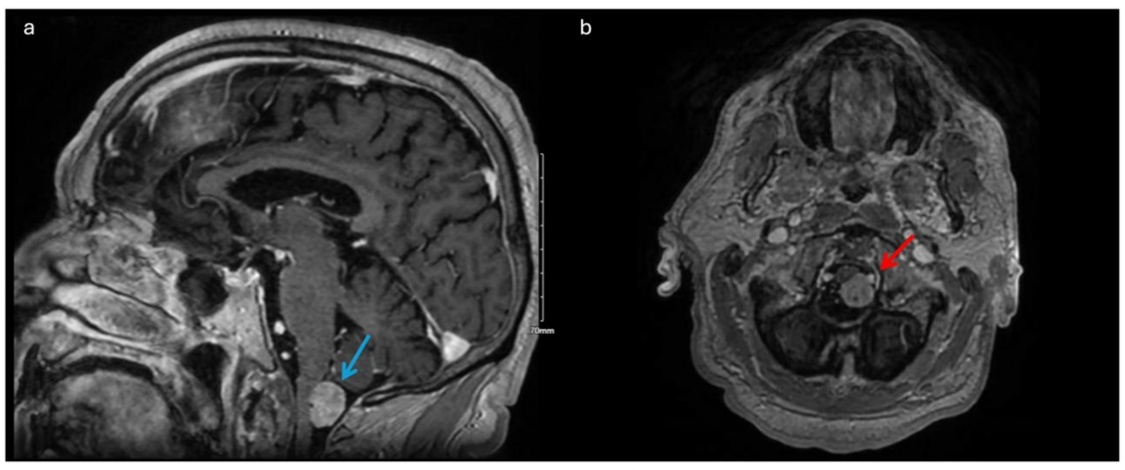

Figure 1. Preoperative T1-weighted postcontrast MRI (sagittal A, axial B) demonstrating a well-circumscribed, homogeneously enhancing intradural extramedullary mass compressing the cervicomedullary junction (blue arrow) and left vertebral artery (red arrow)

Surgical Technique

The patient underwent a midline suboccipital craniotomy with C1 laminectomy using neuronavigation and intraoperative neuromonitoring. After Y-shaped dural opening, the intradural tumor was identified at the left foramen magnum and internally debulked to facilitate mobilization. Limited partial resection of the left occipital condyle was performed to improve lateral access to the tumor–vertebral artery interface. The lesion was carefully dissected from surrounding arachnoid planes, and its dural attachment was coagulated and divided, allowing gross-total resection with direct decompression of the intradural V4 vertebral artery. A thin dural layer was preserved to minimize cerebrospinal fluid leak, and a watertight layered closure was achieved.

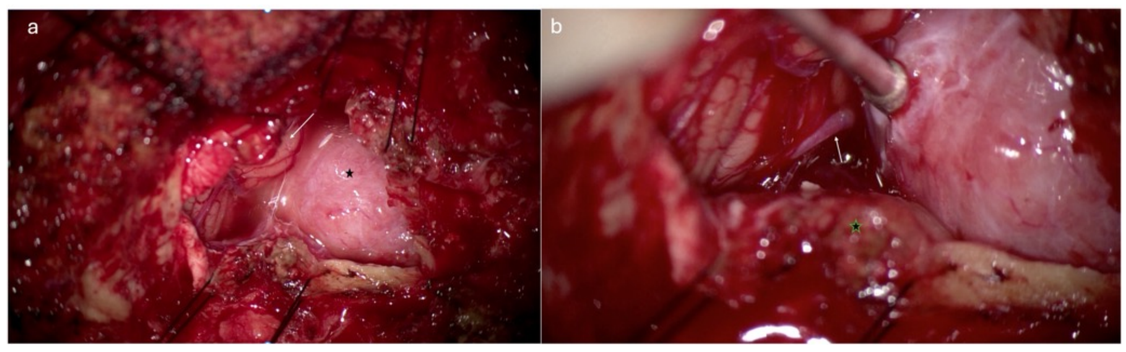

Figure 2. Intraoperative microscopic views after suboccipital craniotomy. (A) Intradural mass on the patient's left (black star) abutting the right cerebellar tonsil (arrow). (B) The right posterior inferior cerebellar artery (arrow) courses along the medial tumor margin with displacement of the left cerebellar tonsil (star).

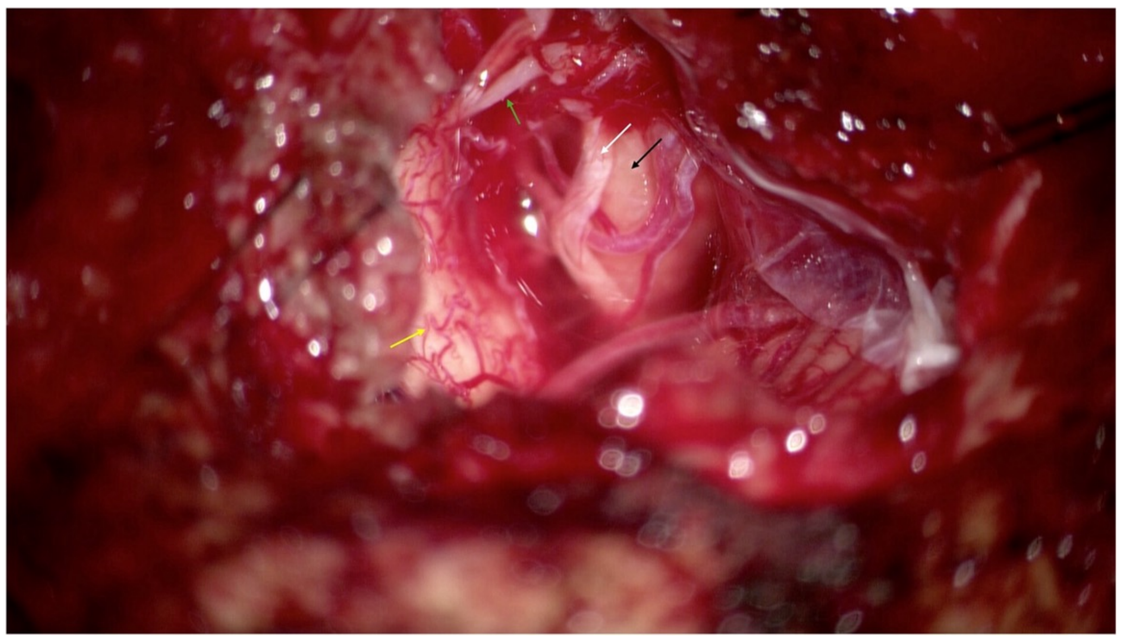

Figure 3. Final intradural view following tumor resection showing decompressed cervicomedullary junction (yellow arrow), intradural left vertebral artery entering the dura (black arrow), and spinal accessory nerve with ascending trunk (white arrow) and lateral dorsal rootlets (green arrow).

Discussion

Bow Hunter's syndrome (BHS) is a cause of vertebrobasilar insufficiency resulting from dynamic vertebral artery compression during physiologic head rotation. Most reported cases are related to degenerative cervical pathology affecting the V2–V3 segments, while tumor-related causes are very rare. Tumor-associated BHS at the V4 segment has not been previously reported. This case illustrates foramen magnum meningioma producing position-dependent compression of the intradural V4 segment, resulting in classic BHS symptoms. The patient's reproducible neurologic deficits with head rotation and immediate resolution in the neutral position highlight the importance of recognizing this characteristic clinical pattern.

Diagnostic evaluation of BHS typically includes vascular imaging capable of demonstrating dynamic occlusion, with digital subtraction angiography considered the gold standard. However, cross-sectional imaging plays a critical role in identifying structural lesions and defining their relationship to the vertebral artery and cervicomedullary junction. In this case, MRI was sufficient to identify the causative lesion and guide surgical planning, emphasizing the need to consider pathology at the craniovertebral junction when standard cervical imaging does not reveal an etiology.

Surgical decompression remains the definitive treatment for symptomatic BHS when a structural cause is identified. Posterior microsurgical resection allowed complete tumor removal and direct decompression of the intradural vertebral artery without the need for fusion, preserving cervical motion. The patient experienced complete and sustained resolution of symptoms without postoperative complications. This case broadens the range of recognized mechanisms of BHS and underscores the importance of including intracranial pathology in the differential diagnosis of patients with reproducible, position-dependent vertebrobasilar symptoms.

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