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Atlantoaxial wiring hardware failure resulting in intracranial hemorrhage and hydrocephalus: illustrative case

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BACKGROUND Atlantoaxial sublaminar wiring has many known complications related to hardware failure, but intracranial hemorrhage is a rare complication.

OBSERVATIONS A 61-year-old female patient with prior atlantoaxial sublaminar wiring for odontoid fracture nonunion experienced decreased level of consciousness due to a subarachnoid and subdural hemorrhage of the posterior fossa with intraventricular extension and hydrocephalus. Rupture of the sublaminar wire with intramedullary protrusion was the cause of the hemorrhage. The patient was treated with ventriculostomy for hydrocephalus and occipital cervical fusion for spinal instability, along with removal of the broken wire and drainage of a hematoma.

LESSONS This uncommon cause of intracranial hemorrhage highlights an additional risk of atlantoaxial sublaminar wiring compared with other atlantoaxial fusion techniques. In addition, this case suggests cervical instrumentation failure as a differential diagnosis of subarachnoid and subdural hemorrhage of the posterior fossa when a history of prior instrumentation is known.

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KEYWORDS sublaminar wiring; subarachnoid hemorrhage; hydrocephalus; spinal

The technique of sublaminar wiring (Brooks wiring) has been well established for atlantoaxial instrumented fusion in the context of instability caused by various pathologies.[1–8] Although different hardware constructs have been developed with different advantages,[2,3,5,9–12] sublaminar wiring continues to be employed in various settings.[1] Atlantoaxial sublaminar wiring–related complications have been described, including hardware failure, nonunion, durotomy, spinal cord injury, and brain injury.[8,13–22]

We describe a rare but serious and potentially fatal complication of atlantoaxial sublaminar wiring failure with an atypical presentation of a progressively comatose patient with posterior fossa and upper spinal subdural and subarachnoid hemorrhage resulting in acute hydrocephalus. We also review the literature of spinal hardware failure in relation to subarachnoid hemorrhage.

Illustrative Case

A 61-year-old female presented to the hospital with neck pain, nausea, vomiting, and no focal deficits. She developed a significant headache with rapidly progressive somnolence requiring intubation. Computed tomography (CT) of the head demonstrated subarachnoid and intraventricular hemorrhage with moderate hydrocephalus and signs of intracranial hypertension. Imaging also revealed a high cervical and posterior fossa subdural hematoma (Fig. 1). She was transferred to our institution with a high-grade subarachnoid hemorrhage and hydrocephalus.

She had a prior history of type 2 odontoid fracture nonunion treated with an odontoid screw and subsequent nonunion treated with atlantoaxial Brooks sublaminar wiring at an outside institution. She had sustained a minor trauma in a swimming class 2 weeks

ABBREVIATIONS CT = computed tomography; EVD = external ventricular drain.
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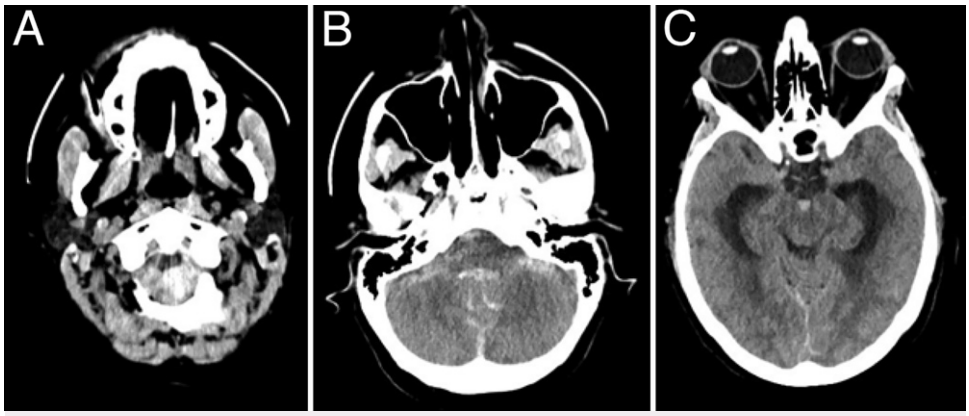

FIG. 1. Axial noncontrast CT of the head showing upper cervical subdural hemorrhage (**A**), posterior fossa subarachnoid hemorrhage (**B**), intraventricular hemorrhage in the fourth ventricle (**B**), and hydrocephalus (**C**).

before consulting, with an acute exacerbation of neck pain for the past 2 days after a mild effort.

CT angiography of the head and cervical spine revealed a fractured cable protruding into the spinal canal and subdural hematoma at the level of C1 and C2. There was no identified vascular lesion (Fig. 2). Digital subtraction angiography eliminated the presence of aneurysms or pseudoaneurysms in the posterior circulation.

She was immediately treated for hydrocephalus with the insertion of an external ventricular drain (EVD). She was then treated surgically for removal of sublaminar wires and underwent C1–2 laminectomy, drainage of the subdural hematoma, and occipitocervical fusion (C0–5). Intraoperatively, the wire was noted to have protruded through the dura into the spinal cord, with the source of bleeding identified in the posterior spinal cord veins.

She experienced a good recovery with no sensory or motor deficits, and the EVD was progressively weaned and removed. She was discharged after 2 weeks with a hospitalization complicated by delirium, hyponatremia, and urinary tract infection. At 2-month follow-up, she had improved cognitively with no residual hydrocephalus and no neurological deficits. Solid occipitocervical arthrodesis was documented 3 years after her operation. However, shortly after that and following an incidental fall with head trauma, she experienced cervicalgia, and multiple undisplaced fractures of the left transverse processes of C7–T3 were found on a CT scan of the cervical spine. She was managed conservatively and remained stable 1 year later, without significant symptoms.

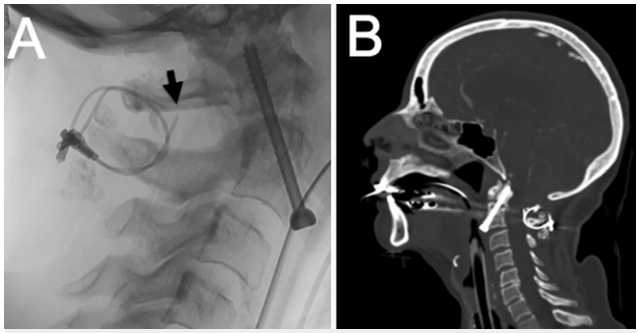

FIG. 2. A: Lateral radiograph of the upper cervical spine with the *arrow* highlighting the fractured sublaminar wire. **B:** Sagittal contrast CT angiography showing fractured sublaminar wire and the absence of other vascular lesions.

Discussion

Complications of Brooks posterior wiring are numerous and well described in the literature.[2,3,5,13–22] Their occurrence can be perioperative, early postoperative, or late postoperative. The most common complication is nonunion, reaching 30% in some series.[3,7] Nonunion can contribute to increased loading of the cables because stability is not achieved, and it can precipitate a fatigue failure and loosening.[15] Cables positioned with excessive bowing of the cable arc into the spinal canal can cause spinal cord compression,[22] and direct spinal cord injury has been described.[20]

Delayed complications can be related to nonunion where excessive long-term hardware stress can lead to loosening of the cables and delayed subluxation,[16] spinal cord compression,[22] fracture of the C1 posterior arch,[14] or even syringomyelia.[21] Delayed cable fracture can also occur as a complication of sublaminar wiring; reports have described cable fracture and migration of the cable into the spinal cord occurring months to years after surgery and the potential to cause transient or permanent neurological deficit, meningitis due to cerebrospinal fluid leakage, or, as seen in our case, a subarachnoid hemorrhage.[8,13,15–17,19]

Observations

In the present case, cable facture led to penetration of the thecal sac and laceration of the posterior spinal cord vessels, resulting in significant intracranial subarachnoid, intraventricular, and subdural hemorrhage with concomitant hydrocephalus requiring EVD placement. Only one similar case of subarachnoid hemorrhage related to atlantoaxial sublaminar wiring was described, by Kakarla et al. in 2010,[17] but a case of wire migration through the dura into the brainstem causing neurological deficits has also been described, by Koziarz et al. in 2017.[8] These cases highlight the risk of sublaminar wiring with the potential for direct brain and spinal cord injury, durotomy, or hemorrhage; other off-midline lateral mass screw fixation constructs limit the risk of injury directly to the spinal cord.[2,3,10,11] These constructs are also more stable than wires under tension.[12] In our case, surgical treatment involved both the drainage of a subdural hematoma and additional instrumented fusion to treat the underlying spinal instability and pseudoarthrosis.

Lessons

Although rare and infrequent, this complication of intracranial hemorrhage from sublaminar wire breakage highlights two important points. First, in the investigation of subarachnoid hemorrhage or subdural hemorrhage for a patient with a prior history of spinal surgery and cervical instrumentation, instrumentation failure should be considered as an etiology for hemorrhage. Second, we describe an additional potential disadvantage of sublaminar wiring compared with other screw fixation techniques (i.e., direct spinal cord injury in the case of wire loosening or rupture).

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Shedid, Benomar, Westwick, Obaid. Acquisition of data: Benomar, Westwick, Obaid, Nzokou. Analysis and interpretation of data: Obaid, Nzokou. Drafting the article: Benomar, Westwick, Obaid, Yuh. Critically revising the article: Shedid, Westwick, Obaid, Yuh. Reviewed submitted version of manuscript: Shedid, Obaid, Nzokou. Approved the final version of the manuscript on behalf of all authors: Shedid. Administrative/technical/material support: Obaid. Study supervision: Shedid, Obaid, Yuh.

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