Delayed myelopathy after remote C1–2 sublaminar wire fixation: illustrative case

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BACKGROUND Atlantoaxial sublaminar wiring complications, both early and delayed, have been documented. However, delayed neurological compromise 27 years after successful fusion is a rare but possible occurrence.

OBSERVATIONS A 76-year-old male, who had undergone C1–2 sublaminar wire fusion for atlantoaxial instability in 1995, presented with symptoms of progressive right arm weakness, falls, and incontinence of bowel and bladder over a 1-week period. Initial imaging workup revealed bowing of the C1–2 sublaminar wires resulting in cervical spinal cord compression and T2-weighted signal changes. A C1–2 laminectomy was performed to remove the wires and decompress the spinal cord with improvement in the patient's neurological status.

LESSONS This rare case highlights the potential for delayed cervical myelopathy and cord compression from sublaminar wires, even after a successful fusion. In patients with a history of sublaminar wiring who experience new neurological deficits, it is essential to evaluate the hardware for migration.

https://thejns.org/doi/abs/10.3171/CASE23126

KEYWORDS sublaminar wire; myelopathy; hardware failure; C1–2 fixation

The use of wires in spinal fixation dates back to the 1890s.1 Although advancements in spinal arthrodesis have reduced their use, sublaminar wires are still occasionally used in scoliosis correction.2 The Brooks and Jenkins C1 and C2 sublaminar wire fixation technique aimed to improve fusion rates using iliac crest bone graft.3 Although the complication rate with sublaminar wires is relatively low, wire migration has been reported in up to 7% of patients and some patients experience transient neural changes during placement.4 Wire loosening and fracture can also occur and lead to significant morbidity, including nonunion and spinal cord, dura, and brainstem injuries.4–12 The longest reported intervals of wire loosening and bowing compared with breakage were 3 and 10 years, respectively.6–8 Here, we describe a case of spinal cord injury due to sublaminar wire migration after 27 years.

Illustrative Case

A 76-year-old male with a history of C1–2 sublaminar wire fusion in 1995 after an assault presented with right arm weakness, bilateral upper extremity numbness, right leg numbness, balance disturbance, and incontinence of bowel and bladder that had developed after a fall 1 week prior to presentation. Additional medical history was notable for chronic kidney disease, type 2 diabetes mellitus, ulcerative colitis, prostate cancer, and a right rotator cuff injury. Physical examination revealed right upper extremity weakness with motor examination as follows: hand intrinsics 4−/5, grip 4−/5, triceps 4−/5, biceps 3/5, and deltoid 2/5. The right lower extremity demonstrated mild weakness with 4/5 strength throughout. The left side was normal in strength, and reflexes were increased with Hoffman’s sign bilaterally.

Computed tomography (CT) imaging of the cervical spine demonstrated anterior displacement of the C1–2 wires (Fig. 1A) with an area of altered density concerning for pannus formation (Fig. 1B). CT also demonstrated a solid posterior fusion bed with no evidence of pseudoarthrosis (Fig. 1B). Magnetic resonance imaging demonstrated neural compression at the level of C1–2, which was worse on the left and associated with a hyperintense T2-weighted signal change in the cervical spinal cord (Fig. 1C and D).
Continuous monitoring was initiated with a mean arterial pressure goal of 85 mm Hg, and the patient was subsequently taken to the operating room for a C1 laminectomy for decompression and removal of the wires (Fig. 1E). Upon removal of the C1–2 lamina and underlying hypertrophied ligamentum flavum, braided wires were observed within the canal and noted to be directly compressing the spinal cord (Fig. 1F). A wire cutter was used to divide the wires, and with minimal traction, they were removed by pulling on the free ends. Removal of the wires led to significant decompression, and no dural injury was noted. Postoperatively, the patient’s motor exam improved on the right with 2/5 strength in the deltoid and 4/5 strength throughout. Additionally, the sensory exam revealed improved sensation throughout the left upper extremity relative to the right upper extremity.

Discussion

Throughout the evolution of spinal fixation with sublaminar wires, numerous complications have been reported, including dural injury, spinal cord injury, brainstem injury, subarachnoid hemorrhage, vertebral fracture, and most frequently, nonunion. Improvements in implantation techniques, wire engineering, and risk factor identification have reduced the risk of sublaminar wire insertion; however, this procedure has fallen out of favor compared with more modern instrumentation techniques. Goll et al. demonstrated that an increased depth of initial wire insertion, lateral anatomical wire insertion (lamina-facet junction), and improper curvature of sublaminar wires are important factors contributing to the risk of spinal cord injury with sublaminar wiring. As a result, a midline approach with the removal of the spinous process combined with a wire curved such that the radius is at least equal to the lamina width minimizes the depth of insertion. Some authors have also recommended the use of braided wires given their improved flexibility, strength, and fatigue-resistant qualities as compared with monofilament wires. However, despite the reported advantages of braided cables, several cases of loosening and breakage have been reported. Several theories regarding the cause of wire loosening and encroachment on the spinal cord have been proposed. One theory suggests that wire loosening and subsequent compression is believed to be caused by chronic wire pressure wearing a groove into the lamina. This allows the wire to loosen, return to a circular shape because of its elastic properties, and encroach upon the spinal cord. Garcia and Gorin suggested that nonunion, which increases wire tension through excessive movement, may give rise to premature wire fatigue and/or loosening.

Observations

In the present case, with a stable construct of 27 years, we hypothesized that gradual loosening of the wires, as described by Sudo et al., shifted the wires into a compressive position leading to the formation of iatrogenic cervical stenosis. The combination of loose sublaminar wires and cervical stenosis predisposed our patient to developing central cord syndrome secondary to a fall from standing despite a successful C1–2 bony fusion. The longest previously reported case of wire migration was 10 years; thus, we seek to emphasize the importance of recognizing that wire constructs likely lose stability over time and enter a phase of increased vulnerability. Therefore, it is also important to recognize that wire stability may decrease over time and that lower forces than expected may be necessary to shift a loose wire construct and cause central cord syndrome.

Lessons

In the setting of acute neurological decline in patients with a history of sublaminar wiring, a high degree of suspicion for hardware failure should
be maintained. The magnitude of force required to displace sublaminar wires remains unclear and should be expected to be low, especially in cases of prolonged implantation. In the case of wire migration or failure, prompt surgical management to relieve neural compression and/or remove migrated hardware should be considered to maximize recovery.

References

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: Paul, Miller, Stone. Acquisition of data: Miller, Stone. Analysis and interpretation of data: Miller, Stone. Drafting of the article: all authors. Critically revising the article: all authors. Reviewed submitted version of the manuscript: Paul, Miller, Stone. Approved the final version of the manuscript on behalf of all authors: Paul. Administrative/technical/material support: Stone. Study supervision: Stone.

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