Surgical management of atlantoaxial nonunions

CURTIS A. DICKMAN, M.D., AND VOLKER K. H. SONNTAG, M.D.

Division of Neurological Surgery, Barrow Neurological Institute, St. Joseph's Hospital and Medical Center, Phoenix, Arizona

A variety of surgical techniques have been used for internal fixation to treat atlantoaxial instability, with failure rates ranging from 4% to 50% reported in the literature. Patients with rheumatoid arthritis and os odontoideum have particularly high rates of nonunion after C1–2 fixation.

There are several biomechanical reasons for C1–2 nonunions: C1–2 fixation has the widest range of motion of any spinal motion segment, and this motion is increased significantly when there is pathological instability present. Failure to adequately control C1–2 motion during bone healing has led to a number of strategies to improve the fusion rates. Adjunctive use of a halo brace or internal fixation with rigid transarticular screws has been promoted to improve the success rate with surgery.

This article reviews patients who have developed nonunions after C1–2 internal fixation procedures. Pre-disposing factors to nonunion and management strategies to salvage a C1–2 pseudarthrosis are examined in detail.

Clinical Material and Methods

Patient Selection

The medical records and radiographs of patients treated by the authors for nonunion after C1–2 fixation procedures during the last decade were reviewed. Sixteen individuals were identified with C1–2 nonunions: 10 were referred by physicians from other facilities, and six patients developed nonunions after treatment at our institution. The patients’ clinical characteristics, neurological examinations, and radiographic studies; the surgical techniques employed; and the clinical, radiographic, and neurological outcome were reviewed. Clinical examinations, radiographic studies, and patient interviews were performed for follow-up evaluation.

Patient Population

The 16 patients (10 men and six women) had a mean age of 43.7 years (range 20–77 years). Follow up extended from 12 to 79 months (mean 35 months). The condi-
tions responsible for atlantoaxial instability included rheumatoid arthritis in five, os odontoideum in seven, transverse ligament disruption in two, and odontoid fracture nonunion in two. The preoperative atlantoaxial subluxations were measured on flexion and extension radiographs that were corrected for magnification. The mean atlanto-dental interval was 9.1 mm (range 7–13 mm).

Prior to revision surgery by the authors, a total of 20 surgeries had been performed among the 16 patients; all resulted in C1–2 nonunions. Thirteen individuals had one prior failed surgery, two had two prior failed surgeries, and one had three prior failed surgeries.

Operative Techniques Associated With Nonunion

Table 1 displays the surgical methods associated with C1–2 nonunions in the 20 failed operations. Seventeen pseudarthroses had C1–2 wire fixation, one had Halifax clamps, one had a unilateral transarticular screw with a C1–2 cable fixation, and one had an onlay graft with no internal fixation. Patients underwent a total of 18 wiring procedures: 17 were without additional hardware, one had a unilateral titanium C1–2 transarticular screw in conjunction with a wire cable. The wiring techniques associated with nonunions included the Gallie method in eight surgeries, the interspinous method in six, and the Brooks method in four. The type of wire associated with the C1–2 nonunions included monofilament No. 20 wire in 11 surgeries, double-stranded twisted No. 24 wire in four, and multistranded wire cables in three.

The postoperative orthoses that were used for the 20 prior surgeries in the C1–2 nonunions included seven halo braces, one sternooccipital mandibular immobilizer (SOMI), 11 Philadelphia collars, and one Minerva jacket.

<table>
<thead>
<tr>
<th>Methods</th>
<th>No. of Operations</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1–2 wire fixation</td>
<td>17</td>
</tr>
<tr>
<td>Halifax clamps</td>
<td>1</td>
</tr>
<tr>
<td>unilateral transarticular screw &amp; wire cable</td>
<td>1</td>
</tr>
<tr>
<td>onlay graft, no internal fixation</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>20</td>
</tr>
</tbody>
</table>

The bone grafting techniques associated with the C1–2 nonunions included autogenous iliac crest bone grafts in 16 patients, allograft iliac bone in one, and no bone graft in three. Among the three patients without bone graft, two had methyl methacrylate with wire, and one had Halifax clamps without any graft.

There were 31 risk factors for nonunion among 10 patients. Six patients had no risk factors for nonunion that could be identified. Among the remaining 10 patients, multiple factors contributed to nonunion including four patients suffering from malnutrition, four taking steroids, eight taking nonsteroidal antiinflammatory drugs, three taking cytotoxic and immunosuppressive drugs, seven who smoked cigarettes, and five with osteoporosis.

Neurological presentations included seven individuals with myelopathy and nine with occipital radicular pain. All patients also had neck pain at the time of presentation of their pseudarthrosis.

Table 2 displays the sites of pseudarthrosis. Among the 20 surgical procedures, 13 instances of pseudarthrosis developed between the C-1–graft interface (Fig. 1). No instances of failure occurred solely at the C-2–graft interface. In the one patient with allograft, pseudarthroses were present simultaneously between C-1 and C-2 and the allograft. Three individuals had complete resorption of their bone grafts. All three patients who had no bone graft placed at surgery developed hardware failure. One patient had an onlay graft without hardware because cables cut through the bone of C-1 and C-2 at the time of her initial surgical procedure.

Table 3 displays the types of hardware failure that occurred among the C1–2 nonunions. There were a total of 19 procedures that developed failed hardware: 13 failures were due to broken wires or cables; three had wire or cable cut through the bone surfaces like a Gigli saw; two were due to a cable that cut through the C-1 ring; one because a wire pulled through the C-2 spinous process. A

<table>
<thead>
<tr>
<th>Sites</th>
<th>No. of C1–2 Nonunions</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-1–graft interface</td>
<td>13*</td>
</tr>
<tr>
<td>C-2–graft interface</td>
<td>0</td>
</tr>
<tr>
<td>interface between graft &amp; C-1, &amp; C-2</td>
<td>1†</td>
</tr>
<tr>
<td>complete bone graft resorption</td>
<td>3</td>
</tr>
<tr>
<td>no bone graft placed, hardware failed</td>
<td>3</td>
</tr>
<tr>
<td>total</td>
<td>20</td>
</tr>
</tbody>
</table>

* All 13 cases had an iliac crest autograft.
† One case underwent an iliac crest allograft.
flexible cable loosened without breaking in one patient (Fig. 2), a Halifax clamp loosened in one, and a titanium screw broke in one (Fig. 3).

Patient Treatment

A variety of strategies were used to revise the pseudarthroses and to treat the patients’ medical conditions: the pharmacological and metabolic inhibitors of fusion were reduced or eliminated whenever possible; nutritional supplementation was provided; cytotoxic, steroid, anti-inflammatory, and immunosuppressive drugs were discontinued for at least 1 week preoperatively and for 2 weeks postoperatively; and enzyme, hormone, and mineral deficiencies were corrected. Five of the seven cigarette smokers stopped smoking; transdermal nicotine patches and nicotine gum were avoided.

Surgical Revision

Surgical revision of the C1–2 pseudarthroses was performed in all 16 patients (Table 4). All 16 cases had autologous iliac crest bone grafts placed at the time of surgery. New C1–2 fixation was performed in 13 individuals and occipitocervical fixation was performed in three. Revised C1–2 cable or wires were used in conjunction with a halo brace in three patients; each had a construct placed that compressed the bone grafts between the arches of C1–2 (Fig. 4). Ten patients were treated by means of C1–2 transarticular screws. Eight individuals underwent posterior transarticular screw fixations that were supplemented with a cable and a compressed bone graft (Fig. 5). Two patients had anterior atlantoaxial facet screws. Occipitocervical fixation was performed in three patients because one had basilar invagination and two had destruction of the posterior arches of C-1 and C-2 that precluded adequate internal fixation.

Postoperative Treatment

Five of the 16 patients were treated postoperatively with halo orthoses, primarily to protect the fixation from failing acutely in the presence of osteoporotic bone. A postoperative SOMI brace was used in one patient and Philadelphia collars were used in 10. No electromagnetic stimulators, bone morphogenic protein, or demineralized bone was used to induce osteogenesis perioperatively.

Results

There was no operative mortality and no permanent sequelae from any perioperative complications. There were 11 complications in eight patients related to the revi-
sion surgery. Three patients had intraoperative dural tears that were repaired with sutures; one developed a postoperative cerebrospinal fluid (CSF) leak from his neck wound that required a temporary external lumbar drain; the CSF leak ceased without sequelae. Three individuals developed wound infections: there were two superficial infections involving the neck wound and one iliac crest–graft site wound. All the wound infections resolved with local treatment and antibiotic medications. Two patients developed localized cellulitis at a halo pin site that was satisfactorily treated by changing the pin sites and providing local wound care and oral antibiotic medications. One individual developed both pneumonia and a mild myocardial infarction, but fully recovered without sequelae after medical treatment.

Postoperative Follow-Up Study

Postoperative follow up after a mean of 35 months (range 12–79 months) demonstrated that 15 (94%) of the 16 patients had a stable fixation construct. Thirteen patients developed an osseous union; two had a stable fibrous union. One patient had a nonunion that remained in stable alignment for 2 years. He was in an automobile accident 2 years postoperatively that fractured his anterior C1–2 transarticular screws. He subsequently had persistent C1–2 instability and occipital radicular pain, but remained neurologically intact and refused any further surgery. One patient with rheumatoid arthritis developed fused facets at C2–3 and C3–4, which was not clinically significant, but was apparent on postoperative radiographs (Fig. 6).

There were no operative or postoperative neurological complications. Among the seven individuals who had myelopathy, five with neurological defects improved postoperatively, two had stabilization of their myelopathy, and none had worsened neurological function. Among the nine patients with occipital radicular pain, only one had persistent radicular pain; this was the patient with the fractured screws and the nonunion.

All originally presented with neck pain. None of the patients had worse neck pain on follow up; four had persistent neck pain that was not different from their original neck pain; seven had persistent but markedly less neck pain; five had complete resolution of their neck pain.

Discussion

There are a variety of reasons for failure of C1–2 internal fixation procedures. These reasons can be categorized as metabolic, pathological, pharmacological, biomechanical, and technical.

Metabolic, Pathological, and Pharmacological Inhibitors of Fusion

A variety of diseases, nutritional, hormonal, and enzyme deficiencies, and pharmacological agents interfere with bone healing.6,28,32 These factors can be effectively manipulated to facilitate fusion. The strategies that were employed in our patients included discontinuing or reducing cytotoxic agents, steroids, antiinflammatory agents, and immunosuppressive drugs at least 1 week preoperatively and for at least 2 weeks postoperatively. Nutritional supplementation was provided to patients with malnutrition or absorptive deficiencies. Internal medicine and...
endocrinology consultations were invaluable for helping to identify and correct enzyme, hormonal, vitamin, and mineral deficiencies. Patients were strongly encouraged to discontinue the use of tobacco products perioperatively. Nicotine causes a high nonunion rate because it interferes with vascularization of healing bone grafts, therefore transdermal nicotine patches, nicotine gum, and chewing tobacco were also avoided. We did not use electromagnetic stimulators, demineralized bone, or bone morphogenetic protein for salvaging nonunions because of lack of clinical availability or because they have not yet been conclusively demonstrated to clinically augment cervical spinal fusion; however, these may be considered potential adjuncts to the armamentarium for revision surgery.

Biomechanical Reasons for C1–2 Nonunions

The atlantoaxial motion segment has the widest range of motion of any spinal motion segment; the normal 80° to 90° range of motion for bilateral axial rotation accounts for over half of all cervical rotation. Under conditions of pathological destabilization of C1–2, rotational, and translational motions are increased dramatically in all directions. Abnormal translation (subluxations) and increased rotational motion develop. Inadequate control of C1–2 motion during bone healing is an important precipitant of the development of nonunion, whereas rigid fixation of the spine during the immediate postoperative phase of bone healing appears to facilitate fusion. Biomechanical studies have demonstrated that wiring techniques, especially the Gallie methods, are significantly less effective in controlling C1–2 motion, especially axial rotation, than transarticular screws. Alternative strategies that can be used for rigid spinal fixation include external immobilization with a halo brace or internal fixation with transarticular C1–2 screws. These techniques can successfully augment the fixation of a wired bone graft at C1–2. If C1–2 cannot be directly fixed, then an occipitocervical fixation can be performed to internally stabilize the spine.

Technical Reasons for C1–2 Nonunions

There are a variety of technical reasons that cause C1–2 nonunions to occur, and all of these can be surgically prevented: failure to compress the bone graft between the arches of C-1 and C-2, failure to place any bone graft, and failure to use autologous bone are simple and easily correctable reasons for nonunion. Inadequate preparation of the fusion bed, especially inadequate decortication of the C-1 ring, is a preventable cause of nonunion. Thirteen of the nonunions developed at the C-1–graft interface, which implicates pathological motion at C-1 and/or inadequate preparation of the C-1 ring in contributing directly to nonunion. If rigid internal fixation cannot be achieved or if the bone is soft and osteoporotic, a postoperative halo brace should be used.

The types of implants that are used and their techniques of application contribute substantially to nonunion and implant failure. Monofilament wire can fatigue and break relatively easily. Although multistranded wire cables are relatively fatigue-resistant, they can cut through osteoporotic bone. To avoid cables failing in osteoporotic bone, one can use decreased cable tension (6–8 in/lbs of torque) and apply a halo brace postoperatively. Titanium implants (cables or screws) are notch sensitive and are more susceptible to fatigue than their steel counterparts. Despite their imaging advantages, titanium implants may be less desirable than steel implants for patients at risk of implant failure. Additionally, if a rigid internal fixation cannot be achieved intraoperatively, a rigid cervical orthosis should be used postoperatively to control C1–2 motion.

Conclusions

The majority of C1–2 pseudarthroses occur between C-1 and the graft and are associated with failed hardware. Atlantoaxial instability due to failed surgery can be successfully salvaged if the pathological, pharmacological, biomechanical, and technical problems are effectively resolved. Fusion can be achieved in a substantial proportion of revision surgeries. The greatest success can be gained by using autologous bone grafts, adequately controlling C1–2 motion (rigid transarticular screws or a halo brace), compressing the bone graft between the arches of C1–2, meticulously preparing the fusion bed, and optimizing the pharmacological and metabolic factors for promoting bone fusion.

References


C. A. Dickman and V. K. H. Sonntag