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Surgical treatment of nonunited fractures of the odontoid process, with special reference to occipitocervical fusion for unreducible atlantoaxial subluxation or instability

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Abstract Fifty-seven consecutive patients treated surgically for non-united fractures of the odontoid process were reviewed. All patients presented late, exhibiting neurological deficits subsequent to nonunion. Delay in presentation was between 6 and 120 months (mean 32 months) after the original injury, due to missed diagnosis or inappropriate management. Seven patients who were reduced in traction underwent a Gallie atlantoaxial fusion. In the remaining 50 patients who were unreducible, an occipitocervical arthrodesis was performed. They were followed up for a minimum of 2 years, except one who died from postoperative respiratory failure. All patients obtained a solid bony union, includ-

ing two in whom nonunion occurred following atlantoaxial fusion, and occipitocervical fusion was added as a rescue. Thirty-eight patients achieved excellent neurological recovery, nine still had some disability, five retained their neurological deficits and two reported a deterioration. In two patients, a recurrence in a traumatic episode was experienced long after a resolution. Our findings demonstrate that occipitocervical arthrodesis is preferable for unreducible subluxation or instability of atlantoaxial articulation in nonunion of odontoid fractures.

Key words Odontoid process · Spinal injuries · Joint instability · Spinal fusion

Introduction

Fractures of the odontoid process have been reported with increasing frequency in recent years, and account for more than 10% of all cervical spine fractures, or the majority of upper cervical spine injuries in children and elderly patients [27]. Although different methods of treatment have been advocated, the outcomes are not as satisfactory as might be expected. A significant nonunion rate has often been documented, especially in unstable type II fractures according to the classification of Anderson and D'Alonzo [3], when inappropriately treated [4, 10, 14, 29]. On the other hand, fractures of the dens are frequently overlooked and undiagnosed, because the patients are unconscious or intoxicated, or have associated injuries that are more severe and obvious. Occasionally, the clinical symptoms are nonspecific and examination findings

are seemingly benign in patients involved in a low-energy episode. Maltreated or untreated odontoid fractures may develop into nonunion or malunion, thereby leading to atlantoaxial subluxation or instability. When delayed neurological symptoms are caused, surgical management is usually indicated [11, 13]. This study was a retrospective review of 57 consecutive patients who underwent posterior fusion for nonunion of odontoid fractures, particularly occipitocervical arthrodesis for unreducible atlantoaxial subluxation or instability subsequent to nonunion.

Materials and methods

Between 1980 and 1995, 57 consecutive patients were referred to the Changzheng Hospital in Shanghai for evaluation and treatment. There were 38 males and 19 females whose ages ranged from 12 to 64 years. The most common cases of injury were falls

from a height (36 patients), followed by motor vehicle accidents (13 patients), pedestrian accidents (six patients) and sporting injuries (two patients). All patients presented late, 6–120 months (average 32 months) after the original injury.

Of these patients, 39 had been treated with external immobilization in a collar or a plaster cast after the original accident. Thirty-one of them had achieved complete functional recovery without any complaints after initial treatment, but delayed neurological symptoms developed subsequently. The remaining eight of the 39 patients treated in this way experienced a resolution of some neurological deficits, but subsequently suffered a progressive deterioration. There were 18 patients who were completely free of symptoms and accepted no treatment during the initial period following the traumatic event. In three of them, by the time of presentation at our hospital, the injury had long since been forgotten and was only remembered as a result of specific questioning after review of their radiographs.

All patients developed local symptoms (Table 1), but neurological deficits were the major reason for seeking medical attention (Table 2).

The radiographs at the time of presentation at our hospital, including anteroposterior, lateral and open mouth views, showed the lesion. Thirty-five tomographs and 29 computed tomographic (CT) scans were also available to make the diagnosis. There were 51 type II and six type III fractures according to the classification of Anderson and D'Alonzo [3]. Flexion-extension views were available for all patients, demonstrating that fractures were displaced in 53 patients: anteriorly in 38, posteriorly in four, laterally in six, and both anteriorly and posteriorly in five. Of them, significant displacement, defined as an atlas-dens distance greater than 5 mm, was identified in 36 patients and the deformity was fixed in 21. Magnetic resonance (MR) imaging was performed on 26 patients, and spinal cord compression was noted in all these cases.

All patients were placed in tong traction for 1–2 weeks. Seven patients who were reduced in traction underwent a Gallie atlantoaxial fusion (Fig. 1). However, reduction was unsatisfactory in the remaining 50 patients, and sublaminar wiring seemed impossible or difficult because of a high risk of neurological complications. Thus, an occipitocervical arthrodesis [21] was performed in patients with unreducible atlantoaxial subluxation or instability. The autograft bone harvested from the anterior iliac crest was placed between the occiput and the axis after the posterior atlantal arch was resected. An occipital flap was reflected to cover the at-

las and provided an additional layer between the dura and bone graft. In 12 patients, the margin of the foramen magnum was removed at the same time. Postoperative immobilization in the plaster cast continued for 3–4 months.

Results

There was one postoperative death from respiratory failure after occipitocervical fusion. All surviving patients obtained a solid bony union (Fig. 2), except two, on whom an occipitocervical fusion was performed after a failed atlantoaxial arthrodesis and a solid fusion was then obtained. They were followed up for 2–16 years (average, 4 years 8 months) after fusion had been determined. Thirty-eight patients achieved excellent neurological recovery with full ability to walk and return of hand function. Nine patients moderately improved, but still retained some disability. Five patients retained their neurological deficits and two reported a deterioration of their myelopathy following surgery. Two patients returned to their preoperative status in a traumatic episode after a long period during which the neurological deficits had been resolved.

Improvement of the neurological symptoms was assessed by the Japanese Orthopaedic Association (JOA) scoring system for cervical myelopathy [19]. The average recovery rate of JOA score from before surgery to the final follow-up, calculated as proposed by Hirabayashi et al. [18], was 51.4% (range –12.5 to 100%). On the radiographs, the C1-C2 angle [25] was measured before surgery and after fusion was confirmed. The results showed that the average C1-C2 angle improved from 22.3° preoperatively (range 5°–59°) to 19.6° postoperatively (2°–59°).

Discussion

The odontoid process, with its attached ligamentous structures, is the keystone of stability at the atlantoaxial articulation, providing restraint against anteroposterior dislocation in combination with the ligamentous restraints and anterior atlantal arch. According to the rule of thirds [39], the spinal cord and odontoid are each approximately one-third the diameter of the spinal canal, leaving the remaining third as the safety space, allowing for some degree of pathological displacement. However, when the odontoid process is fractured, the proximal fragment of the odontoid may be displaced along with the atlas, and displacement of greater than one-third the diameter of spinal canal will make the spinal cord compressed. Displacement of odontoid fractures will not only lead to compression of the spinal cord, but will also interfere with fracture healing by decreasing the contact area of the fragments. Ryan and Taylor [28] demonstrated that in displaced type II fractures, the contact area between the fragments is less than the radiographs might suggest. Pure posterior displacement of only 39% reduces the contact area by 50%,

Table 1 Local symptoms at presentation

Symptoms	N
High cervical and/or occipital pain	51
Neck stiffness and restriction of motion	42
Neck weakness and instability	47
Dizziness	11
Torticollis	9

Table 2 Neurological deficits at presentation

Deficits	N
Myelopathy	26
Radiculopathy	6
Myelopathy and radiculopathy	17
Myelopathy and cerebellopathy	1
Myelopathy and cranial neuropathy	3
Myelopathy, radiculopathy, cranial neuropathy and cerebellopathy	4

Fig. 1 A–C A patient with a nonunited fracture of the odontoid with symptoms of cord compression. The radiograph on admission showed the anteriorly displaced fracture (**A**). The odontoid fragment was reduced significantly after skeletal traction (**B**) and atlantoaxial fusion (**C**)

Fig. 2 A solid occipitocervical fusion was obtained after surgery

and any additional lateral displacement will further reduce the contact area. Adequate reduction means at least 70% overlap of the fragments in the lateral view, with near anatomical alignment in the anteroposterior view. They recommended that a maximal displacement of 20% in both planes, which would leave a surface contact area of 64%, may be acceptable. Hadley et al. [16] suggested that a statistically significant higher incidence of nonunion is related to an initial displacement of 6 mm or greater. Furthermore, the blood supply to the odontoid process may be disrupted with increasing severity of fracture displacement, thereby contributing to poor fracture healing, although a rich arterial arcade surrounding the odontoid has been demonstrated [2, 31].

The implications of nonunited odontoid fractures and subsequent atlantoaxial subluxation or instability in progressive myelopathy have long been recognized [5–8, 12, 26, 32, 33, 39]. In the past, odontoid fractures were thought to be almost uniformly fatal, and death often occurred as a result of a delayed progressive myelopathy. Osgood and Lund [26] reported that 10 of 55 patients with fractures of the odontoid process in their series did not have paralysis at the time of the initial injury, which subsequently caused death after a second, trivial injury. Schwarz and Wigton [30] also documented two cases of delayed myelopathy, thought to be secondary to a nonunited fracture of the odontoid process with instability. In 1974, Anderson and D'Alonzo [3] found over 50 reported

cases of delayed myelopathy after a literature review. As for the pathophysiological mechanisms, Sherk and Nicholson [33] believed that abnormal micromotion of the odontoid against the spinal cord due to nonunion will lead to thinning of the cord. The repeated subluxations apply compression to the cord from the fragment and/or the displaced posterior atlantal arch. Furthermore, continuous compression may be induced by a fixed subluxation of atlantoaxial articulation. Secondary to long-standing subluxation, the extradural fibrous bands cause additional narrowing of the spinal canal and may be, therefore, another factor of cord compression [40]. Compression also inflicts damage on microvascular blood supply to the cord. It will, combined with direct compression to the cord, lead to progressive irreversible myelopathy. However, patients with nonunion of odontoid fractures present as a clinical spectrum, varying widely in degree of involvement, although atlantoaxial subluxation or instability is sufficient to create symptoms. The neurological deficits may be progressive, intermittent or static. Some begin at the time of injury, whereas others have a delayed onset long after the initial injury [1, 3] or may be asymptomatic. In this series, two patients could not even remember the initial injury. The degree of displacement of the odontoid fracture has been suggested as the main determinant of severity of neurological deficits [34, 41], but we feel that the speed of progression of the displacement may be an important factor in the variability of the clinical spectrum. This may furnish an acceptable explanation as to why the degree of neurological symptoms is often not parallel to the degree of fracture displacement.

When an odontoid fracture is nonunited or malunited, operative treatment is generally required [9, 11, 13, 17, 23], especially for those with progressive instability and neurological deficits. Theoretically, atlantoaxial arthrodesis is a more logical and effective choice in stabilizing a united odontoid fracture and in decompression of neural contents. Preoperative reduction with skeletal traction may be of great assistance in achieving a successful outcome of the surgery. In recent years, a number of new techniques for internal fixation of the atlantoaxial complex and odontoid fracture have been described [15, 20, 25, 37]. However, atlantoaxial arthrodesis is only indicated in a reduced position. In our study, nonunion of

odontoid fracture is mostly associated with significant subluxation or instability of atlantoaxial articulation. For these cases, adequate reduction is difficult or impossible to achieve, and sublaminar wiring atlantoaxial arthrodesis will increase the risk of neurological compromise. Smith and associates [35] suggested that it might be safer to perform an atlantoaxial fusion in situ without sublaminar wiring or an occipitocervical fusion following posterior decompression when a deformity is unreducible by preoperative traction. In this series, decompression was mandatory and preoperative reduction with traction was unsuccessful in most patients with neurological deficits, so an occipitocervical fusion with posterior decompression was performed. This method is also applied as a rescue when atlantoaxial arthrodesis is impossible. In contrast to the insignificant changes of atlantoaxial deformity that result, as defined by the C1-C2 angle, neurological improvement can be expected in most patients. Although some restriction of occipitocervical motion was noted, the patients usually rated the outcome as satisfactory when these restrictions were balanced against their neurological recovery. Transoral anterior decompression has been proposed by some authors [11, 13, 22, 24]. This technique provides a straightforward route for removing the anterior abnormalities, but fails to stabilize atlantoaxial articulation at the same time, so that posterior fusion remains necessary [11]. In the authors' opinion, if patients have unchanged or increased neurological deficit attributable to persistent anterior compression of the odontoid that could be delineated by MRI, after posterior decompression and fusion, a subsequent anterior decompression may be required.

In conclusion, undiagnosed and maltreated fractures of the odontoid process usually lead to nonunion of fractures and subsequent subluxation or instability of atlantoaxial articulation. When the deformities and neurological deficits are progressive, operative treatment is indicated to relieve the compression of the spinal cord and to stabilize the atlantoaxial articulation. Preoperative reduction with skeletal traction should be stressed in order to facilitate atlantoaxial arthrodesis. If the dislocations remain unreducible, occipitocervical fusion is an effective and safe alternative on the basis of posterior decompression. Solid bony union and neurological recovery may be expected with this procedure.

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