Review Article

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Os Odontoideum in Children

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Abstract

Os odontoideum is a rare entity of the second cervical vertebra, characterized by a circumferentially corticated ossicle separated from the body of C2. The ossicle is a distinct entity from an odontoid fracture or a persistent ossiculum terminale. The diagnosis may be made incidentally on imaging obtained for the workup of neck pain or neurologic signs and symptoms. Diagnosis usually can be made with plain radiographs. MRI and CT can assess spinal cord integrity and C1-C2 instability. The etiology of os odontoideum is a topic of debate, with investigative studies supporting both congenital and traumatic origins. A wide clinical range of symptoms exists. Symptoms may present as nondescript pain or include occipital-cervical pain, myelopathy, or vertebrobasilar ischemia. Asymptomatic cases without evidence of radiologic instability are typically managed with periodic observation and serial imaging. The presence of atlantoaxial instability or neurological dysfunction necessitates surgical intervention with instrumentation and fusion for stability.

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Neither of the following authors nor any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this article: Dr. Hedequist and Dr. Mo.

J Am Acad Orthop Surg 2020;28: e100-e107

DOI: 10.5435/JAAOS-D-18-00637

Os odontoideum is a rare clinical entity of the second cervical vertebra, characterized by a circumferentially corticated ossicle separated from the body of C2. The ossicle is typically located dorsal and slightly cranial to the anterior arch of C1. The ossicle is distinct from an acute odontoid fracture or a persistent ossiculum terminale. The etiology is unclear, with support in the literature for both congenital and traumatic causation.[1-7]

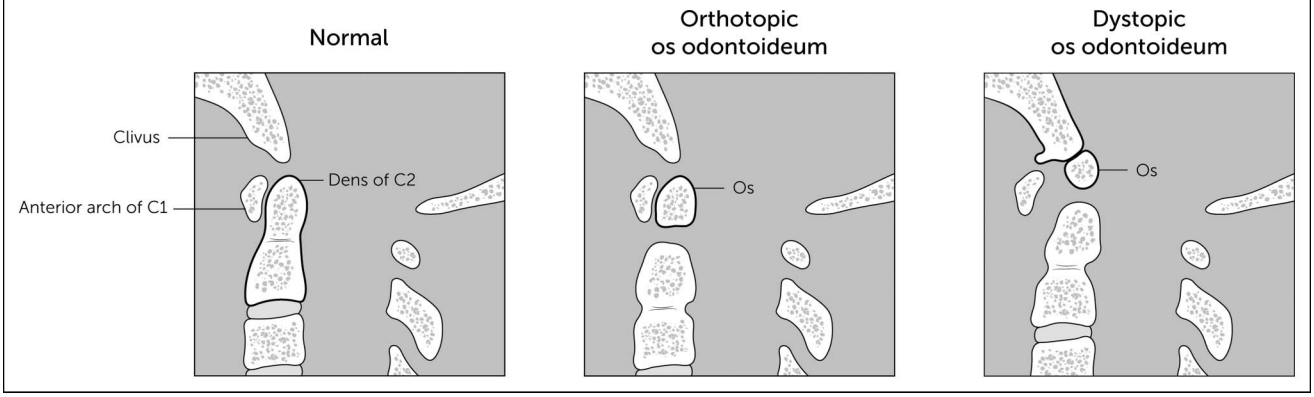
The clinical presentation of patients with os odontoideum is variable. Some patients present asymptomatically and are diagnosed by incidental imaging findings. Others present with diffuse, nonspecific neck pain or neurologic deficits secondary to atlantoaxial instability and spinal cord compression.[8] Owing to the rarity and severity of clinically symptomatic os odontoideum, diagnosis and management are paramount to mitigating morbidity and mortality. This article will review the anatomy and theories of pathophysiology of os odontoideum, as well as diagnosis, management, and treatment options.

Embryology

The embryologic formation of the atlas and axis derives from the segmentation of somites. The fourth occipital sclerotome (proatlas) forms the apex of the dens, the posterior superior arch of the atlas, and the apical, alar, and cruciate ligaments. The first spinal sclerotome forms the anterior arch of the atlas, the posterior inferior aspect of the atlas arch, and the body of the dens. The body of the dens fuses with the proatlas to form the odontoid process. The second spinal sclerotome contributes to the body of the axis and the arches.[9]

When the odontoid process begins to ossify, two centers appear prenatally on each side of a midline that fuses into a single mass by birth.[10] At

Figure 1



Illustrations depicting normal anatomy, orthotopic os odontoideum, and dystopic os odontoideum.

birth, the subdental synchondrosis, a cartilaginous band, separates the odontoid process from the body of the axis.[10] This band lies invaginated within the body of the axis and ultimately contributes to the body of the axis. The subdental synchondrosis is present in most children younger than 4 years and disappears by the age of 8 years.[10-13] The tip of the odontoid process is characterized by a separate ossification center. Typically visualized by age 3 to 4 years, this ossiculum terminale fuses with the body of the dens by the age of 12 years.[11]

Anatomy

The atlantoaxial joint allows for approximately 47° of rotation.[14] The atlas (C1) articulates superiorly with the occiput, and inferiorly, the ring of C1 surrounds the dens of the axis (C2). The atlas does not have a vertebral body. The transverse ligament is the primary stabilizer between C1 and C2, running posterior to the odontoid process, locking it against the anterior arch of C1. A synovial capsule is located anteriorly between the odontoid and the transverse ligament.[14]

The vascular supply to the odontoid process originates from the vertebral artery at the level of the third cervical vertebra, which gives rise to anterior and posterior ascending arteries that anastomose in a vascular arcade surrounding the dens and provides penetrating branches in the region of the alar ligaments. In addition, branches arising from the internal carotid arteries make contributions to the anterior ascending branches.[15]

Fielding et al[10] described two anatomic types of os odontoideum—orthotopic and dystopic. In orthotopic os odontoideum, the ossicle is in the normal anatomic position of the odontoid process and moves with the anterior arch of C1. Dystopic odontoideum is characterized by an ossicle that is located near the base of the occipital bone adjacent to the foramen magnum. The ossicle may be partially fused to the occiput with a variable relationship to the anterior arch of C1 (Figure 1).

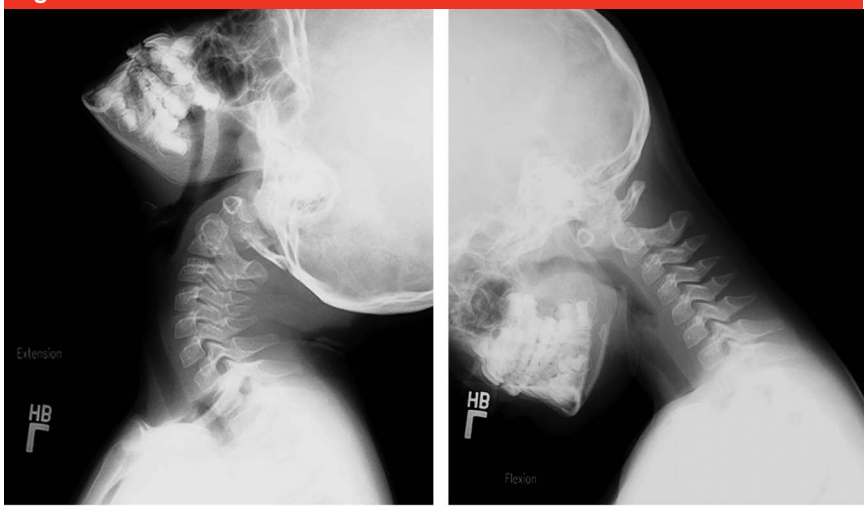
The ossicle of os odontoideum is cranial relative to the expected position of the odontoid tip, bound to the anterior arch of C1 through an intact transverse ligament.[16] This ossicle is located just dorsal and slightly cranial to the anterior arch of C1. Potential atlantoaxial instability arises from the ability of the atlas and the ossicle to luxate on the axis, giving rise to a notable risk of spinal cord injury.[1]

Pathophysiology

The etiology of os odontoideum is controversial, with support in the literature for both congenital and traumatic origins.[1-7] The literature supporting the congenital origin of os odontoideum notes findings in patients without a history of trauma, and often anomalies of the odontoid process are more common in patients with congenital syndromes such as trisomy 21, Klippel-Feil malformation, and skeletal dysplasias.[17] Two predominant mechanisms proposed for the origin of congenital os odontoideum are found.[10] In the first mechanism, the normal secondary ossification center of the odontoid process fails to fuse with the main process of the process.[18,19] In the alternative mechanism, the primary ossification center of the main aspect of the odontoid process fails to fuse with the body of the axis. Failure of fusion between ossification centers, failure of migration, failure of segmentation, or nontraumatic osteonecrosis are believed to be the reasons.[13] In addition, a few reported cases of familiar genetic expression are found.[12,20,21]

Others favor a traumatic origin of os odontoideum. Some patients with a previously normal odontoid

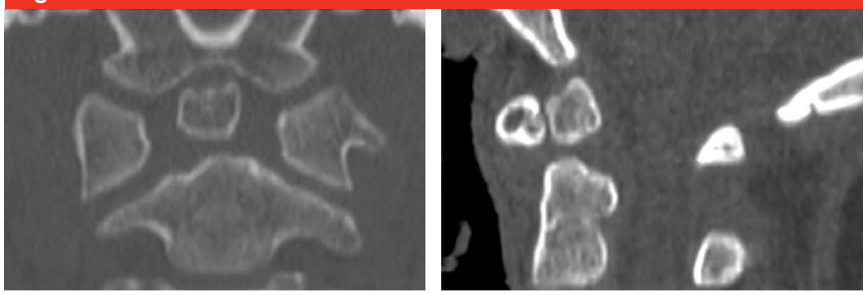
Figure 2



A **B**

A and **B**, Dynamic flexion-extension plain radiographs demonstrating extension (**A**) and flexion (**B**) positions with C1-C2 subluxation on cervical flexion.

Figure 3



A **B**

A and **B**, CT coronal (**A**) and sagittal (**B**) slices showing a well-corticated, circumferential mass consistent with os odontoideum.

process as evidenced on preexisting imaging may develop os odontoideum after recorded trauma.[1,3,5-7,10,22-24] Trauma likely induces either a fracture or an injury to the vascular supply of the odontoid process, leading to localized osteonecrosis. Type II odontoid fractures are believed to disrupt this anastomosis, resulting in a high rate of nonunion.[25]

Stillwell and Fielding theorized that compromise of the odontoid vascular supply leads to bony resorption.[1,10] In a case report by Schuler et al,[16] a 2-year-old girl with trauma was initially imaged with cervical radiographs, which revealed normal findings. Four months later, radiographs demonstrated resorption of the dens with reducible atlantoaxial subluxation. Owing to the intact superior blood supply through the accessory, alar, and apical ligaments, a continued support of the tip is found, whereas compromise of the inferior vascularity through the body of the axis results in localized resorption.[6]

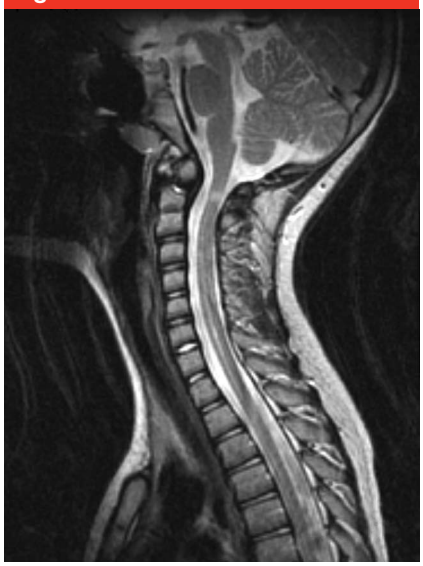
Another potential etiology is that os odontoideum is a consequence, rather than a cause, of hypermobility at the atlantoaxial joint.[26] Stevens et al[27] presented a series of patients with Morquio-Brailsford disease who were treated with occipitocervical arthrodesis and noted regeneration of normal or near-normal odontoid processes and a closure of the neural arch defects. All the patients presented with odontoid dysplasia with hypoplastic dens and detached distal portions. The authors suggest that ligamentous laxity leads to instability and hypermobility, creating abnormal shearing stresses at the cartilaginous stage of development that in turn interferes with ossification and allows for the subsequent interposition of the transverse ligament and establishes a nonunion with hypermobility. The authors theorize that this hypermobility leads to repeated microtrauma, fracturing the odontoid process, which then retains the apical fragment due to the bloody supply from the apical supply.

Clinical Presentation and Diagnosis

Patients with os odontoideum are commonly diagnosed incidentally and present without symptoms. Other children will have clinical symptoms ranging from nonspecific occipital-cervical discomfort to severe findings of myelopathy or progressive quadriparesis. The condition may lead to atlantoaxial instability and subsequent compression of the spinal cord or vertebrobasilar arteries. Atlantoaxial instability can lead to stenosis with resultant myelopathy secondary to vascular compromise, tension of the cord, or bony compression.[8] Vertebrobasilar ischemia can occur due to either kinking or thrombosis of the vertebral arteries.

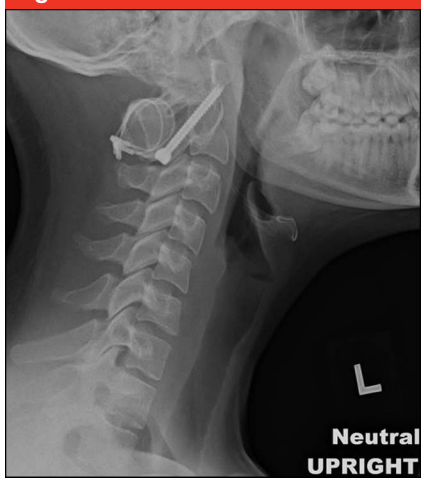
Owing to the rarity of the disease, variable clinical presentations, and high morbidity secondary to atlantoaxial instability, a high index of suspicion for os odontoideum is necessary in patients with reports of

Figure 4



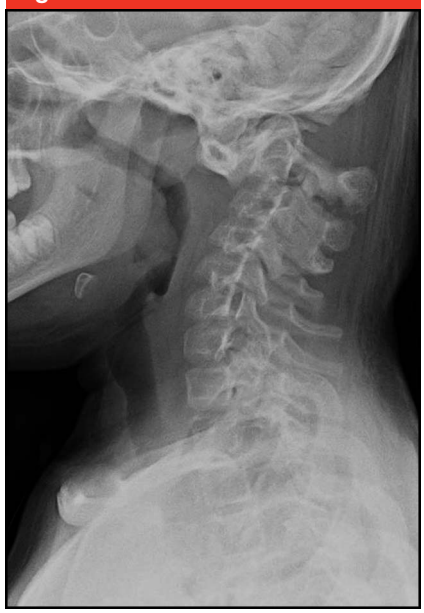
Sagittal T2 MRI demonstrating signal intensity in the spinal cord at the level of C2.

Figure 5



A postoperative plain lateral radiograph demonstrating intact alignment and implant.

Figure 6



A plain lateral radiograph with evidence of C1-C2 instability.

cervical myelopathy. The clinical manifestations of os odontoideum may resemble those found in degenerative disk disease of the cervical spine, cervical spondylosis, and atlantoaxial subluxation. A careful history and physical examination paired with radiographic findings can guide the diagnosis toward os odontoideum.

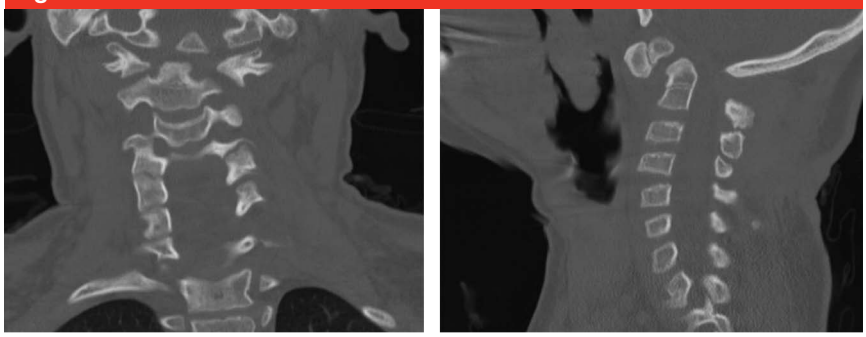
The main diagnosis to consider against os odontoideum is an acute fracture of the odontoid process. Radiographically, os odontoideum is characterized by a circumferentially corticated ossicle that is separated from a hypoplastic odontoid process characterized by a transverse gap. The diagnosis is reinforced with the absence of a recent history of trauma, radiographic absence of sharp fracture lines, and some have suggested the presence of sclerosis and hypertrophy of the anterior tubercle of the atlas. The ossicle is typically located dorsal and slightly cranial to the anterior arch of C1. A rounded or hypertrophic anterior C1 arch can help support a diagnosis of os odontoideum and may be suggestive of chronic instability.[28]

Secondary to the rarity of the disease, a paucity of the literature regarding the sensitivity and specificity of imaging in children with os odontoideum is found. Plain radiographs of the cervical spine are necessary in children with neck pain and/or neurologic dysfunction and first line in the diagnosis of os odontoideum. Preferred views include AP, lateral, and open mouth odontoid views. Dynamic lateral flexion and extension radiographs are useful for assessing atlantoaxial stability.

These views can demonstrate abnormal subluxation of C1 relative to C2 either in extension or flexion.[16,29] Some studies have suggested that the absolute diameter of the spinal canal correlates with poor outcome.[30,31] Spierings and Braakman[7] proposed a critical canal diameter of 13 mm to predict a high risk of cord injury. In their study, a canal diameter of 13 mm or less was associated with a 10-fold risk of the development of permanent cervical myelopathy.

While not required for diagnosis, complex imaging modalities such as CT and MRI provide additional information for the diagnosis and management of os odontoideum. CT scans allow for the delineation of the osseous anatomy of the atlantocervical junction and corresponding relations to the arrangement of the vertebral arteries when used in conjunction with angiography. CT scans are also helpful in defining the anatomy of C1 and C2 in children who are undergoing surgical treatment. MRI provides information regarding the spinal cord and the presence of cord signal changes, which in the face of os odontoideum is an absolute indication for surgery. Hypertrophy of the anterior arch of C1 has been shown to be suggestive of chronic instability.[28] Information regarding the presence of spinal cord compression and pathology guides the diagnosis and treatment of those with os odontoideum. MRI should be routinely obtained in the patient with os odontoideum who presents with neurologic signs or symptoms and can help elucidate findings such as myelomalacia. MRI may also be helpful in patients with painful os odontoideum where there may be synovial inflammation consistent with neck pain.

Figure 7

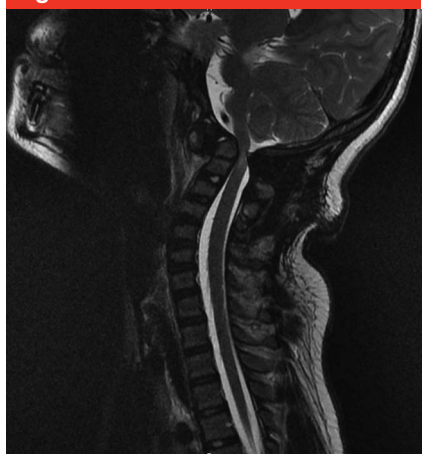


A

B

A and **B**, CT coronal (**A**) and sagittal (**B**) slices CT showing a well-corticated, circumferential mass consistent with os odontoideum. Subluxation of C1 and os odontoideum relative to the C2 body.

Figure 8



Signal enhancement on sagittal T2 MRI within the spinal cord at the level of C2 with notable stenosis of the foramen magnum.

Management

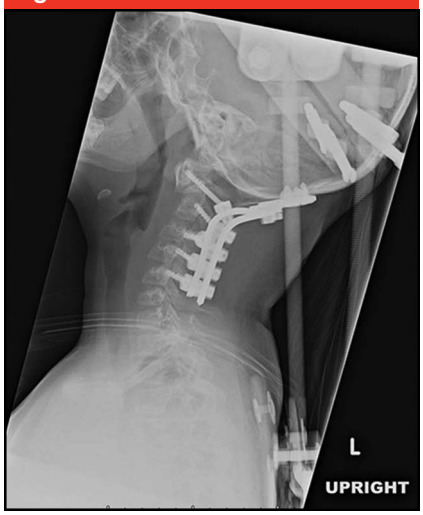
Treatment of patients with os odontoideum can be difficult because of the variable and poorly understood natural course of os odontoideum. Nonsurgical treatment modalities, for those with incidentally diagnosed or asymptomatic os odontoideum without radiographic instability, may include observation with serial imaging and longitudinal follow-up. Initially stable os odontoideum may progress to develop atlantoaxial instability either secondary to natural progression or due to acute trauma.[7,30-32] Serial observation to detect signs of notable symptoms or radiographic instability may decrease the risk of morbidity and mortality. Patients should be counseled regarding the potential risks of instability and neurologic deficits. Patients may additionally benefit from activity modification, including cessation of contact sports. Documented cases of sudden death or notable neurological deficits after minor injuries or even intubation secondary to positioning in those with os odontoideum are found.[26,31,33,34]

Although the use of prophylactic spinal fusion is controversial in those without symptoms, patients with os odontoideum and evidence of instability or myelopathic symptoms are indicated for surgical fusion with a primary goal of achieving cervical spine stability. Historical methods described in the literature include posterior C1-C2 wiring combined with autologous iliac fusion.[35,36] Fielding et al[10] described a series of 27 patients who underwent Gallie type C1-C2 internal fixation and fusion, of which 26 patients had successful results. The successes of these methods are offset by required prolonged immobilization and subsequent increased morbidity, typically postoperative halo immobilization or the use of Minerva jackets for several months.

More contemporary techniques include the use of transarticular screw fixation and polyaxial screw/rod fixation and fusion of C1-C2. Transarticular C1-C2 screw fixation may be used to provide atlantoaxial stability. Transarticular screws follow the path of C2 pars screws. The advantages of this technique include high fusion rates and good biomechanical stability achieved coupled with low profile instrumentation. This is, however, a technically demanding technique that places the vertebral artery at risk.[37] The use of transarticular screws is only possible when the atlantoaxial joint is reduced, which limits the use of transarticular screws in those patients with marked instability or atlantoaxial rotatory subluxation. Klimo et al[31] published a series of 76 patients with os odontoideum who underwent C1-C2 transarticular screw fixation, with a 90% rate of clinical improvement.

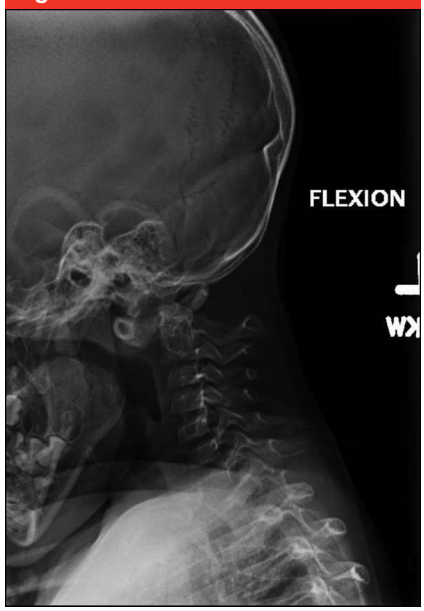
Harms and Melcher described polyaxial screw/rod fixation and fusion in the literature in 2001, popularizing a technique introduced by Goel.[38,39] Lateral mass screws are placed in C1 and connected to C2 pars, C2 pedicle, or translaminar screws to obtain stability. Instrumentation of the C1 and C2 lateral mass and pars allows for intraoperative reduction when necessary. A decreased risk of vertebral artery injury as compared with the use of wiring is found. Goel et al[40] described a series of 160 patients, 48 of whom had os odontoideum. One hundred fifty-seven of these patients had successful stabilization of the atlantoaxial junction. Rigid fixation provided by transarticular screw fixation and polyaxial screw/rod fixation diminishes the need for rigid external postoperative immobilization in older children/adolescents. Adjunctive use of external postoperative immobilization may still be used if compliance to postoperative

Figure 9



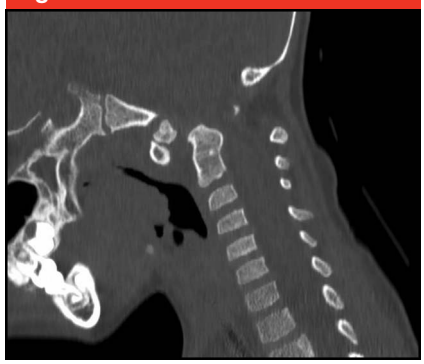
A plain lateral radiograph demonstrating an intact implant and alignment of the C1-C2 level.

Figure 10



Dynamic flexion plain radiographs demonstrating C1-C2 instability.

Figure 11



A CT sagittal slice showing a well-corticated, circumferential mass consistent with os odontoideum. Subluxation of C1 and os odontoideum relative to the C2 body.

restrictions is of concern or additional stability is desired.

Examples of Patients

Patient 1

A 6-year-old previously asymptomatic girl presented with an acute onset of neck pain and paresthesias radiating into her fingers after a gymnastic session. She and her family reported no recollection of trauma. On initial workup, dynamic plain radiographs (Figure 2, A and B) demonstrated C1-C2 subluxation with cervical flexion. CT imaging (Figure 3, A and B) revealed a well-corticated, circumferential body with a transverse gap, consistent with os odontoideum. T2 MRI (Figure 4) imaging showed signal enhancement within the spinal cord at the level of C2.

The patient was taken to the operating room (OR) for C1-C2 transarticular screw fixation. The procedure was well tolerated by the patient, and a halo vest was applied postoperatively. Imaging on follow-up demonstrated stable atlantoaxial levels with stable fixation (Figure 5).

Patient 2

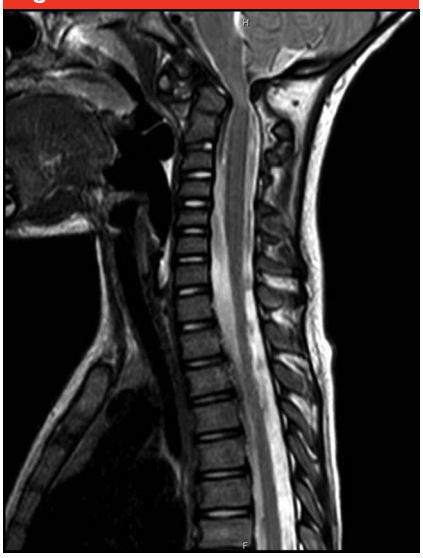
A 10-year-old girl with a history of Down syndrome presented for evaluation of worsening neurologic symptoms. The patient had sustained a fall at the age of six years and was immediately ambulatory afterward, although per family, she developed progressive weakening of her left upper and lower extremities, as well as degreased declination of cognitive and verbal status. Previous medical records from this period were unavailable. One year before presentation, the patient was treated at another institution with occiput to C4 fusion with subsequent failure of fixation and removal of an implant. Initial imaging at the time of presentation included plain radiographs (Figure 6). Preoperative CT imaging (Figure 7, A and B) revealed a well-corticated, circumferential body with a transverse gap at the C2 level consistent with os odontoideum. Os odontoideum was in a forwardly flexed position relative to the body of the atlas. T2 MRI (Figure 8) imaging showed signal enhancement within the spinal cord at the level of C2 with notable stenosis.

The patient was brought to the OR for reduction of os odontoideum as well as placement into a halo vest and traction. The patient's myelopathy gradually improved with sequential traction over the course of 1 month, at which time she was brought back to the OR for occiput to C5 fusion and foramen magnum decompression. On follow-up, the patient continued to have improvement of her myelopathic symptoms and was able to ambulate independently. Postoperative plain radiograph imaging showed stable atlantoaxial levels with intact fixation (Figure 9).

Patient 3

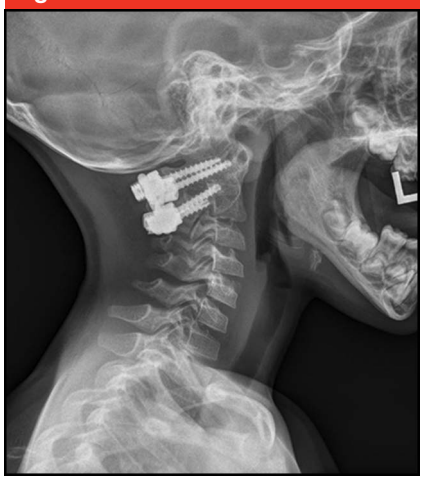
A 4-year-old boy presented for clinical evaluation after an acute, transient episode of quadriplegia lasting approximately 20 minutes. The patient was otherwise healthy and had been playing at the time of the incidence. He had no known history of trauma. Initial workup demonstrated evidence of atlantoaxial instability (Figure 10), and the presence of os odontoideum was confirmed on CT imaging (Figure 11). Signal cord changes and foramen

Figure 12



Signal enhancement on sagittal T2 MRI within the spinal cord at the level of C2 with notable stenosis of the foramen magnum.

Figure 13



A plain lateral radiograph taken at a 6-month follow-up demonstrating an intact implant and a fusion mass.

magnum stenosis were evident on MRI (Figure 12).

The patient was treated surgically with a C1 lateral mass screw and C2 pedicle screw construct and fusion. The patient was placed in halo traction at the beginning of the procedure and kept immobilized for 1 month postoperatively. The patient did well postoperatively with no evidence of neurologic deficit. Implant placement and C1-C2 stability were confirmed on dynamic plain radiographs (Figure 13).

The Authors' Preferred Technique

The operative treatment of os odontoideum in children is an instrumented fusion of C1 and C2. To maintain cervical stabilization during intubation of the patient is our practice. Once the airway has been secured, we place the patient in a halo crown and vest to secure the head and neck for positioning in the prone position. The patient may then be safely turned prone, and the back of the halo vest is removed. Once positioned, reduction of os odontoideum is checked under fluoroscopic imaging. Routine neuromonitoring is used throughout the procedure. To use posterior instrumentation consisting of C1 lateral mass screws coupled to C2 screws which may either be pars/pedicle screws or translaminar screws is the senior author's preference. Preoperative CT scanning is mandatory to assess optimal screw planning. Most children have anatomy which will allow for this construct to be placed, even in children as young as 3 years of age. C1 lateral mass placement is technically challenging due to the dissection of the venous plexus off of the lateral mass screw entry point. Visualization is aided by judicious use of electrocautery, and when needed, a C2 ganglionectomey may be done. The length of the C1 screw must be longer than expected with a notable portion of the screw out of the bone because the dorsal entry point is much further anterior to the dorsal starting point of C2 screws.

Placement of C2 pars/pedicle screws is technically more demanding than that of C2 translaminar screws and places the vertebral artery at risk. However, if the anatomy of the C2 isthmus and position of the vertebral artery allow for C2 pars/pedicle screws to be placed, then it is the author's preference because they are minimally prominent, fit in line with the C1 lateral mass screws, and allow for a greater surface area available for a structural graft between C1 and C2. Placement of C2 screws is greatly facilitated by understanding the anatomy and by avoiding venous plexus bleeding during exposure. We do not use navigation for screw placement and rely on meticulous exposure and fluoroscopic guidance for screw placement. We still recommend placing a structural graft of the autologous iliac crest between C1 and C2 to facilitate fusion. The graft can be secured by suture or cable fixation by placing the cables underneath the ring of C1 and around or through the spinous process of C2. Abundant cancellous grafts can then be placed posterior-lateral between decorticated C1 and C2. We routinely use halo fixation postoperatively for 6 weeks in children younger than 8 years or in older children if there is concern regarding patient activity or noncompliance of collar use.

Summary

Although the natural history of os odontoideum remains unclear, many cases of os odontoideum are diagnosed incidentally. The clinical presentation of os odontoideum is variable, with some patients presenting asymptomatically and others reporting symptoms ranging from nonspecific neck pain to myelopathy or permanent paralysis. The potential for morbidity and mortality and subsequent atlantoaxial instability should prompt a high index of suspicion. Diagnosis is dependent on plain radiographs and a corresponding clinical history and physical examination. Initial diagnosis can be made with plain radiographs consisting of AP, lateral, open mouth

odontoid, and dynamic lateral flexion and extension views. Management can be further guided by CT and MRI to assess the osseous anatomy as well as the spinal cord for compression or signal changes. Surgical intervention is indicated for those with clinical symptoms of neurologic deficit and pain and/or gross instability.

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