Isolated unilateral rupture of the alar ligament

Case report

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Only 6 cases of isolated unilateral rupture of the alar ligament have been previously reported. The authors report a new case and review the literature, morbid anatomy, and pathogenesis of this rare injury. The patient in their case, a 9-year-old girl, fell head first from a height of 5 feet off the ground. She presented with neck pain, a leftward head tilt, and severe restriction of right rotation, extension, and right lateral flexion of the neck. Plain radiographs and CT revealed no fracture but a shift of the dens toward the right lateral mass of C-1. Magnetic resonance imaging of the cervical spine showed signal hyperintensity within the left dens-atlas space on both T1- and T2-weighted sequences and interruption of the expected dark signal representing the left alar ligament, suggestive of its rupture. After 12 weeks of immobilization in a Guilford brace, MRI showed lessened dens deviation, and the patient attained full and painless neck motion.

Including the patient in this case, the 7 patients with this injury were between 5 and 21 years old, sustained the injury in traffic accidents or falls, presented with marked neck pain, and were treated with external immobilization. All patients had good clinical outcome. The mechanism of injury is hyperflexion with rotation. Isolated unilateral alar ligament rupture is a diagnosis made by excluding associated fracture, dislocation, or disruption of other major ligamentous structures in the craniovertebral junction. CT and MRI are essential in establishing the diagnosis. External immobilization is adequate treatment.

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KEY WORDS • alar ligament • ligamentous rupture • cervical spine injury • hyperflexion-rotation injury

Isolated alar ligament rupture is rare. Most cases of alar ligament rupture are seen in the context of atlantoaxial dislocation, condylar fractures, and atlantoaxial rotatory fixation.1,3,9,15,18 Only 6 cases of isolated alar ligament rupture have been previously reported in the literature (Table 1).4,5,7 We are reporting a case of unilateral rupture of the alar ligament resulting from a fall in a 9-year-old child.

Case Report

A 9-year-old girl fell head first from a tree branch 5 feet from the ground and sustained a direct impact injury to the occiput. She was taken to an outside emergency department, where she was found to have antegrade and retrograde amnesia and occipital scalp swelling but no neurological deficits. She also complained of headache and posterior neck pain and stiffness. Initial cervical spine radiographs and results of a CT scan were interpreted as normal (Figs. 1 and 2). She was discharged home in a hard cervical collar.

The child's neck pain and stiffness worsened during the next 2 days, and she was brought to our clinic with severe neck pain and a head tilt to the left. On examination, she had severe limitation of right rotation, extension, and right lateral flexion. Flexion, left rotation, and left lateral flexion were almost full and minimally uncomfortable (Fig. 3). The original radiographs and CT scan from 2 days previously were reviewed; they showed no fracture but revealed a lateral shift of the dens toward the right lateral mass of C-1 (Figs. 1 and 2). The mean condyle–C1 interval was normal on both sides (right, 1.80 mm; left, 1.84 mm), suggesting absence of atlantooccipital dislocation (Fig.
TABLE 1: Summary of reported cases of isolated rupture of the alar ligament

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Mechanism of Injury</th>
<th>Clinical Presentation</th>
<th>Associated Injury</th>
<th>X-Ray</th>
<th>CT</th>
<th>MRI</th>
<th>Side</th>
<th>Management</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Briem et al., 2002</td>
<td>15, M</td>
<td>gymnastics (pike), hyperflexion</td>
<td>neck pain, restricted neck motion</td>
<td>none</td>
<td>deviated dens to lt</td>
<td>deviated dens to lt</td>
<td>T1, T2 signal hyperintensity in widened LDAS</td>
<td>rt</td>
<td>hard collar, 4 wks†</td>
<td>healed w/o consequences</td>
</tr>
<tr>
<td></td>
<td>10, F</td>
<td>fell 2 m, hyperflexion†</td>
<td>neck pain</td>
<td>none</td>
<td>deviated dens to rt</td>
<td>deviated dens to rt</td>
<td>T2 signal hyperintensity in widened LDAS</td>
<td>lt</td>
<td>hard collar, 4 wks†</td>
<td>w/o apparent permanent damage</td>
</tr>
<tr>
<td>Demetrious, 2007</td>
<td>21, F</td>
<td>RTA (driver), lt front collision</td>
<td>neck pain, restricted neck motion</td>
<td>none</td>
<td>normal‡</td>
<td>normal‡</td>
<td>T2 hyperintense changes in widened LDAS</td>
<td>lt</td>
<td>conservative</td>
<td>asymptomatic at 6 mos</td>
</tr>
<tr>
<td>Caird et al., 2009</td>
<td>17, F</td>
<td>RTA (pedestrian)</td>
<td>neck pain</td>
<td>femoral fracture</td>
<td>NA</td>
<td>deviated dens to rt</td>
<td>T2 signal hyperintensity in widened LDAS</td>
<td>lt</td>
<td>halo, 12 wks</td>
<td>returned to full activities at 12 wks</td>
</tr>
<tr>
<td></td>
<td>15, F</td>
<td>RTA (passenger)</td>
<td>neck pain</td>
<td>femoral fracture</td>
<td>normal</td>
<td>deviated dens to rt</td>
<td>signal hyperintensity§ in widened LDAS</td>
<td>lt</td>
<td>halo, 12 wks</td>
<td>full, painless neck motion at 6 mos</td>
</tr>
<tr>
<td></td>
<td>5, F</td>
<td>RTA (passenger)</td>
<td>torticollis</td>
<td>C-4 nerve injury</td>
<td>normal</td>
<td>deviated dens to lt</td>
<td>T1 signal hyperintensity in widened LDAS; disruption of alar ligament¶</td>
<td>rt</td>
<td>hard collar, 4 mos</td>
<td>resolution of torticollis &amp; full, painless neck motion at 1 yr</td>
</tr>
<tr>
<td>present case</td>
<td>9, F</td>
<td>fell 5 ft, hyperflexion†</td>
<td>neck pain, restricted neck motion, head tilt</td>
<td>none</td>
<td>OMV: deviated dens to rt</td>
<td>deviated dens to rt</td>
<td>T1 &amp; T2 hyperintense changes in widened LDAS; disruption of lt alar ligament</td>
<td>lt</td>
<td>Guilford brace, 12 wks</td>
<td>full &amp; painless neck motion at 4 mos</td>
</tr>
</tbody>
</table>

* LDAS = lateral dens-atlas space; NA = not available; OMV = open mouth view; RTA = road traffic accident.
† Presumed.
‡ Reported as normal.
§ Did not specify T1 or T2.
¶ MRI 4 months postinjury.
Isolated unilateral alar ligament rupture

MRI of the cervical spine done on postinjury Day 2 showed the following findings. The dens was deviated to the right; there was signal hyperintensity on both T1- and T2-weighted sequences within the widened gap between the upper half of the dens and the left lateral mass of C-1 (lateral dens-atlas space) indicative of edema and/or hemorrhage from a ruptured left alar ligament. The expected dark signal of the left alar ligament appeared interrupted, which was also suggestive of rupture. The tectorial membrane and transverse atlantal ligament were intact, and the subjacent spinal cord was normal (Fig. 4).

A diagnosis of isolated left alar ligament rupture was made, and the patient was immobilized in a Guilford brace (G.A. Guilford and Sons Orthotic Laboratory) for 3 months.12 Her neck pain lessened gradually. At 2 weeks postinjury, the head tilt had resolved, the pain was very mild, and her range of neck movements in all motion planes was much improved. Follow-up MRI of the cervical spine done on postinjury Day 19 showed resolving T1 and T2 signal hyperintensity (Fig. 5). Flexion-extension radiographs of the cervical spine obtained at 4 weeks postinjury showed no cervical instability in the sagittal plane.

At 3 months, MRI of the cervical spine showed that the deviation of the dens was less marked, and the initial T2 signal hyperintensity in the left lateral dens-atlas space had faded (Fig. 6). The Guilford brace was removed 4 months postinjury, when the patient had painless and full range of neck motion.

Discussion

Functional Anatomy and Biomechanics

The alar ligament originates from the lateral or posterolateral aspect of the upper one-third of the dens. Its major component, the occipital portion, inserts on the medial aspect of the ipsilateral occipital condyle in close proximity to the occiput-C1 joint. Dvorak and Panjabi observed that in about two-thirds of their cadaver specimens there was an additional extension of the alar ligament to the lateral mass of C-1 (atlantal portion),8 although this finding was not seen by others.10 The main part of the alar ligament measures 11–15 mm in length, 3–8 mm in height, and 2–4 mm in thickness.8,10 In an MRI study of healthy individuals, coronal images showed the alar ligament running caudocranially from the dens to the occipital condyle in 67% of those studied, and horizontally in the rest (Fig. 7A). In the axial plane, the 2 alar ligaments formed an inverted V in 54%, a straight transverse line in 40%, and only rarely a true V (Fig. 7B–D).2

Functionally, the alar ligaments play an important role in strapping the occiput-C1-C2 complex together.9 As a pair, the alar ligaments limit axial rotation and lateral flexion of the occiput in relation to the axis. Rotation of the head to one side stretches the contralateral alar ligament first and later the ipsilateral ligament. At the
beginning of lateral flexion of the neck, the contralateral occipital portion of the alar ligament is relaxed, but the atlantal portion is stretched. Further lateral flexion causes both portions of the contralateral alar ligament also to tense up. Neck extension stretches both alar ligaments, especially those aligned in an inverted V or a straight line in the axial plane, which are the majority (Fig. 7B and C) (Table 2). The alar ligaments also tense up with flexion though they play a lesser role in controlling neck flexion, which is mainly checked by the tectorial membrane. In cadaver studies, the alar ligament is maximally stretched, and therefore most vulnerable to being torn, when the neck is subjected to excessive flexion-rotation to the contralateral side.

Histologically, the alar ligament is composed mainly of collagen fibers and scant elastin fibers; its lack of stretchability thus determines its main biomechanical behavior. For example, the alar ligament ruptures or

![Fig. 4. MRI of the cervical spine (postinjury Day 2). A–C: Axial T2-weighted images showing deviation of the dens to the right and T2 signal hyperintensity (asterisk) in the widened left lateral dens-atlas space. The dark signal of the left alar ligament (LAL) is disrupted, indicative of rupture. The right alar ligament (RAL) and the transverse atlantal ligament (TL) are intact. D–E: Axial T1-weighted images showing T1 signal hyperintensity (double asterisk) in the widened left lateral dens-atlas space. F: Sagittal T2-weighted image showing an intact tectorial membrane (TM) and normal thecal sac and spinal cord.](image1)

![Fig. 5. MRI of the cervical spine (postinjury Day 19). A–C: Axial T2-weighted images showing the same degree of deviation of the dens and T2 signal hyperintensity (asterisk) in the left lateral dens-atlas space as on postinjury Day 2. The dark signal of the left alar ligament is disrupted. D: Axial T1-weighted image showing loss of the bright T1 signal that was evident in the left lateral dens-atlas space (double asterisk), though the T2 hyperintensity is less than in the images obtained on postinjury Day 2 (Fig. 4).](image2)

![Fig. 6. MRI of the cervical spine (3 months postinjury). A: Coronal T2-weighted image showing lessened dens deviation to the right. B: Coronal T2-weighted image through part of the alar ligament showing possible healing, indicated by a restored dark band from the dens to the left occipital condyle. A lingering “gray” focus on the left alar ligament may indicate the original rupture site. C and D: Axial T2-weighted images showing further lessening of T2 signal hyperintensity (asterisk) in the left lateral dens-atlas space and less deviation of the dens.](image3)
Isolated unilateral alar ligament rupture

avulses from its attachment at different lengths depending on the speed of the stretch. With a slow stretch rate of 10 mm/sec to a load of 360 N, the alar ligament accommodates until it reaches an elongation of 11.3 to 14.1 mm. With a fast stretch rate of 920 mm/sec to the same load, as in the situation of many neck injuries, the alar ligament fails after an elongation of only 0.35 mm.16

Pathogenesis of Isolated Alar Ligament Rupture

Given the alar ligament’s anatomical and biomechanical characteristics, unilateral rupture of one alar ligament most probably occurs when the head is subjected to sudden contralateral rotation and hyperflexion, when the forces involved are violent and abrupt, yet not enough to disrupt the sturdier tectorial membrane and transverse atlantal ligament, whose destruction presumably calls for much larger forces. With pure unilateral alar ligament rupture, the atlantoccipital joint is not disrupted, the craniovertebral junction is not destabilized, and there is minimal to no brain injury.

Imaging Diagnosis

The first clue to the diagnosis is the lateral deviation of the dens in relation to the lateral masses of C-1, which can sometimes be seen on open-mouth radiographs (Fig. 1), but is best displayed on axial and coronal CT images. This deviation was found in our patient and in 5 of the 6 reported cases, suggesting that significant lateral dental deviation is a pathognomonic sign of unilateral alar ligament rupture.4,5,7 The mechanism is most likely due to overpull of the remaining intact alar ligament.

The definitive evidence of alar ligament injury is provided by MRI. The most reliable, though indirect, sign of rupture is signal hyperintensity on axial T2-weighted images within the lateral dens-atlas space, now widened due to the ruptured ligament. The increased T2 signal represents edema and/or hemorrhage, the latter especially if the T1 sequence also shows hyperintensity, indicating the presence of extracellular methemoglobin (Fig. 4). Five of the 6 published cases reported the T2 abnormality.4,5,7 More difficult is the demonstration of the actual rupture.4,5,7 The normal ligament appears hypointense in both T1- and T2-weighted MR images, and its rupture is usually inferred by a discontinuity of the expected dark signal.2 This task is complicated by the uncertain orientations of the 2 ligaments in relationship to the dens and to each other. Thus in the coronal MRI, the 2 ligaments would be “captured” in the same cut if they are lined up in a straight transverse plane, but not if they diverge dorsally or ventrally. In the latter cases, the ligament on one side may not be shown to advantage when the reformatting plane of the coronal image is aligned with the slanted ligament on the other side. The inability to visualize both dark structures may therefore represent a technical idiosyncrasy rather than true unilateral rupture. In our case, the discontinuous dark shadow is evident on the axial T2-weighted images (Fig. 4).

We recommend that when unilateral alar ligament rupture...
rupture is suspected on the basis of plain radiographs, a CT scan can be obtained to rule out fracture and atlantooccipital dislocation, followed by MRI to confirm the diagnosis and to rule out concomitant injury of other ligaments. Only then can the diagnosis of isolated alar ligament rupture be established.

Clinical Features, Management, and Outcome

To date, only 7 cases (including the present case) of isolated unilateral alar ligament rupture have been reported (Table 1). The patients were all between 5 and 21 years of age (median 15 years). Six were female. Four patients sustained the injury in a traffic accident, 2 from falls, and 1 during gymnastics. In 3 cases, the mechanism of injury was thought to be hyperflexion. All patients presented with marked neck pain. Our patient and a 5-year-old girl had documented head tilt and/or torticollis to the injured side. No patient had spinal cord injury or significant head injury.

When the diagnosis of isolated unilateral alar ligament rupture is established, external immobilization should be adequate treatment. Among the reported cases, 2 patients were immobilized in a halo orthosis for 12 weeks; and 3 patients wore a hard collar for 4 weeks (2 patients) to 4 months (1 patient). In one patient, the mode of external immobilization, if any, was not specified.

We prefer the Guilford brace because it provides more rigid immobilization than a cervical collar, and it incorporates a deep chin piece that limits rotation better than any other commercially available cervical-thoracic orthosis.

Outcome is generally good for patients with this injury. At follow-up points ranging from 4 weeks to 1 year, all 6 reported patients became painless and retained full range of neck motion without demonstrable cervical instability (Table 1). Our patient and all available pictures posted in the literature fulfill this criterion.

Tentative Recommendations

The diagnosis of unilateral alar ligament rupture is suspected when a recently injured child presents with a persistently painful neck with severely restricted motion, especially if accompanied by a head tilt or fixed torticollis. The open-mouth radiograph or CT dictated by routine trauma protocols would have shown a shifted odontoid process in relation to the C-1 lateral masses. The exact pathological “shift threshold” is unknown, but since most ligamentous injuries fall on a continuum of destruction ranging from sprain to partial tear to complete rupture, and the shift is likely proportional to the amount of disruption, we propose that a shift should be considered pathological if the wider gap exceeds 1.5 times the width of the narrower gap. Our case and all available pictures posted in the literature fulfill this criterion.

The next tentative recommendation concerns the use of external immobilization and its duration. External immobilization facilitates soft tissue healing. It is reasonable to expect fibrous union in cases of incomplete rupture, but it is unknown what actually happens in cases

### TABLE 2: Anatomy of the Alar Ligament

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Study Design</th>
<th>Dens (Origin)</th>
<th>Shape</th>
<th>Length (mm)</th>
<th>Orientation</th>
<th>Attachments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dvorak &amp; Panjabi, 1987</td>
<td>cadaveric cervical spine specimens (n = 29)</td>
<td>superior border: 11; caudal border: 13</td>
<td>in cross-section: elliptical</td>
<td>length: 3.2 x 6 mm</td>
<td>craniocaudal 47%, horizontal 32%, caudocranial 21%</td>
<td>1) anterior part of occipital condyle, 2) presence of an atlantal portion</td>
</tr>
<tr>
<td>Osmotherly et al., 2013</td>
<td>cadaveric cervical spine specimens (n = 11)</td>
<td>length: 11-15</td>
<td>in cross-section: ovoid</td>
<td>length: 5-8 mm</td>
<td>craniocaudal 36%, horizontal 64%</td>
<td>1) attachment is on lat/posterolateral aspect of upper 1/3 of dens; 2) highest point of attachment is 1.72 mm below tip of dens; 3) total height of ligament is 5–8 mm</td>
</tr>
<tr>
<td>Baumert et al., 2009</td>
<td>MRI of healthy subjects (n = 52)</td>
<td>length: 11.5</td>
<td>in cross-section: trapezoid, triangular</td>
<td>length: 5-8 mm</td>
<td>transverse 54%, inverted V 46%</td>
<td>1) attachment is on lateral border of dens; 2) highest point of attachment is 1.72 mm below tip of dens; 3) total height of ligament is 5–8 mm</td>
</tr>
</tbody>
</table>

* Orientation may be very different in life compared to orientation in cadaveric specimens.
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of complete rupture when the 2 free ligament stubs are retracted far apart, or how long it takes to achieve sturdy union. We based our 3-months endorsement not on any statistical science, but on our experience with immobilizing children with spinal cord injury without radiographic abnormality (SCIWORA), in that we have encountered recurrent SCIWORA after 2 months of bracing but never after 3 months.¹⁴ We assumed 2 months might not be sufficient for solid healing and thought an additional month of bracing a sensible compromise.

Finally, even if full fibrous union does occur with immobilization, it is uncertain what measure of long-term stability it provides. Judging from the complete lack of pain and resumption of fluid neck motions after the 3-month bracing period in our patient, it is not unreasonable to presume safe conduct of “normal” daily activities after bracing in a patient with a unilateral (not bilateral) rupture. The real caveat is in predicting the future risk for sundry sports. Again, not drawing on verifiable data, we recommend that children wait at least 9 months after bracing in a patient with a unilateral (not bilateral) rupture. The real caveat is in predicting the future risk for sundry sports. Again, not drawing on verifiable data, we recommend that children wait at least 9 months after treatment before returning to contact sports. One should perhaps remember that if alar ligament rupture is at all like an ankle collateral ligament tear, a state of permanent instability may indeed develop from repetitive injuries.

Disclosure

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 to the study and manuscript preparation include the following. Conception and design: Pang, Wong. Acquisition of data: Wong, Fan. Analysis and interpretation of data: Wong. Drafting the article: Wong, Ernest. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Pang. Study supervision: Pang.

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