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MRI assessment of the alar ligaments in the late stage of whiplash injury – a study of structural abnormalities and observer agreement

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Abstract Our aim was to characterise and classify structural changes in the alar ligaments in the late stage of whiplash injuries by use of a new MRI protocol, and to evaluate the reliability and the validity of this classification. We studied 92 whiplash-injured and 30 uninjured individuals who underwent proton density-weighted MRI of the craniovertebral junction in three orthogonal planes. Changes in the alar ligaments (grades 0–3) based on the ratio between the high signal area and the total cross-sectional area were rated twice at a 4-month interval, independently by three radiologists. Inter- and intraobserver statistics were calculated by ordinary and weighted kappa. Cases classified differently were reviewed to identify potential causes for disagreement. The alar ligaments were satisfactorily demonstrated in all cases (244 ligaments in 122 individuals). The lesions, 2–9 years after the injury, varied from small high-signal spots to high signal throughout the cross-sectional area. Signal was highest near the condylar insertion in 82 of 94 ligaments, indicating a lesion near that insertion, and near the dental insertion in eight, indicating a medial lesion. No grade 2 or 3 lesion was found in the control group. At least two observers assigned the same grade to 214 ligaments (87.7%) on the second occasion. In 30 ligaments (12.3%) this agreement was not obtained. Pair-wise interobserver agreement (weighted kappa) was fair to moderate (0.31–0.54) in the first grading, improving to moderate (0.49–0.57) in the second. Intraobserver agreement (weighted kappa) was moderate to good (0.43–0.70). Whiplash trauma can cause permanent damage to the alar ligaments, which can be shown by high-resolution proton density-weighted MRI. Reliability of classification of alar ligament lesions needs to be improved.

Keywords Cervical spine · Craniovertebral junction · Whiplash injury · Alar ligament · Magnetic resonance imaging

Introduction

Whiplash is defined as an injury caused by hyperextension and subsequent hyperflexion of the cervical spine associated with sudden acceleration and/or deceleration of the head, most often caused by a road traffic accident [1]. The craniovertebral ligaments are vulnerable to this type of high-speed trauma. In a postmortem study of 30 cases of craniocerebral trauma, Saternus et al. [2] found rupture of 11 alar ligament. Rupture was seen also after less severe injuries. In a dissection study, 20 of 21 individuals who had died in traffic accidents without craniovertebral dislocation showed alar ligament injuries [3]. In a CT study of 43 traffic accident victims with clinically suspected rotational instability of the neck, 26 showed increased rotation between the occiput and the

atlas and between the atlas and axis [4], strongly suggesting alar ligament insufficiency. However, other workers have not reproduced these findings [5]. The reason could be that functional CT is difficult to standardise because rotation of the head beyond the normal range causes pain and dizziness.

Several investigations have been performed to establish the role of imaging in whiplash trauma. Plain films are typically normal, and MRI has revealed only degenerative changes [6] with the same prevalence as in asymptomatic individuals [7, 8, 9]. One study of the alar ligaments in healthy individuals showed extensive anatomical variation and concluded that changes in these ligaments would have little clinical relevance [10]. Although a few MRI studies have been performed to assess the alar ligaments after whiplash trauma [11, 12], none has reliably described ligamentous lesions.

We introduced a new MRI protocol for showing the ligaments and membranes in the craniovertebral junction [13], which we have used to classify alar ligament changes in healthy and whiplash-injured individuals to examine the validity of these changes. We investigated the reliability of this classification by comparing the rating from three observers. Finally we re-examined cases in which the classification was divergent, to identify reasons for disagreement.

Materials and methods

All individuals diagnosed as having had a recent whiplash injury by a local physician in seven rural communities in Western Norway were registered prospectively from 1992 to 1998, 342 in all. The inclusion criteria corresponded to the type 2 of the Québec task force, which proposed the following classification of whiplash-associated disorders [1]: grade 0: no complaint about the neck and no physical signs; grade 1: neck pain, stiffness, or tenderness only; no physical sign(s); grade 2: neck complaint(s) and musculoskeletal sign(s) including decreased range of motion and point tenderness; grade 3: neck complaints and neurological sign(s) including decreased or absent deep tendon reflexes, weakness and sensory deficits; and grade 4: neck complaints and fracture or dislocation. Symptoms such as dizziness, tinnitus, headache or memory loss can be manifest in all grades.

The patients were graded both in the acute phase and 12–16 weeks later. Those with in grades 1 and 3) at this point were excluded. Plain films of the neck were normal in all the patients in the acute stage.

From the 342 patients, a random sample of 100 was invited to participate in this study: 93 accepted and gave informed consent, while seven declined or did not answer; one had to be excluded later due to claustrophobia. Hence we studied 92 injured subjects, 33 males and 59 females, mean age 40 years, range 14–61 years. The mean time between injury and MRI examination was 6 years, range 2–9 years.

We also included 30 individuals, 11 men and 19 women, mean age 46 years, range 28–66 years, with no history of head or neck trauma as a control group. They came from the same seven Norwegian communities and were matched for age and sex.

Images were obtained at 1.5 tesla, using fast spin-echo (SE) proton-density-weighted sequences. With the aid of a sagittal locator image, we obtained 12 2 mm thick, interleaved contiguous axial sections covering the area from the foramen magnum to the base of the dens. A coronal sequence covered from the anterior arch of the atlas to halfway through the spinal canal. We used TR 2200 and TE 15 ms, matrix 224×512, and field of view (FOV) 127×203 mm for both. We acquired four signals, using an echo train length of seven and receiver bandwidth 130 Hz/pixel. Phase-encoding was from left to right, and we applied saturation pulses superior and inferior to the axial sections and anterior and posterior to the coronal sections. Imaging time was 4 min 47 s for each sequence.

A coronal section through the alar ligaments was used as a locator image to create the sagittal sections. We obtained 15 2 mm interleaved, contiguous sections from the right to the left occipital condyle. The imaging parameters were as for the axial and coronal sections except for the following: TR 2660 TE 15 ms, matrix 322×512, FOV 184×210 mm. We applied superior, inferior and anterior saturation pulses. Imaging time was 6 min 13 s. Pixel size was 0.57×0.40 mm in all three planes, which means that lesions down to 0.5 mm could be discriminated. Examination time for all three sequences, including patient positioning, was approximately 30 min.

Normal ligaments show low signal intensity [14, 15, 16], whereas increased signal intensity is regarded as a sign of injury [17, 18, 19]. To differentiate between mild, moderate and severe lesions we estimated the ratio between any high-signal part and the total cross-sectional area of the alar ligament. The image with maximal cross-sectional abnormal signal was selected for this grading. In equivocal cases, the observers were instructed to take coronal and axial images into account as well. A summary of the criteria is shown in Table 1. Two observers (JK, GM) had 10 years of MRI experience, the third (HN) 3 years. To ensure that the criteria were mutually understood, we performed a pilot study of ten cases, compared the results, and discussed differences in application of the criteria prior to commencing the main study.

The images of the injured and uninjured individuals were mixed in random order, unknown to the interpreters. The alar ligaments were graded according to the criteria in Table 1. The images were graded again by the same three radiologists 4 months later. Ligaments rated two or more steps differently on the first and second occasions were reassessed to identify reasons for such a divergence, as were ligaments which all three observers graded differently on the second occasion. Evaluation of image quality, identification of displacement and analysis of structural patterns were done by one observer (JK).

Kappa coefficients were used to evaluate intraobserver agreement (comparing the first and second gradings) and pair-wise interobserver agreement. Ordinary kappa coefficients were calculated on basis of all four possible MRI gradings (0–3), and pairs of

Table 1. Criteria for classification of alar ligament lesions based on maximal cross-section involvement in sagittal images

Grade	Criterion[a]
0	Low signal throughout entire cross-section
1	High signal in 1/3 or less of cross-section
2	High signal in 1/3–2/3 of cross-section
3	High signal in 2/3 or more of cross-section

[a]observers were instructed to let axial and coronal images influence grading: ligaments with a markedly posterior orientation, evident on axial images, are cut obliquely on sagittal sections, and the possibility of volume averaging should be kept in mind; high signal on sagittal images is usually accompanied by a corresponding increase on coronal images, and when sagittal image grading is equivocal, signal change on coronal images should be used to up- or downgrade

grades combined (0–1 versus 2–3). The ordinary kappa puts equal weight on one and more than one-step disagreement. To include the amount of disagreement, weighted kappa was calculated as well. Differences in rating were weighted as follows: full agreement: 1; a one-step difference: 0.67; a two-step difference: 0.33; and a three-step difference: 0.00. We also calculated 95% confidence intervals for the kappa coefficients [20]. Kappa < 0.20 is generally regarded as indicating poor, 0.21–0.40 fair, 0.41–0.60 moderate, and > 0.60 good agreement [21]. Consistent differences between the first and second gradings, and pair-wise differences between two observers, where one consistently reported higher or lower numbers of abnormalities, were examined by use of McNemaŕs test for symmetry [22, 23].

Results

Image quality was excellent in 111 cases and slightly less so in 11 because of motion artefact. However, the alar ligaments were well defined in both coronal and sagittal images in all 122 cases. Structural changes, if present, were usually seen both in sagittal and coronal images. Axial images showed the orientation of the ligament, but gave little additional information on the degree of structural change.

Pair-wise interobserver calculations for all four alar ligament grades separately are given in Table 2, and show a kappa indicating fair to moderate agreement (0.31–0.42); the weighted kappa indicated moderate agreement (0.49–0.57). Dichotomising the groups (grades 0–1 and 2–3), we found kappas indicating moderate to good agreement (0.46–0.65). One observer (JK) reported more grade 2–3 lesions than the others ($P < 0.05$). Reasons for interobserver disagreement are given in Table 3. In the majority of these cases signal intensity was intermediate, which made the criteria difficult to apply.

Intraobserver agreement is shown in Table 4. The kappa for all four alar ligament grades separately was fair to good (0.26–0.63), whereas the weighted kappa was moderate to good (0.43–0.70). With dichotomised

Table 3. Main reasons for pair-wise interobserver 2 or 3-step disagreement in the second grading of 244 ligaments and for similar intraobserver disagreement between first and second gradings (244 ligaments graded twice) for all three observers

Reasons for disagreement	Number of ligaments	
	Interobserver	Intraobserver
Reduced image quality	2	6
Volume averaging due to very oblique ligament	1	2
Homogeneous intermediate signal[a]	10	10
Heterogeneous intermediate signal[a]	10	16
Speckled appearance[a]	4	5
Erroneous use of criteria	3	20
Total	30	59

[a]in all or part of the cross-section

Table 2. Pair-wise interobserver agreement, kappa coefficient for agreement and *P* value given by McNemaŕs test for symmetry, from the second grading 122 right and 122 left alar ligaments (grades 0–3) and dichotomised (grades 0–1 and 2–3) by three observers (JK, GM, HN)

Observers	% in grade(s)				Disagreement (%)	Kappa coefficient (95% confidence interval)		*P*
	0	1	2	3		Ordinary	Weighted	
Right alar ligament								
JK vs GM	45.1	5.7	3.3	9.0	36.9	0.41 (0.28–0.53)	0.51 (0.38–0.64)	0.07
HN vs JK	37.7	8.2	8.2	4.1	41.8	0.39 (0.29–0.50)	0.54 (0.43–0.64)	1.00
GM vs HN	37.7	13.1	3.3	4.1	41.8	0.36 (0.24–0.48)	0.49 (0.38–0.61)	0.16
Left alar ligament								
JK vs GM	42.6	2.5	3.3	7.4	44.6	0.31 (0.19–0.42)	0.50 (0.38–0.61)	0.34
HN vs JK	39.3	6.6	9.8	5.7	38.6	0.42 (0.30–0.53)	0.57 (0.46–0.67)	0.67
GM vs HN	38.5	8.2	4.1	5.7	43.5	0.34 (0.22–0.46)	0.50 (0.39–0.62)	0.58
	0–1		2–3					
Right alar ligament								
JK vs GM	63.1		15.6		21.3	0.46 (0.30–0.63)		< 0.01[1]
HN vs JK	63.1		22.1		14.8	0.65 (0.50–0.79)		0.03[1]
GM vs HN	70.5		14.8		14.7	0.58 (0.40–0.75)		0.10
Left alar ligament								
JK vs GM	59.8		18.9		21.3	0.49 (0.33–0.66)		0.08
HN vs JK	61.5		20.5		18.2	0.57 (0.41–0.73)		0.05
GM vs HN	64.8		15.6		19.6	0.48 (0.30–0.66)		1.00

[1]JK rated significantly more grade 2-3 lesions than GM and HN

Table 4. Intraobserver agreement, kappa coefficient for agreement and McNemar's test for symmetry, based on 122 right and 122 left alar ligaments graded twice (grades 0–3) and dichotomised (grades 0–1 and 2–3) by three observers (JK, GM, HN)

Observer	% in grade(s)				Disagreement (%)	Kappa coefficient (95% confidence intervals)		*P*
	0	1	2	3		Ordinary	Weighted	
Right alar ligament								
JK	34.4	3.3	11.5	13.9	36.9	0.48 (0.37–0.59)	0.67 (0.58–0.76)	< 0.01[1]
GM	51.6	17.2	0.8	9.0	21.4	0.63 (0.53–0.75)	0.70 (0.58–0.81)	0.33
HN	24.6	12.3	10.7	4.1	48.3	0.33 (0.20–0.45)	0.48 (0.38–0.59)	< 0.01[1]
Left alar ligament								
JK	35.2	7.4	11.5	13.9	31.7	0.55 (0.43–0.66)	0.69 (0.60–0.78)	< 0.01[1]
GM	48.4	11.5	4.9	7.4	27.8	0.54 (0.41–0.66)	0.61 (0.49–0.73)	0.06
HN	22.1	13.9	6.6	4.1	53.3	0.26 (0.14–0.38)	0.43 (0.32–0.54)	< 0.01[1]
	0–1		2–3					
Right alar ligament								
JK	53.3		31.1		15.6	0.68 (0.54–0.81)		< 0.01[1]
GM	78.7		12.3		9.0	0.69 (0.50–0.86)		0.23
HN	57.4		23.0		19.6	0.56 (0.41–0.71)		< 0.01[1]
Left alar ligament								
JK	53.3		31.1		15.6	0.68 (0.54–0.81)		< 0.01[1]
GM	70.5		16.4		13.1	0.63 (0.46–0.80)		0.21
HN	56.6		18.9		24.5	0.44 (0.27–0.60)		0.02[1]

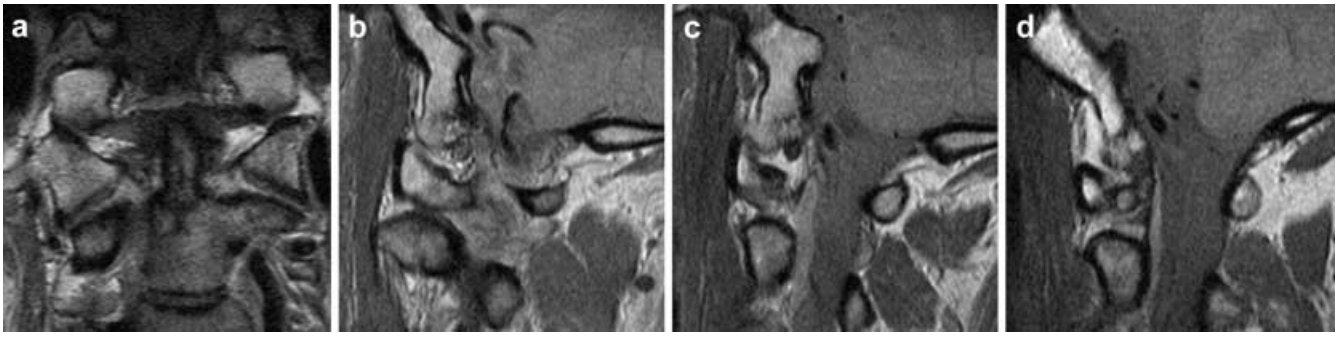
[1]JK and HN rated significantly lower grades in their second evaluation

groups (grades 0–1 and 2–3), the agreement was good for two observers and moderate for the third (0.44–0.69). Two observers (JK, HN) assigned significantly lower grades on the second occasion than on the first ($P < 0.05$). There were 59 ligaments in 42 cases (8% of all ligaments) rated two or three steps differently on the second occasion from the first. Reasons for this intraobserver disagreement were the same as for interobserver disagreement (Table 3); intermediate signal intensity was the major difficulty. For 20 ligaments no reason other than flawed interpretation could be identified.

Typical examples are shown in Figs. 1, 2, 3 and 4. The signal changes interpreted as lesions were typically seen on 2–4 sagittal images, and varied from sharply circumscribed bright lesions in an otherwise dark structure to less demarcates areas of increased signal. Increased signal was usually most prominent near the condyle or the dens, and faded gradually towards the other end of the ligament.

In second occasion two or all three observers concurred about grading in 214 ligaments (87.7% of the total). Based on such agreement there were 120 grade 0 ligaments in 76 cases, 52 in the uninjured group. Of these, 110 ligaments in 69 cases showed low signal throughout their entire cross-section, appearing dark

Fig. 1a–d. Typical appearances. **a** Coronal proton density-weighted image (2200/15 ms) with lines marking the plane for **b** right **c** left alar ligaments. Note diffuse high signal in the lateral part of the right alar ligament *(arrow)*, and normal low signal on the left. **b, c** Sagittal proton density-weighted images (2660/15 ms) of the alar ligaments. **b** Homogeneous high signal covering the entire cross section *(arrowhead)*, classified as a grade 3 lesion. **c** Normal ligament showing low signal throughout its cross section *(arrowhead)*. **d** Ligament with slightly higher signal, appearing dark grey, also classified as normal *(arrowhead)*



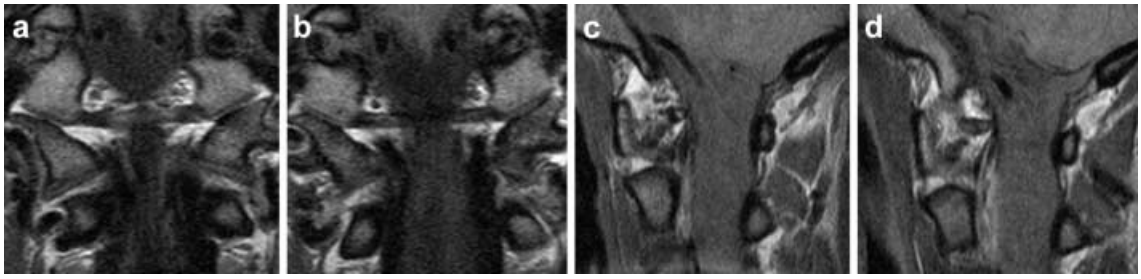


Fig. 2a–d. Grade 1 lesions. **a, b** Consecutive interleaved coronal proton density-weighted images with lines marking the plane for **c** right **d** left alar ligaments. Note high signal in the medial part of both ligaments (arrows), fading away laterally (small arrowheads), indicating lesions near the dens. **c, d** Sagittal proton density-weighted images of the alar ligaments. **c** High signal in the posterior part of the right alar ligament *(arrowhead)* covering about one-third of its cross-sectional area, classified as a grade 1 lesion. **d** The left alar ligament is somewhat thicker with two separate, slightly high-signal lesions *(arrowheads)*, also classified as grade 1

(Fig. 1c). Another 10 ligaments, in seven cases showed slightly increased signal, appearing dark grey (Fig. 1d). They were slightly thicker, probably due to a looser fibre structure.

We assigned grade 1 to 42 ligaments in 33 cases, four from the uninjured group. The higher signal was central in 25 ligaments (Fig. 2d), ventral in 10 and dorsal in seven (Fig. 2c). It was most marked laterally in 35 and medially in the remaining seven (Fig. 2a). In coronal images, a longitudinal high-signal streak or slightly increased signal within the substance of the ligament could usually be seen (Fig. 2b).

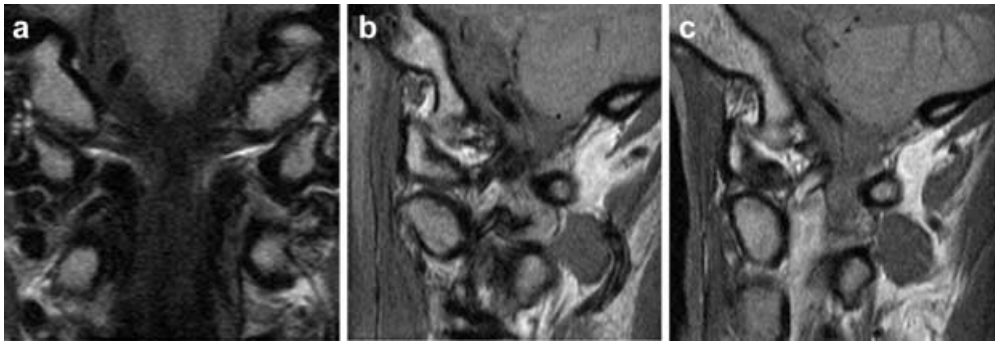
There were 29 ligaments classified as grade 2, in 27 cases, none in the uninjured group; 18 were central (Fig. 3b), four ventral and three dorsal (Fig. 3c). In four of these ligaments the cross-section appeared speckled. The signal increase was maximal laterally in 27 (Fig. 3a) and medially in two. Coronal images showed either stripes of increased signal or generally increased signal, most prominent near one of the insertions.

Fig. 3a–c. Grade 2 lesions. **a–c** As Fig. 1. Note high signal in the lateral half of both alar ligaments *(arrows)*, indicating lesions near the condyles. **b, c** Sagittal proton density-weighted images of the alar ligaments. **b** The posterior part of the right ligament shows areas of high signal *(arrowheads)* covering between one- and two-thirds of the cross-section, and therefore classified as a grade 2 lesion. **c** About half of the cross-section of the left ligament shows increased signal intensity, also classified as grade 2 *(arrowhead)*

We placed 23 ligaments in 18 cases in grade 3, none in the uninjured group. The high signal involved the entire cross-section in 11 ligaments (Fig. 4c), and more than two-thirds in the remaining 12 (Fig. 4b). All 23 ligaments showed a low-signal rim, which demarcated it from the surrounding soft tissue (Fig. 1b, 4b, 4c). The maximum signal increase was near the condylar insertion in 20 ligaments (Fig. 4a) and near the dental insertion in three. Coronal images showed prominent bands of increased signal (Fig. 4a) or generally increased signal (Fig. 1a). Discontinuity, as a sign of total disruption, was not seen in any case.

There were 30 ligaments (12,3% of the total), including four in the uninjured group, which all three observers graded differently. The major causes were ligaments with intermediate heterogeneous signal (Fig. 5a), multiple bright spots, giving a speckled appearance (Fig. 5b), and intermediate signal throughout the cross-sectional area (Fig. 5c).

The atlas was displaced to the left in three cases (Fig. 6) and to the right in another three (all in the injured group). Mean displacement was 2.0 mm (range 1.7–2.6 mm); apparent displacement < 1.5 mm was not recorded. In these six cases, the alar ligaments on the two sides were graded as normal in three cases, grades 1 and 2 in one case, 0 and 3 in another and as 1 and no agreement in the last.



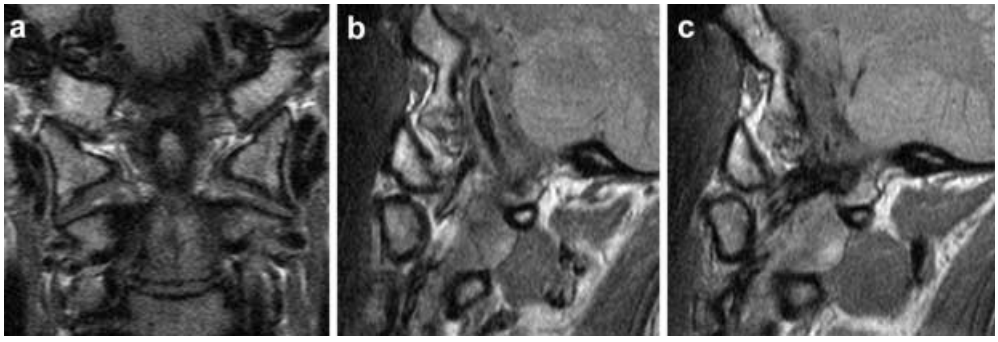


Fig. 4a–c. Grade 3 lesions. **a–c** As Fig. 1. High signal in the lateral part of both alar ligaments, fading towards the dens *(arrows)*, indicates lesions near the condylar insertion. Both show high signal covering the entire cross-section, except for a small low-signal area anteriorly in the right ligament *(small arrow)*. The outer lining of elastic fibres preserves the continuity of the ligaments *(arrowheads)*

Discussion

We found no grade 2 or 3 lesions in the uninjured group, which we take to indicate that these are caused by a whiplash injury, the only known neck trauma in the study group. Until now, there is no evidence that any diagnostic test or imaging method contributes to the diagnosis of whiplash injury in the acute or the chronic stages. Several MRI studies of the cervical spine have been performed to search for possible soft-tissue lesions [6, 7, 8, 9]. The protocols used include sagittal T1- and T2-weighted sequences, short tau inversion-recovery (STIR), and axial T1 or T2, usually with slices 4–5 mm thick. The attention has been directed towards neck posture, facet joints, discs, longitudinal ligaments, spinal cord, muscles and other soft tissues. Only two studies have focused on the alar ligaments. One showed no abnormality in the seven patients examined [11]. The other described complete or partial rupture of the alar ligaments in some cases [12], but with little imaging material presented and unclear interpretation criteria. We established an imaging protocol designed specifically for the craniovertebral junction [13]. For detailed demonstration of the alar ligaments good spatial resolution and optimised contrast are critical. A slice thickness of 2 mm gives excellent spatial resolution. T2-weighted images gave inadequate discrimination between ligament, bone and soft tissue due to a low signal-to-noise ratio. T1-weighted images afforded poorer contrast resolution and thus less ability to differentiate small variations in signal. We therefore found a proton-density weighted sequence the technique of choice for assessment of ligamentous abnormalities.

Increased signal within a ligament is used as a sign of partial or total disruption [17, 18, 19]. Our investigation was based on the assumption that the degree of injury

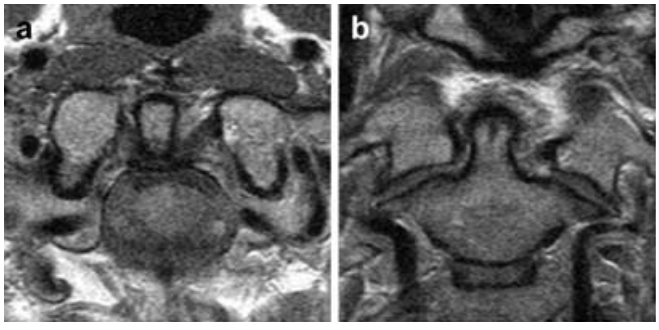
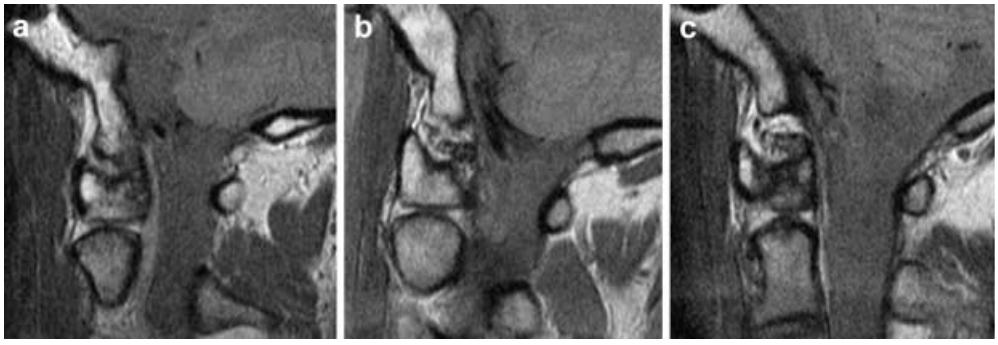


Fig. 6a, b. Lateral displacement of the atlas. **a** Axial **b** coronal proton density-weighted images showing a lateral displacement of the atlas to the left. The dens is 2.6 mm off the midline. There is no atlantoaxial rotation to explain this displacement

Fig. 5a–c. Cases rated differently by the three observers. Sagittal proton density-weighted images showing three different alar ligaments in cross-section. **a** Heterogeneous intermediate signal intensity, highest posteriorly *(arrowheads)*, whereas the anterior portion gives somewhat lower signal *(arrow)*. **b** Central high-signal areas, giving a speckled appearance *(small arrowheads)*. **c** Intermediate signal throughout most of the cross-section *(arrows)*



correlates with the extent of increased signal in a cross-section. We therefore used primarily sagittal images, but with a contribution from coronal sections, particularly in equivocal cases and for assessment of the ligament-bone transition zone. Axial sections gave inadequate images of the alar ligaments in most cases and added little information.

Our study group was randomly selected from a well-defined cohort of whiplash-associated disorder, clinical grade 2 at 12–16 weeks after injury; we added 30 randomly selected healthy individuals as a control group. MRI was performed more than 2 years after the injury to ensure that healing was complete.

Pair-wise interobserver agreement was moderate for two observers and fair for the less experienced one. Intraobserver agreement was moderate and good for the two most experienced observers and fair for the less experienced one. Major disagreement (two or more steps) was found in 12% of ligaments imaged when the second gradings were compared pair-wise, and in 8% comparing each observer's first and second grading. Inconsistency in radiological diagnosis will mislead clinicians and reduce the usefulness of examinations. It is therefore important to identify findings susceptible to different interpretations and to disclose sources of disagreement. In most alar ligaments, a lesion was shown as high signal covering part of its cross-section. However, in some ligaments the entire cross-section showed homogeneous or heterogeneous intermediate signal. Disagreement occurred mainly with ligaments showing this intermediate signal (''grey'' ligaments) over all or most of their cross-section. This appearance can be due to low fibre density, a normal variant, or to a sprain affecting the whole cross-section. Some ligaments showed spots of bright signal, giving a speckled appearance. In these cases, an estimation of the total high signal area was made. A better understanding of the normal anatomy and injury patterns, combined with more experience in using the criteria, will probably reduce such inconsistencies.

In grading ligamentous lesions, there will, however, always be equivocal cases. Hence, a one-step difference in grading does not necessarily indicate real disagreement. The weighted kappa coefficient was introduced, and as expected considerably better values were found when degree of disagreement was taken into consideration.

Because of a great variation in the tensile strength of normal alar ligaments [24] grade 1 lesions may have little clinical significance. To evaluate the effectiveness of our method in distinguishing normal and slightly abnormal (grades 0–1) from obviously injured ligaments (grades 2–3), we dichotomised the data. Moderate to good kappa values were then obtained for both intra- and interobserver agreement. Our view is, however, that with more experience and better MRI equipment it should be possible to classify alar ligament lesions consistently into three grades of severity.

Two of three observers diagnosed significantly fewer abnormalities on their second grading. Differing thresholds in the assessment of continuous variables may depend on ''response bias'', i.e., the tendency to prefer one category to another [25]. A better insight into the anatomy and injury patterns may explain a higher threshold for a verdict of pathology in the second grading. Consistent differences between observers did not occur.

Lesions were observed in the lateral or medial parts of the ligament, with none in only its midportion. Ligaments show a gradual transition from connective tissue to bone. Histologically, four zones are described: ligament – fibrocartilage – mineralised fibrocartilage – bone [26, 27]. Of the 94 lesions, 82 were in the lateral half of the alar ligament, with maximum signal increase near the condylar insertion, indicating strongly that this transition zone represents the weakest point and the predominant site for injury of an alar ligament. This corresponds with the findings of Saldinger et al [28] who, testing alar ligaments in vitro, found rupture to occur at the interface between mineralised and nonmineralised fibrocartilage.

The increased signal within the ligament is probably due to atrophy of fibres and fat replacement. Ligaments have uniform microvascularity, the feeding vessels penetrating at the site of insertion [29]. Since alar ligament lesions occur in the ligament-bone transition zone, impaired nutrition may be a cause of atrophy. We did not find ligamentous discontinuity as a sign of total disruption. However, in about half of the 23 grade 3 lesions an dark outer lining was the only remnant of the ligament. Alar ligaments consist of a central core of collagen with a few elastic fibres peripherally. Elastic fibres tolerate elongation up to 200%, collagen only 8% [28]. The elastic fibres preserve continuity in severely injured ligaments.

Substantial (more than 1.5 mm) sideways shift of the atlas was seen in six cases. We found no correlation between this displacement and alar ligament lesions. It is well known that atlantoaxial rotation can imitate lateral displacement on open-mouth radiographs [30]. In our cases there was no such rotation. A recent study showed lateral displacement in 10 of 50 normal individuals [10]. No clinical correlate of lateral shift of the atlas is known.

The long-term morbidity and therapeutic and medicolegal implications for whiplash patients underline the importance of establishing an accurate diagnosis based on objective criteria. Increased craniovertebral rotation following a whiplash injury, previously shown, strongly signifies alar ligament insufficiency. Our study confirms that these ligaments are vulnerable to this type of trauma, and that the severity of the lesions can be graded using high-resolution MRI. The reliability of grading, however, needs improvement. We call attention to the craniovertebral junction when searching for a structural

substrate for the late whiplash syndrome. The clinical implications of the radiological abnormalities should be investigated further.

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