**Case Series**

**Mineralisation of the longitudinal odontoid ligament in three horses identified on computed tomographic examination**

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**Summary**

Three horses that were presented for head shaking and/or suspected neck pain and in which marked mineralisation of the longitudinal odontoid ligament of the dens was identified on CT examination are described. There are currently no reports of mineralisation of the longitudinal odontoid ligament in horses in the literature, other than as part of a congenital malformation. Although the significance of this finding to the presenting clinical problems in the cases described remains speculative, an association between mineralisation of the longitudinal odontoid ligament and compatible clinical signs merits further investigation.

**Key word:** horse;mineralisation; longitudinal odontoid ligament; computed tomography; headshaking

**Introduction**

The anatomy and appearance of the ligamentous structures of the occipitoatlantoaxial (OAA) region in normal horses, as imaged using a 3 tesla MRI scanner, has recently been described (Gutierrez-Crespo *et al*. 2014). There are multiple reports of clinically significant mineralisation of the ligamentous structures of the OAA joints in humans (Kobayashi *et al.* 2001; Sim and Park 2006) and a single report of a similar condition in a dog (Hamilton *et al*. 2016). Although the anatomy and number of ligaments involved in the OAA articulation in humans and dogs differs to that of the horse, the function of these ligaments in all species is essentially the same; to support the OAA region whilst allowing principally dorsoventral flexion of the cervical spine at the atlanto-occipital (AO) joint and significant axial rotation at the atlantoaxial (AA) articulation (Levine *et al.* 2007).

The longitudinal odontoid ligament of the horse has its caudal attachment on the dorsal surface of the odontoid process of C2. It consists of two symmetric bands of ligamentous fibres, with a divergent V shape that attach cranially to the transverse inner surface of the atlas, cranial to the fossa of the dens (Gutierrez-Crespo *et al.* 2014). These ligaments may be considered functionally analogous to the alar ligaments of the dog and of man, although in both of the later species the ligaments continue rostrally to insert on to the caudal part of the occiput. The longitudinal odontoid ligament of the horse may be considered the functional continuation of the dorsal longitudinal ligaments of the rest of the cervical spine (Fig 1) (Gutierrez-Crespo *et al.* 2014).

Dystrophic mineralisation of ligaments and tendons has been reported in many different anatomical locations in the horse (Garvican *et al.* 2016; O’Brien and Smith 2018; Kadic *et al.* 2019) Calcification/mineralisation may occur either due to metastatic deposition following abnormalities of calcium metabolism, or as a response to injury, degeneration, or inflammation of a ligament (Kumar *et al.* 2017). Dystrophic calcification is common in the tendons and ligaments of horses (Smith *et al.* 2008; O’Brien and Smith 2018) compared to metastatic calcification. In many cases ectopic mineralisation of ligamentous structures in the horse is considered an incidental finding, whereas in others clinical signs and diagnostic investigations suggest significance of these abnormalities (O’Brien and Smith 2018; Kadic *et al.* 2019).

The purpose of this case report is to describe the identification and appearance of mineralisation within the longitudinal odontoid ligament in three horses that were presented for further investigation of headshaking and/or suspected neck pain.

**Case histories and clinical signs**

***Case 1:*** A 13-year-old, Welsh Section A gelding was presented for CT evaluation of the head and cranial cervical spine following a 3-week history of progressive behavioural abnormalities, including headshaking, suspected to be consistent with poll pain. Headshaking, in a vertical plane, had been noticed during showing and in-hand exercise.

***Case 2:*** A 10-year-old, Dutch Warmblood gelding was presented for CT investigation of suspected oral or head and neck pain. The horse had reportedly become reluctant to work in an outline and accept bit contact 7 weeks prior to presentation. The signs had reportedly developed acutely following dental treatment during which the horse was sedated with the head resting on a head stand. Physiotherapist assessment had reportedly identified pain in the region of the atlas.

***Case 3:*** A 18-year-old, Thoroughbred cross mare was presented for investigation of suspected neck pain and perceived neck guarding associated with an unexplained fall 1 week prior to presentation. The mare had a history of transient neck pain 3 years previously, which had resolved following a short course of phenylbutazone. Following the most recent presentation radiographs of her cervical vertebrae were obtained and mild degenerative changes were identified which included osteoarthritis and enlargement of the caudal cervical facet joints.

**CT examinations**

In case 1, with the patient under general anaesthesia, due to a failed initial attempt at adequate standing sedation. General anaesthesia was induced withketamine (2.7mg/kg iv) and diazepam (0.07mg/kg iv). Following induction, general anaesthesia was maintained with sevofluranewith an end-tidal anaesthetic agent concentration of between 3.1 – 3.2%. In cases 2 and 3, each computed tomographic examination was performed with the patient restrained under standing sedation using a combination of acepromazine (0.03mg/kg iv), morphine (0.1mg/kg iv) and romifidine(0.06-0.08mg/kg iv). Images were obtainedusing a 16 slice, 90cm bore CT scanner1, mounted on a sliding gantry system. The head and neck of the patient were supported on a custom made radiolucent carbon fibre table2 (Fig. 2). Scanning parameters varied slightly between size of patient but most images were acquired using 5mm slice collimation, 70cm field of view, 120 KVp and 300 mAs. All CT images were reconstructed using a sharp bone algorithm (5mm slice thickness and 1.mm or 0.5mm slice thickness) and using a smooth soft-tissue algorithm (1.0 or 0.5mm slices). Images were viewed on a computer monitor, using proprietary DICOM software(HOROSTM, GNU Lesser General Public License, Version 3.0, LGPL 3.0). The longitudinal odontoid ligament was subjectively analysed according to size, shape, margination and attenuation and objectively analysed, using region of interest analysis, to calculate the maximum (Max) and minimum (Min) Hounsfield unit (HU) and standard deviation (SD) values. Overall conclusion of interpretation was arrived at by consensus amongst all authors.

**Imaging Findings**

***Case 1:*** Multiple focal regions of mineralisation were identified in both branches of the longitudinal odontoid ligament (Fig.3). There was subjective enlargement and poor definition of the margins of both lobes of the ligament. Objective values, max 500 HU Min -5 HU 144.0 SD. There were also subtle subchondral bone thickening and irregularity of the cranial part of C1 at the AO articulation. Other abnormalities were infundibular cemental hypoplasia of the majority of the maxillary cheek teeth (07’s, 08’s, 09’s, 10’s Triadan system), subtle subchondral bone irregularity of the mandibular condyle of the left temporomandibular joint and focal, discrete mineralisation in the intra-articular disc of the same joint. There was also focal, 2mm diameter region of mineralisation in the right lateral ventricle of the brain but no evidence of increased ventricular size or abnormality of overall shape of the ventricles.

***Case 2:***Multiple focal areas of moderate mineralisation were evident in both lobes of the longitudinal odontoid ligament (Fig.4). There was subjective enlargement and poor definition of the dorsal margin of the left lobe of the ligament compared to the right and slight effacement of the dorsal border of the ligament with the ventral margin of the spinal cord. There was mild irregularity of the cranial, dorsal part of the dorsal process of C2 at the site of attachment of the dorsoatlantoaxial ligament. Objective values, Max 231 HU, Min -57HU, SD 64.35. In addition, moderate, diffuse, mineralisation of the cartilages of the larynx and a small focal single region of mineralisation in the right and left soft tissue ventral to the longus capitis muscle of the neck were identified. This horse also had a focal, irregularly shaped mass of mixed bone and soft tissue attenuations, within the left caudal maxillary sinus, and partly attached to the lateral wall.

***Case 3:*** Multiple focal regions of increased and decreased attenuation were identified in both lobes of the longitudinal odontoid ligament (Fig.5). Several large focal areas of increased attenuation were surrounded by regions of irregularly shaped hypoattentuation. Objective values, Max HU 544 Min -4, SD 136.0. Both lobes of the ligament were subjectively enlarged and poorly marginated. Infundibular cemental hypoplasia of the maxillary 09’s, 10’s and 11’s (Triadan system) was noted. Reflective of the mares age, very little reserve crown of any of the teeth remained. There was mild diffuse mineralisation of the laryngeal cartilages.

**Discussion**

The above cases, presented for headshaking and/or neck pain, were identified to have mineralisation of the longitudinal odontoid ligament. To the authors’ knowledge, mineralisation of the longitudinal odontoid ligament has not been previously reported in the OAA joints of horses with normal vertebral morphology. A single case report exists of mineralisation, identified in the ligaments of the OAA joint of a warmblood mare by CT examination. The OAA joint in this horse was markedly malformed and the mineralisation was described as being within the apical ligament of the dens (Bru̎nisholz *et al.* 2019). After studying the anatomical description and CT images provided in that case report, the authors of the current report believe that the ligament reported is not the apical ligament but is actually the longitudinal odontoid ligament. Detailed anatomical descriptions of the ligaments of the OAA region of the horse, compared with other mammals, are notably lacking in the current literature, hence the confusion in nomenclature.

There are difficulties in imaging the area of the longitudinal odontoid ligament. It cannot be fully assessed radiographically due to superimposition by the arches of C1 and the dens of C2 on laterolateral, oblique lateral and ventrodorsal radiographs of this region. Complete rupture of the odontoid ligaments, may be identified by recognition of a reduced distance between the dorsal lamina of C1 and the dens of C2 but even quite large depositions of dense mineral deposits would not be expected to be visible in normally aligned OAA articulations. Similarly, although we have not investigated the appearance of mineralisation of the OAA ligaments on nuclear scintigraphic imaging, it is likely that the depth of ligament and the overlying bone would make any increase in radiopharmaceutical impossible to detect. The normal anatomy of the OAA of the horse, as seen on 3T MRI examination has been described (Gutierrez-Crespo *et al.* 2014). The longitudinal odontoid ligament was reported as presenting as isointense to the musculature of the neck on T1W images, moderately hypointense to neck muscles on PD-SPIR sequences, markedly hypointense to muscles of the neck and isointense to cortical bone in T2W sequences. This would mean that regions of partial mineralisation may be very difficult to fully appreciate, particularly in T2W images, as they would be of similar signal intensity to some of the ligament fibres and bone attachments.

The CT appearance of the longitudinal odontoid ligament in normal horses has not been described. We found that the longitudinal odontoid ligaments could be consistently visualised on sagittal, dorsal and transverse multiplanar reconstruction (MPR) images, similar to reports of MRI examination. Identification of the margins of this ligament was easier when the horse was imaged standing, rather than recumbent under general anaesthesia. The degree of rotation of the atlas relative to the axis meant that if a horse was imaged in any degree of rotation to the midline plane, MPR images had to be aligned to either the dens of C2 or to the body of C1 in turn, extending image evaluation time. This did not alter the assessment of maximum or minimum HU of the longitudinal odontoid ligaments but rotation did make subjective assessment of symmetry and size of the two lobes of the ligament more difficult. A slightly modified bone window (300 WL, 900 WW) appeared to allow the best assessment of presence or absence of mineralisation, whilst still being able to delineate the margins of the ligament.

Mineralisation within soft-tissue structures can result from calcification and/ or ossification and appear on CT evaluation as focal or diffuse areas of increased attenuation. Extrapolating from the literature regarding the ligaments of the OAA region in other species and mineralisation of ligaments other than those of the OAA region in the horse, we speculate that the possible aetiopathogenesis of mineralisation of the longitudinal odontoid ligament of the horse may include accumulative repetitive strain, acute trauma, age-related change, inflammation, genetic predisposition or, less likely, metastatic mineralisation.

Repetitive stress or traumatic injury of the ligaments of the OAA junction may be related to the biomechanics and normal ranges of motion (ROM) of the AO and C1-C2 articulations. Zsoldos and colleaguesdemonstrated that the principal site of axial rotation of the equine cervical spine at walk is located at the atlantoaxial joint (Zsoldos *et al.* 2010). At walk, horses also demonstrated significant dorsoventral flexion/extension at C1-C2. Earlier studies on cadaver cervical spines, assessing total ROM of each cervical articulation, with the musculature removed and only the joint capsules and ligaments remaining intact, identified the AO articulation to have the greatest capacity for dorsoventral flexion/ extension. The degree of dorsoventral flexion/ extension of the AO and of C1-C2 articulation was significantly reduced in adult horses when compared to cadaver spines of foals (Clayton *et al.* 1989). These findings may be important when considering ligament pathology at the C1-C2 articulation. The reported ROM displayed at the C1-C2 junction in combination with the reduction of natural ROM in adult horses compared with foals, may mean that headshaking type behaviour actually induces pathological changes within the supporting ligaments of this region rather than the pathological changes resulting in the headshaking. The reduced ROM in adult horses in the dorsoventral flexion/ extension direction when compared to foals may indicate an age related predisposition to injury of these ligaments in horses that display excessive dorsoventral flexion/ extension, such as in headshaking or repeated athletic work that involves increased flexion of this region (e.g. dressage).

In case 2, the imaging findings were suggestive of chronic (mineralisation) and more recent (lobe enlargement) injury. The history of recent dental treatment raised the possible theory that prolonged OAA extension when the head was placed on a head stand may have resulted in acute exacerbation of longer standing disease.

Focal calcification of the ligaments of the atlas have been reported in the human literature and predominantly results from traumatic injuries, inflammatory diseases or attributed to ageing (Kobayashi *et al.* 2001; Sim *et al.* 2006; Mohamed and Aziz 2009). Mineralisation following surgical trauma has also been reported within tendons and ligaments of the appendicular skeleton, where pain and weakness may be a feature (O’Brien *et al.* 2012).

Abnormalities of calcium metabolism resulting in hypercalcaemia may also be causes for mineralisation of the longitudinal odontoid ligament. “Crowned dens” syndrome has been described in humans, resulting in neck pain and attributed to calcification of the neighbouring odontoid process. This has previously been reported in combination with calcium deposition diseases (Sato *et al.* 2005; Wu *et al.* 2005). In calcium deposition diseases (calcium hydroxyapatite and calcium pyrophosphate dehydrate) the CT examination identifies the calcification to be surrounding the odontoid process in a ‘halo-like’ distribution affecting predominantly the transverse ligament of the atlas (Sato *et al.* 2005; Wu *et al.* 2005), whereas, the three cases presented in this report all had focal mineralisation within, rather than surrounding, the longitudinal odontoid ligament.

Inflammatory causes of abnormalities of the OAA ligaments have been reported in rare cases within the human literature. Grisel’s syndrome, a condition mainly identified in children, results in non-traumatic, atlantoaxial rotatory subluxation as a consequence of upper airway infection and inflammation, leading to alterations of the ligaments of the atlas. The most accepted pathogenesis of this syndrome states that, because there is communication between the pharyngeal veins and the ventral vertebral veins, pharyngeal inflammation results in increased vascular flow to the ligaments of the OAA region. This results in ligament oedema and inflammation with subsequent development of laxity (Barecelos et al. 2014). This is an interesting concept and perhaps worthy of further consideration in the investigation of equine patients with cranial cervical pain, especially considering the wide spectrum of infectious and inflammatory conditions of the pharyngeal region that may be encountered in the horse.

Mineralisation and/ or granulomatous reaction of the intervertebral joint capsule and of the surrounding ligamentous connective tissue has been described in a horse caused by onchocercosis (Hestvik *et al.* 2006). It seems unlikely that a parasitic infection would localise specifically to a small, deeply located, discrete structure, such as the longitudinal odontoid ligament, but this possibility cannot be discounted without further histopathological investigations (Hestvik *et al.* 2006).

There are limitations to our case report, prohibiting identification of clinical significance of the mineralisation of the longitudinal odontoid ligament in horses. All three cases are from a referral population and they are heterogenous in signalment, presentation and other diagnostic investigation. Only a limited number of other diagnostic investigations were performed to rule out other causes of the clinical signs and there are numerous other differential diagnoses that may account for the headshaking behaviour and neck pain displayed by these horses. However, diagnostic analgesia of this region is not possible.

In addition, it is uncertain whether the finding of mineralisation of the longitudinal odontoid ligament is a primary cause of discomfort or if it occurs as a result of other causes of pain and subsequent changes in neck or head movement or is an incidental finding. Idiopathic or trigeminal mediated headshaking is a diagnosis of exclusion, usually being diagnosed in the absence of other aetiologies e.g. dental pathologies, progressive ethmoidal haematoma, aural mites, allergic rhinitis, sinusitis and photic headshaking (Cook 1979; Lane and Mair 1987; McGorum and Dixon 1990; Madigan *et al.* 1995; Newton *et al.* 2000). More recently Thomson *et al.* (2019) discussed the challenges in differentiating between musculoskeletal pain and trigeminal-mediated headshaking. None of the horses in our report were identified as performing other behavioural signs of trigeminal-mediated headshaking, such as snorting and rubbing the nose (Lane and Mair 1987; Pickles *et al.* 2014a and b).

Although some reported dental abnormalities can lead to clinical signs of headshaking (Lane and Mair 1987; Dixon et al. 2000), this was not believed to be the case in the three reported cases. The imaging identified infundibular cemental hypoplasia in case’s 1 and 3. The pathological consequence of this condition, infundibular caries, is a very common finding with a reported prevalence of 45% (Borkent et al 2017). To date, the condition has not been reported to cause any of the signs seen in the cases described here. Temporomandibular joint cysts and subchondral bone irregularity of the mandibular condyle (case 1) were deemed incidental and of no clinical significance (Carmalt et al. 2016). This was also the case for the diffuse mineralisation of the cartilages of the larynx (case 3) and the small focal bodies of mineralisation in the ventricles of the brain (case 1); consistent with mineralisation of cholesterol granulomas (Capucchio et al. 2010).

All of the horses in this series remain alive and therefore histopathological examination of the abnormalities identified on CT evaluation was not possible.

**Conclusions**

The clinical significance of mineralisation of the longitudinal odontoid ligament of the horse remains speculative. Our observations indicate that the longitudinal odontoid ligament may be imaged using CT examination, in both the standing sedated and anaesthetised horse and that focal regions of hypo or hyperattenuation may be identified along with poor definition and subjective enlargement of either, or both lobe of the ligament. Further studies investigating the normal and abnormal CT appearance, function, and pathology of this ligament in the horse are required.

**Manufacturers’ addresses**

**1** Canon aquillion \* \* Canon Medical Systems Ltd. Crawley, UK.

2Bibby precision engineering, Wirral, UK

**Conflict of interest statement**

No competing interests have been declared.

**Ethical animal research**

Not applicable.

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**Authorship**

All authors contributed to the study design, data analysis/ interpretation and preparation of the manuscript. All authors gave their ﬁnal approval of the manuscript.

**Reference List**

Barecelos A. C. S., Patriota G. C. and Netto A. U. (2014) Nontraumatic atlantoaxial rotatory subluxation: Grisel syndrome. Case report and literature review. *Global Spine J*. **4.** 179-186.

Borkent, D., Reardon, R.J.M., McLachlan, G., Smith, S. and Dixon, P.M., 2017. An epidemiological survey on the prevalence of equine peripheral dental caries in the United Kingdom and possible risk factors for its development. *Equine Vet. J.*, **49**(4), 480-485.

Bru̎nisholz H. P., Wildhaber N., Hoey S., Ruetten M., Boos A. and Ku̎mmerle J.M. (2019) Congenital occipitoatlantoaxial malformation in a warmblood mare. *Equine Vet Educ.* 31. **5**. 242-247.

Carmalt, J.L., Kneissl, S., Rawlinson, J.E., Zwick, T., Zekas, L., Ohlerth, S. and Bienert‐Zeit, A. (2016) Computed tomographic appearance of the temporomandibular joint in 1018 asymptomatic horses: a multi‐institution study. *Vet. Radiol. Ultrasound*, **57**(3), pp.237-245.

Capucchio, M.T., Márquez, M., Pregel, P., Foradada, L., Bravo, M., Mattutino, G., Torre, C., Schiffer, D., Catalano, D., Valenza, F. and Guarda, F. (2010) Parenchymal and vascular lesions in ageing equine brains: histological and immunohistochemical studies. *J. Comp. Pathol.*, **142**(1), 61-73.

Clayton H. M. and Townsend H. G. G. (1989) Cervical spinal kinematics: a comparison between foals and adult horses*. Equine Vet. J*. **21,** (3), 193-195.

Cook, W.R. (1979) Headshaking in horses Part 1. *Equine Pract.*1, 9-17.

Dixon, P.M., Tremaine, W.H., Pickles, K., Kuhns, L., Hawe, C., Mccann, J., McGorum, B.C., Railton, D.I. and Brammer, S. (2000) Equine dental disease Part 3: a long‐term study of 400 cases: disorders of wear, traumatic damage and idiopathic fractures, tumours and miscellaneous disorders of the cheek teeth. *Equine Vet. J.*, **32**(1), 9-18.

Garvican E. R., Wylie C. E., Payne R. J., Smith R. K. W. and Head M. J. (2016) Mineralization of the equine palmar/plantar annular ligament treated by surgical resection. *Vet. Surg*. **45**, 602-608.

Gutiérrez-Crespo, B., Kircher, P. R. and Carrera, I. (2014) 3 tesla magnetic resonance imaging of the occipitoatlantoaxial region in the normal horse. *Vet Radiol. Ultrasound.* **55**, (3), 278-285.

Hamilton L. C., Driver C., Tauro A., Campbell G. and Fitzpatrick N. (2016) Mineralization of the transverse ligament of the atlas causing compressive radiculopathy: resolution following odontoidectomy and atlantoaxial arthrodesis. *Vet. Comp Orthop. Traumatol*. **29**, (3), 253-258.

Hestvik G., Ekman S. and Lindberg R. (2006) Onchocercosis of an intervertebral joint capsule causing cervical vertebral stenotic myelopathy in a horse. *J. Vet. Diagn. Invest.* **18**. 307-320

Kadic D.T.N., Minshall G.J. and Wright I.M. (2019) Surgical management of marginal tears/avulsions of the suspensory ligament branches in 29 Thoroughbred racehorses. *Equine Vet. J.* **51**, 310-315.

Kobayashi Y., Mochida J., Saito I. Matui S. and Toh E. (2001) Calcification of the alar ligament of the cervical spine: imaging findings and clinical course. *Skeletal Radiol.* **30**, 295-297.

Kumar V., Abbas A., Aster J. (2017) Robbins Basic Pathology. Elsevier, 10th Ed. 25-26

Levine J.M., Levine G.J., Hoffman A.G., Mez J. and Bratton G.R (2007) Comparative anatomy of the horse, ox and dog. Equine Comp. Cont. Educ. Pract.Vet. 2. 279-292.

Lane, J.G. and Mair, T.S. (1987) Observations on headshaking in the horse. *Equine Vet.* *J.***19**, 331-336.

Madigan, J.E., Kortz, G., Murphy, C. and Rodger, L. (1995) Photic headshaking in the horse: 7 cases. *Equine vet. J.* **27**, 306-311.

McGorum, B.C. and Dixon, P.M. (1990) Vasomotor rhinitis with headshaking in apony. *Equine vet. J.* **22**, 220-222.

Mohamed S. C. and Aziz A. A. (2009) Calcification of the Alar ligaments mimics fracture of the craniovertebral junction (CVJ). An incidental finding from computerised tomography of the cervical spine following trauma. *Malaysian Journal of Medical Sciences.* **16,** (4), 69-72.

Newton, S.A., Knottenbelt, D.C. and Eldridge, P.R. (2000) Headshaking in horses: possible aetiopathogenesis suggested by the results of diagnostic tests and several treatment regimes used in 20 cases. *Equine Vet. J.*, **32**(3), 208-216.

O’Brien E.J.O., Frank C.B., Shrive N.G., Hallgŕimsson B. and Hart D.A. (2012) Heterotopic mineralization (ossification or calcification) in tendinopathy or following surgical tendon trauma. *Int. J. Exp. Pathol.* **93**, 319-331.

O’Brien E.J.O. and Smith R. K. W. (2018) Mineralization can be an incidental ultrasonographic finding in equine tendons and ligaments. *Vet. Radiol. Ultrasound*. **59**. 613-623.

Pickles, K.J., Aleman, M., Marlin, D.J. and Adams, V.J. (2014a) Owner-reported response to treatment of 130 headshaking horses.Am.Assoc. *Equine Pract.* **60**, 176-183.

Pickles, K. J,, Madigan, J. and Aleman, M. (2014b) Idiopathic headshaking: is it still idiopathic? *Vet. J.* **201**, 21-30.

Sim K. B. and Park J. K. (2006) A nodular calcification of the alar ligament simulating a fracture in the craniovertebral junction. *Am. J. Neuroradiol.* **27,** 1962-3.

Sato, T., Hagiwara, K., Sasaki, M., Matsuno, H. and Akiyama, O. (2005) Crowned dens syndrome. *Internal Med.* **44(2)**:160.

Takizawa N., Nomura A. and Fujita Y. (2016) Rare cause of nuchal pain: calcification of the alar ligament. *J Rheumatol*. **43,** 177-178.

Thomson K., Chan C. and Dyson S. (2019) Head tossing behaviour in six horses: Trigeminal-mediated head-shaking or musculoskeletal pain? *Equine Vet. Educ.* [Epub ahead of print] <https://doi.org/10.1111/eve.13084>.

Wu, D.W., Reginato, A.J., Torriani, M., Robinson, D.R. and Reginato, A.M. (2005) The Crowned Dens Syndrome as a Cause of Neck Pain: Report of Two New Cases and Review of the literature. *Arthritis Rheum.* **53(1)**:133–137.

Zsoldos R. R., Groesel M., Kotschwar A. B. and Peham C. (2010) A Preliminary modelling study on the equine cervical spine with inverse kinematics at walk. *Equine Vet. J*. **42**, (suppl. 38) 516-522.

**Figure and Table Legends**

**Figure 1a:** Schematic diagram of the anatomical location of the longitudinal odontoid ligament in the horse (adapted from the anatomical description in Gutiérrez-Crespo et al. 2014). Orientated from dorsal images at the level of the atlantoaxial region. (\*) delineating the lobes of the longitudinal odontoid ligament (Gutiérrez-Crespo *et al.* 2014).

**Figure 1b:** Left: C1 (altas) caudal view. Right: C2 (axis) cranial view. To outline the landmarks of the longitudinal odontoid ligament cranial and caudal attachments. It has caudal attachments at the dorsal surface of the odontoid process of the axis (C2) (>). The ligament radiates (‘V’ shaped) and its cranial attachments are to the transverse rough surface of the floor of the atlas (C1) (+), cranial to the fossa of the dens (C2).

**Figure 2:** Multidetector, 16 slice, 90cm bore, CT scanner (Canon, Aquilion), mounted on a sliding gantry system. The head and neck of the patient supported on a custom made radiolucent carbon fibre table. The horse in this photograph is undergoing a general anaesthetic for the CT to be performed.

**Figure 3: Case 1 -** Multiple focal regions of mineralisation in both lobes of the longitudinal odontoid ligament (white arrows). **A)** Dorsal MPR at the level of the AA joint. Top is rostral. Left is left and right is right. **B)** Dorsal MPR at the level of the AA joint. **C)** Right parasagittal MPR at the level of the OAA. Left is rostral. Right is caudal. **D)** Transverse MPR at the level of mid-caudal level of C1.

**Figure 4:** **Case 2 -** Multiple focal areas of moderate mineralisation in both lobes of the longitudinal odontoid ligament at the level of the dens of C2 (white arrows). **A)** Dorsal MPR at the level of the AA joint. Top is rostral. Left is left and right is right. **B)** Right parasagittal MPR at the level of the OAA. Left is rostral. Right is caudal. **C)** Transverse MPR at the mid-caudal level of C1. Dorsal is dorsal. Ventral is ventral.

**Figure 5: Case 3 -** Multiple focal regions of increased and decreased attenuation in both lobes of the longitudinal odontoid ligament (white arrows). **A)** Dorsal MPR. Top is rostral. Left is left and right is right. **B)** Right parasagittal MPR at the level of the OAA. Left is rostral. Right is caudal. **C)** Transverse MPR at the mid-caudal level of C1. Dorsal is dorsal. Ventral is ventral. **D)** Transverse MPR of C1 at the level of C1-C2 articulation. Dorsal is dorsal. Ventral is ventral.