Epidemiological and morphological characteristics of incomplete ossification of the dorsal neural arch of the atlas in dogs with atlantoaxial instability

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Received October 4, 2017.
Accepted December 27, 2017.

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OBJECTIVE
To retrospectively evaluate the epidemiological and morphological features and outcome of surgical treatment of incomplete ossification of the dorsal neural arch of the atlas (IODA) in dogs with atlantoaxial instability (AAI).

ANIMALS
106 AAI-affected dogs that underwent ventral fixation of the atlantoaxial joint.

PROCEDURES
Medical records and CT images for each dog were reviewed. Dogs were allocated to 1 of 2 groups on the basis of the presence or absence of IODA or of dens abnormalities (DAs) in CT images.

RESULTS
Of the 106 dogs with AAI, 75 had and 31 did not have IODA; 70 had and 36 did not have DAs. Incomplete ossification was present in the cranialmost, central, or caudalmost portion of the dorsal neural arch of the atlas in 59, 39, and 28 dogs, respectively; 2 or 3 portions were affected in 29 and 11 dogs, respectively. The mean CT value (in Hounsfield units) for the midline of the dorsal neural arch of the atlas in dogs with IODA was significantly lower than that for the same site in the dogs without IODA. The mean age at surgery for dogs with central IODA was significantly higher than that of the non-IODA group. The severity of spinal cord injury before or after atlantoaxial ventral fixation did not differ between the IODA and non-IODA groups.

CONCLUSIONS AND CLINICAL RELEVANCE
Results indicated that concomitant DAs or IODA is common in dogs with AAI. In dogs with incomplete ossification in the central part of the dorsal neural arch of the atlas, surgical treatment of AAI generally occurs at a middle to advanced age. (*Am J Vet Res* 2018;79:1079–1086)

Small-sized young dogs are commonly affected with AAI, which causes cervical spinal cord compression.[1–5] Atlantoaxial instability in TBDs was first described by Geary et al.[6] It has been reported that at least half the canine cases of AAI involve dogs ≤ 1 year of age,[4] and the onset of canine AAI at middle to advanced ages is sporadic. Atlantoaxial instability results from congenital atlantoaxial joint dysplasia in young TBDs, and the onset of AAI is influenced by various factors.[3–13]

ABBREVIATIONS

3D-MPR	Three-dimensional multiplanar reconstruction
AAI	Atlantoaxial instability
CI	Confidence interval
DA	Dens abnormality
DALR	Dens-to-axis length ratio
HU	Hounsfield units
ID	Intact dens
IODA	Incomplete ossification of the dorsal neural arch of the atlas
ROI	Region of interest
TBD	Toy-breed dog

In 1 study,[2] Beaver et al identified DAs in 76% of 46 dogs with AAI. Often, only the presence or absence of DAs is recorded at the time of AAI diagnosis. Dysplasia and nonunion of the ossification center in the dens of the axis—as observed on radiographic views and CT images—are generally considered examples of DAs. Dens abnormalities such as obvious nonunion of the dens and separation of bone are easily recognized, but more generally, there are no well-defined standards for diagnosis of DAs in veterinary medicine. We have proposed use of the DALR as an objective indicator of DAs.[14] If the DALR is low, DAs should be suspected. If DAs are suspected, even within the clinically normal range, minor trauma may have caused subluxation of the atlantoaxial joint. Therefore, if the DALR is low, it is thought that it may be impossible to obtain a full braking effect from the transverse ligament of the atlas.

In addition to DAs, a few reports[7,15,16] address incomplete ossification of the atlas in dogs as another cause of AAI. Incomplete ossification of the dorsal

neural arch of the atlas involves an ossification abnormality of the dorsal raphe of the vertebral arch or the raphe of the vertebral body and arch during development of the 3 ossification centers of the atlas.[7,15,16] In Beagles, the raphe of the dorsal midline fuses by 106 days after birth and the ventral raphe fuses by 115 days after birth.[17,18] Parry et al[7] conducted a retrospective study of the CT images of 120 dogs to examine the morphological features of the atlas. According to their report, incomplete ossification of the atlas was observed in 12 (10%) dogs aged 4 months to 13 years (median age, 4 years); incomplete ossification of the atlas seemed to be detected more frequently in gun dogs. Five dogs had AAI resulting from incomplete ossification of the atlas, and there was a strong correlation between AAI and incomplete ossification of the atlas (odds ratio, 35.0; 95% CI, 7.0 to 175; $P < 0.001$).

If IODA is present, the atlantoaxial joint may loosen because of the absence of or weakening at the site of attachment of the dorsal atlantoaxial ligament. However, if the dens is normal, the transverse ligament of the atlas maintains the dens in its normal position.[15,16] In humans, IODA is classified into 5 types, ranging from simple rupture to complete absence of the dorsal arch; the incidence is 3% to 5%.[19,20] Nearly all human cases are asymptomatic; exceptions include cases involving fractures or similar trauma-induced injuries.[20–22] In humans, fibrous connective tissue that forms at the site of the bone defect associated with IODA usually enables good stability to be maintained.[19,23,24] Toy-breed dogs, which are susceptible to AAI, have been determined to develop DAs and defects of the transverse ligament of the atlas relatively frequently,[4,8] but there are no reports of incomplete ossification in the raphe of the atlas in these breeds,[7] to our knowledge. However, in our experience, detailed assessment of CT images of clinical cases of AAI, predominantly among TBDs, reveals a surprising number of cases involving bone defects or bone thinning in the dorsal arch of the atlas. The objectives of the study reported here were to retrospectively characterize the epidemiological and morphological features of IODA in dogs with AAI for which surgery was performed and to assess the outcome of surgical treatment for IODA.

Materials and Methods

Dogs

The medical records of 167 AAI-affected dogs that underwent surgery between February 2005 and April 2016 at the Veterinary Medical Teaching Hospital, Nippon Veterinary and Life Science University, or at the YPC Tokyo Animal Orthopedic Surgery Hospital were retrieved for review. For each dog, the diagnosis of AAI had been made on the basis of MRI and CT findings. One hundred six dogs of 11 breeds were followed up after surgery and included in this study. Preoperative CT images were surveyed for IODA, and the dogs were allocated to 1 of 2 groups (ie, dogs with or without IODA). Dogs in the IODA group were further categorized on the basis of the location of the incomplete ossification (ie, in the cranialmost, central, and caudalmost portions of the dorsal arch of the atlas [IODA-cranial, IODA-central, and IODA-caudal groups, respectively]). For each dog, information collected from the medical record included breed, sex, age in months at the time of surgery, body weight at the time of surgery, and severity of pre- and postoperative neurologic abnormalities. The CT images were also evaluated for DAs, the presence of which was the primary cause of AAI. On the basis of this assessment, the dogs were also allocated to a DA or ID group for additional analyses.

CT and morphological evaluations

Image acquisition was performed with 80- and 160-slice CT scanners[a]; settings used were scan speed of 0.5 seconds and slice thickness and interval of 0.5 mm each. For CT evaluation, each dog was anesthetized and positioned in dorsal recumbency; its neck was extended and stabilized with a wedge-shaped stabilizer. The head, cervical portion of the vertebral column, and thoracic portion of the vertebral column were prevented from rotating by use of adhesive tape. Morphological evaluation of the atlas of each dog was performed with image-processing software[b] to reconstruct the CT digital image and medical data into 3D-MPR images. In accordance with the procedures reported by Parry et al[7] and Rivero et al,[25] CT images were obtained with a bone window (window width, 2,500 HU; window level, 500 HU) or soft-tissue window (window width, 658 HU; window level, –14 HU) settings.

Morphological evaluation of the dorsal neural arch of the atlas

For morphological assessment of the dorsal arch of the atlas in all dogs, the cranialmost portion, the central portion, and the caudalmost portion were identified in sagittal images reconstructed by 3D-MPR with the soft-tissue setting for CT images **(Figure 1)**. The same images were reassessed with the bone window setting, and the CT value of the midline region in transverse images of each site was determined **(Figure 2)**. Given that the dorsal arch of the atlas is a structure centered on cortical bone, IODA associated with incomplete ossification of the bone was defined in the present study as a CT value lower (CT value, < 600 HU) than that of D3a (CT value, 600 to 850 HU) in Sogo's classification[26] of bone quality on the basis of CT values.

DAs and DALR

Because of the lack of well-defined standards for the diagnosis of DAs in veterinary medicine (with the exception of cases of obvious nonunion of the dens and separation of the bone), we proposed the use of the DALR as an objective index of DAs.[14] In our previous study,[14] medical records of 153 dogs with AAI that underwent surgery at our institutions between February 2005 and November 2014 were reviewed. Among these dogs, the top 4 breeds were Chihuahua

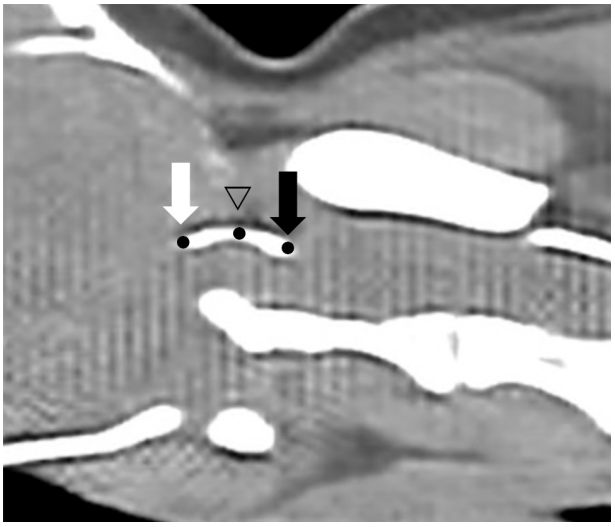

Figure 1—Representative sagittal image (derived via 3D-MPR of CT images obtained with a soft tissue window setting) of the atlantoaxial joint of a 120-month-old sexually intact male Yorkshire Terrier with AAI. The soft tissue window setting had a window width of 658 HU and a window level of −14 HU. For morphological assessment of the dorsal neural arch of the atlas, the cranialmost portion (white arrow), the central portion (arrowhead), and the caudalmost portion (black arrow) were identified in the sagittal image. The same image was subsequently reassessed with a bone window setting.

(n = 28), Toy Poodle (20), Yorkshire Terrier (20), and Miniature Dachshund (12). The study population also included 40 dogs of nonaffected AAI-predisposed breeds (10 Chihuahuas, 10 Toy Poodles, 10 Yorkshire Terriers, and 10 Miniature Dachshunds) and 40 healthy Beagles (a breed that is not predisposed to AAI). Some of the AAI-affected dogs in the previous study[14] were included in the present study. The DALR was calculated as the ratio of the length of the dens to that of the axis body **(Figure 3)**. A low DALR may be predictive of a high probability of DAs. Dens abnormality in the present study was defined as aplasia of the dens or separated bone fragments or a DALR less than the 95% CI of the DALR for the respective breed (Chihuahua and Toy Poodle, < 0.36; Yorkshire Terrier, < 0.34; Miniature Dachshund, < 0.40; Beagle, < 0.39).[14] In our previous study,[14] there was no significant difference in the DALR between the small dog breeds that are prone to AAI and Beagles, a breed of larger dogs. Thus, the 95% CI of the DALR for Beagles was adopted for breeds other than Chihuahua, Toy Poodle, Yorkshire Terrier, and Miniature Dachshund. In dogs with separation or dislocation of the dens, the lengths of the separated bone and the base of the dens remaining on the cranial aspect of the axis were measured; the sum of these 2 measurements was considered the length of the dens for calculation of the DALR. In addition, the dogs with aplasia of the dens were excluded from the analyses.

Neurologic status

All dogs underwent preoperative and postoperative neurologic examinations. The severity of neurologic abnormalities was graded according to the scale of Stalin et al,[27] where 0 = normal, 1 = signs of neck pain with or without mild ataxia, 2 = ambulatory with moderate-to-severe ataxia or paresis, 3 = nonambulatory tetraparesis, 4 = tetraplegia, and 5 = death or euthanasia. For purposes of the present study, recovery was defined as an improvement in neurologic status as determined by neurologic examination findings, compared with neurologic status before surgery, and a regained ability to walk without signs of pain. Dogs with neurologic grades of 0 to 2 were considered to have recovered when normal walking without apparent pain was achieved, and those with grades of 3 or 4 were considered to have recovered when pain-free walking was possible, although mild ataxia or wobbling may have persisted.

Surgical procedure

Each dog was anesthetized and immobilized in dorsal recumbency with its neck extended. The mandible was fixed cranially, and the forelimbs were pulled caudally. Ventral fixation of the atlantoaxial joint was performed by use of the techniques of Schulz et al[28] and Shores et al.[1]

Statistical analysis

Statistical analysis was performed with statistical processing software.[c] The Mann–Whitney *U* test was used for comparisons of age (in months) at the time of surgery and of CT values between the IODA group overall or the individual IODA groups (IODA-cranial, IODA-medial, and IODA-caudal groups) and the non-IODA group, for comparison of body weight and of DALR between the IODA group overall and the non-IODA group, for comparison of the age (in months) at the time of surgery between the DA and the ID groups, and for comparison of severity scores of spinal cord injuries between the IODA and non-IODA groups. χ^2 Tests were used to compare the presence or absence of concomitant IODA in the DA and ID groups, and the outcomes after AAI surgery in the IODA and non-IODA groups. A value of $P < 0.05$ was considered significant.

Results

Computed tomography values of the midline region were measured in transverse CT images at the cranialmost, central, and caudalmost portions of the dorsal arch of the atlas in 106 dogs. Assessment for the presence or absence of dysplasia revealed IODA in 75 dogs (IODA group) and the absence of IODA in 31 dogs (non-IODA group). The IODA group included 21 Yorkshire Terriers, 20 Chihuahuas, 18 Toy Poodles, 6 mixed-breed dogs, 3 Pomeranians, 2 Shih Tzu, 2 Maltese, 1 Miniature Dachshund, 1 Japanese Chin, and 1 Cavalier King Charles Spaniel. The non-IODA group included 12 Miniature Dachshunds, 9 Chihuahuas, 5 Toy Poodles, 3 Yorkshire Terriers, 1 Shih Tzu, and 1 Papillon. Information regarding the sex, age, and body weight of the 106 dogs included

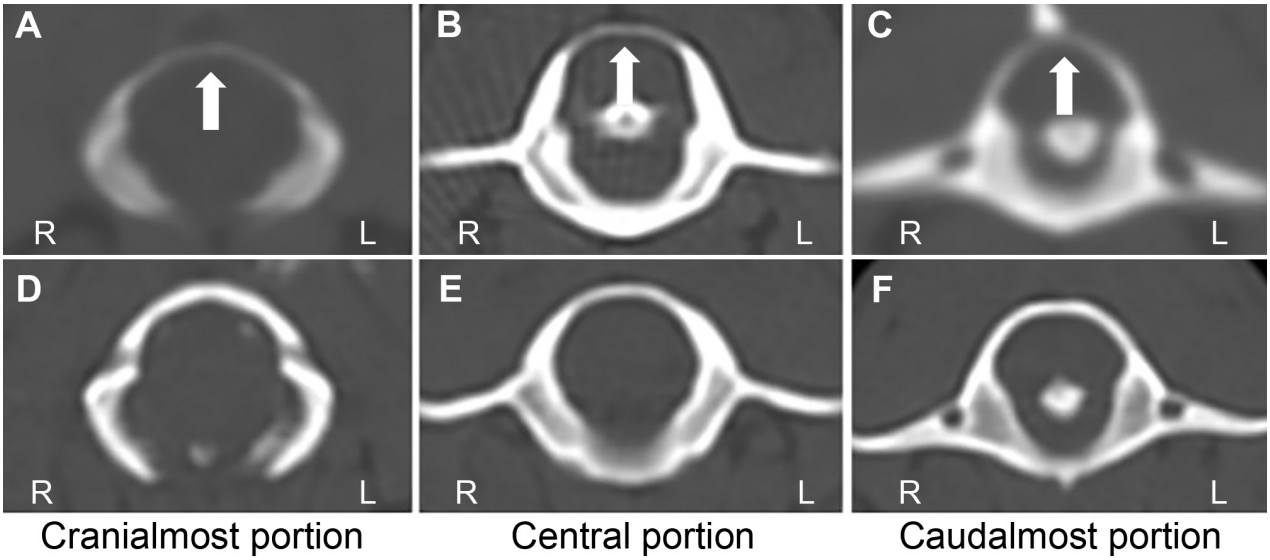

Figure 2—Representative transverse images (derived via 3D-MPR of CT images obtained with a bone window setting) of the cranialmost (A and D), central (B and E), and caudalmost (C and F) portions of the atlas in 6 dogs with AAI. The bone window setting had a window width of 2,500 HU and a window level of 500 HU. Dogs were classified as having IODA when the CT value of the midline region of the dorsal arch of the atlas in any of the 3 portions (arrow) was < 600 HU. A—Image obtained from a 66-month-old castrated male Chihuahua with AAI and incomplete ossification of the cranialmost portion of the dorsal neural arch of the atlas (assigned to the IODA-cranial group). The CT value of the midline region of the dorsal arch of the atlas is 247 HU. B—Image obtained from a 122-month-old sexually intact male Yorkshire Terrier with AAI and incomplete ossification of the central portion of the dorsal neural arch of the atlas (assigned to the IODA-central group). The CT value of the midline region of the dorsal arch of the atlas is 325 HU. C—Image obtained from an 89-month-old spayed female Maltese with AAI and incomplete ossification of the caudalmost portion of the dorsal neural arch of the atlas (assigned to the IODA-caudal group). The CT value of the midline region of the dorsal neural arch of the atlas is 311 HU. D through F—Images obtained from a 96-month-old sexually intact female Chihuahua with AAI and no evidence of IODA (assigned to the non-IODA group). The CT values of the midline region of the cranialmost (D), central (C), and caudalmost (F) portions of the dorsal neural arch of the atlas are 1,502 HU, 1,191 HU, and 1,048 HU, respectively. L = Left. R = Right.

in the study was summarized **(Table 1)**. There was no significant (P = 0.867) difference in mean age between the IODA and the non-IODA groups at the time of surgery. The mean body weight of the non-IODA group was significantly (P = 0.002) higher than that of the IODA group.

Investigation of the location of incomplete ossification in the cranialmost, central, and caudalmost portions of the dorsal arch of the atlas in the 75 dogs in the IODA group revealed that incomplete ossification was present in the cranialmost portion in 59 dogs (IODA-cranial group), in the central portion in 39 dogs (IODA-central group), and in the caudalmost portion in 28 dogs (IODA-caudal group). In 35 dogs, incomplete ossification was present in only 1 portion of the dorsal arch of the atlas, in 29 dogs it was present in 2 portions, and in 11 dogs it was present in all 3 portions. The CT values in the midline of the cranialmost, central, and caudalmost portions of the dorsal arch of the atlas in the dogs with and without IODA were assessed **(Table 2)**.

A comparison of age at the time of surgery for each of the 3 IODA subgroups and the non-IODA group revealed that the mean age at the time of surgery for the IODA-central group was significantly (P = 0.049) higher than that for the non-IODA group **(Table 3)**. The ages at the time of surgery for the IODA-cranial and the IODA-caudal groups were not

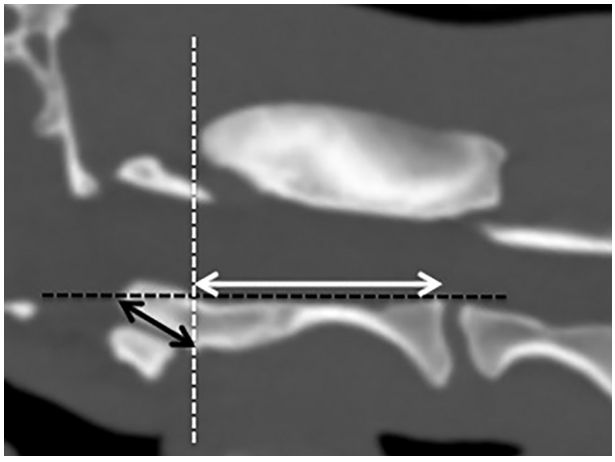

Figure 3—Median image of the axis of a 22-month-old sexually intact male Beagle reconstructed from CT images (window width, 2,500 HU; window level, 500 HU) by 3D-MPR to illustrate the calculation of the DALR in dogs with AAI. To determine the DALR, a line that passes through the tip of the dens and the dorsocaudal aspect of the body of the axis is first drawn (black dashed line). Then, another line is drawn perpendicular to the first, passing through the base of the ventral aspect of the dens (white dashed line). The length of the dens (black double arrow) is defined as the distance from the tip to the ventral base of the dens. The length of the body of the axis (white double arrow) is defined as the distance from the point of intersection of the 2 dashed lines to the dorsocaudal aspect of the axis. The DALR is defined as the ratio of the length of the dens to that of the axis body.

significantly (P = 0.865 and P = 0.726, respectively) different from the age for the non-IODA group.

Assessment of CT images for the presence of DAs revealed aplasia of the dens in 11 of the 106 dogs and separation of the dens was confirmed in 38 of the 106 dogs. Data for dogs with aplasia of the dens were not included in the analysis of DALR; hence, data from 67 dogs in the IODA group and 28 dogs in the non-IODA group were evaluated. The mean ± SD overall DALR for the dogs (excluding those with aplasia of the dens [n = 95]) was 0.41 ± 0.12 (median, 0.39; range, 0.07 to 0.87). There was no significant (P = 0.163) difference in DALR between the IODA group (mean, 0.40 ± 0.12; median, 0.38; range, 0.07 to 0.87) and the non-IODA group (mean, 0.44 ± 0.12; median, 0.40; range, 0.24 to 0.77).

The presence of DAs was evident in 70 of 106 (66.0%) dogs (DA group); 36 of 106 (34.0%) dogs had no obvious DAs (ID group). Concomitant IODA was observed in 51 of 70 (72.9%) dogs in the DA group and 24 of 36 (66.7%) dogs in the ID group; no significant (P = 0.507) difference was observed in the rate of concomitant IODA between the 2 groups. The presence of DAs was evident in 19 of 31 (61.3%) dogs of the non-IODA group, and 12 of 31 (38.7%) dogs had neither IODA nor DAs.

The age at the time of surgery for the DA group (mean, 35.5 ± 41.4 months; median, 12.0 months; range, 5 to 149 months) was significantly lower (P < 0.001) than the age at the time of surgery for the ID group (mean, 60.1 ± 44.0 months; median, 60.0 months; range, 8 to 154 months). Dens abnormalities were detected in all 59 dogs in the IODA-cranial group, in 32 of 39 dogs in the IODA-central group, and in 19 of 28 dogs in the IODA-caudal group.

For the IODA and non-IODA groups, mean neurologic grade before ventral fixation of the atlantoaxial joint was 2.5 and 2.3, respectively; after surgery, the neurologic grade was 0.4 and 1.1, respectively. The assessed severity of spinal cord injury before ventral fixation of the atlantoaxial joint in the IODA and non-IODA groups did not differ (P = 0.806); similarly, spinal cord injury grade after surgery was not significantly (P = 0.142) different between the 2 groups. The postoperative recovery percentage in the IODA group was 94.7% (71/75 dogs), which was significantly (P = 0.001) higher than the postoperative recovery percentage in the non-IODA group (71.0% [22/31 dogs]; **Table 4**). In the IODA group, 2 dogs died as a result of respiratory failure within 7 days after surgery. In the non-IODA group, 2 dogs died as a result of respiratory failure within 10 days after surgery; a third dog died of aspiration pneumonia during the in-patient care period, and a fourth dog died of status epilepticus 1 month after surgery.

Table 1—Demographic information for 106 dogs with AAI that did or did not have evidence of IODA on preoperative CT images and subsequently underwent atlantoaxial ventral fixation.

	Group	
Variable	**IODA (n = 75)**	**Non-IODA (n = 31)**
Sex (No. of dogs)		
Sexually intact male	22	10
Castrated male	16	3
Sexually intact female	26	12
Spayed female	11	6
Age (mo)		
Mean ± SD	44.9 ± 45.7	41.6 ± 39.1*
Median	19.0	17.5
Range	5.0–154.0	5.0–125.0
Weight (kg)		
Mean ± SD	2.3 ± 1.2	3.0 ± 1.3†
Median	2.0	3.0
Range	1.0–9.0	1.0–7.0

*Mean value for the non-IODA group was not significantly (P = 0.867) different from that for the IODA group. †Mean value for the non-IODA group was significantly (P = 0.002) different from that for the IODA group.

Discussion

The apical ligament, alar ligament, and transverse ligament of the atlas, which stabilize the atlantoaxial joint, are attached to the dens of the

Table 2—Comparison of the CT values (HU) in the midline region of the cranialmost, central, and caudalmost portions of the dorsal neural arch of the atlas in the dogs with and without IODA in Table 1.

	Portion of the dorsal arch of the atlas		
Group	**Cranialmost**	**Central**	**Caudalmost**
IODA (n = 75)			
Mean ± SD	374.0 ± 119.5[a]	376.4 ± 112.9[b]	305.0 ± 142.2[c]
Median	384.0	378.0	309.0
Range	60.0–593.0	85.0–582.0	39.0–586.0
Non-IODA (n = 31)			
Mean ± SD	1,093.0 ± 322.9[a]	1,358.8 ± 435.1[b]	1,338.8 ± 396.1[c]
Median	1,046.0	1,331.1	1,295.0
Range	655.0–1,912.0	787.0–2,484.0	808.0–2,476.0

Computed tomography values of the midline region were measured in transverse CT images (obtained with a bone window setting) at the cranialmost, central, and caudalmost portions of the dorsal arch of the atlas in 106 dogs. The bone window setting had a window width of 2,500 HU and a window level of 500 HU. Dogs were classified as having IODA when the CT value of the midline region of the dorsal arch of the atlas in any of the 3 portions was < 600 HU.

[a–c]Values with the same superscript letter differ significantly (each P < 0.001).

Table 3—Comparison of age at the time of surgery for AAI among the IODA subgroups (IODA-cranial [n = 59], IODA-central [39], and IODA-caudal [28] groups) and the non-IODA group (n = 31) of dogs with AAI in Tables 1 and 2.

		Age (mo)		
Group	**Subgroup**	**Mean ± SD**	**Median**	**Range**
IODA	IODA-cranial	42.2 ± 45.2	14.0	5.0–154.0
	IODA-central	61.9 ± 48.9*	49.0	5.0–154.0
	IODA-caudal	46.3 ± 45.8	27.5	6.0–149.0
Non-IODA	—	41.6 ± 39.1*	17.5	5.0–125.0

*The age at time of surgery for the IODA-central group was significantly (P = 0.049) higher than that of the non-IODA group. There were 75 dogs in the IODA group. In 35 dogs, incomplete ossification was present in only 1 portion of the dorsal arch of the atlas, in 29 dogs it was present in 2 portions, and in 11 dogs it was present in all 3 portions.

— = Not applicable.

Table 4—Neurologic assessment of spinal cord injury before and after ventral fixation of the atlantoaxial joint in the dogs with and without IODA in Table 1.

			Postoperative neurologic outcome		
Group	**Preoperative neurologic grade**	**No. of dogs**	**Recovered**	**No improvement**	**Died**
IODA	0	6	6	0	0
	1	11	11	0	0
	2	33	31	2	0
	3	11	10	0	1
	4	14	13	0	1
Total		**75**	**71**	**2**	**2**
Non-IODA	0	3	3	0	0
	1	4	3	0	1
	2	14	12	1	1
	3	6	3	3	0
	4	4	1	1	2
Total		**31**	**22**	**5**	**4**

For each dog, the severity of neurologic abnormalities before and after ventral fixation of the atlantoaxial joint was graded as follows: 0 = normal, 1 = neck pain with or without mild ataxia, 2 = ambulatory with moderate-to-severe ataxia or paresis, 3 = nonambulatory tetraparesis, 4 = tetraplegia, and 5 = death (or euthanasia). Recovery was defined as an improvement in neurologic status as determined by neurologic examination findings, compared with neurologic status before surgery, and a regained ability to walk without signs of pain. Dogs with neurologic grades of 0 to 2 were considered to have recovered when normal walking without pain was achieved, and those with grades of 3 or 4 were considered to have recovered when pain-free walking was possible, although mild ataxia or wobbling may have persisted.

axis; thus, DAs result in AAI. Other factors have also been reported to cause AAI, among which incomplete ossification of the atlas has been shown to be highly correlated with AAI.[7] To our knowledge, no prior reports have addressed incomplete ossification of the atlas in TBDs, which are susceptible to AAI. However, the present study involving retrospective assessment of CT images from clinical cases of AAI—primarily among TBDs—revealed that 70.8% (75/106) of dogs with AAI have bone dysplasia in the dorsal arch of the atlas. Because the dorsal arch of the atlas was thin, it was difficult to set a region of interest for the measurement of the CT value of IODA. When a circular region of interest is designated, neighboring soft tissues are included in the measurement region; as a result, there is a risk that the CT value of IODA is low. Thus, for this measurement, the cranialmost, central, and caudalmost points of the dorsal arch of the atlas were examined, and the highest CT value for the dorsal arch of the atlas was used.

Incomplete ossification of the dorsal neural arch of the atlas with AAI is often detected in Yorkshire Terriers, Chihuahuas, and Toy Poodles, but only 1 of 13 Miniature Dachshunds in the present study had IODA. Incomplete ossification of the dorsal neural arch of the atlas is very rare in Dachshunds, and dogs of this breed often have a dorsal arch of the atlas that is normal in structure. If the dorsal arch has incomplete ossification, then dorsal stabilization (a surgical treatment for AAI) does not result in an adequate braking effect and thus is contraindicated. However, dorsal stabilization can be performed in AAI-affected Miniature Dachshunds that have a normal dorsal arch of the atlas. In the present study, dogs with AAI and concomitant IODA were assigned to subgroups on the basis of the presence or absence of IODA in each of 3 portions (cranialmost, central, and caudalmost portions) of the dorsal neural arch of the atlas. Assessment of these subgroups revealed that dogs with incomplete ossification of the central portion had a higher mean age at the time of surgery for AAI onset

than non-IODA dogs. Thus, if the central portion of the dorsal arch of the atlas has incomplete ossification, a full braking effect of the dorsal atlantoaxial ligament cannot be obtained, and the ligaments attached to the dens will continue to be chronically exposed to an excessive tension load. This excessive tension load may induce secondary degeneration or rupture of these ligaments and trigger the onset of AAI in a dog's middle to advanced age.

Incomplete ossification of the dorsal arch of the atlas has been recognized as postnatal abnormal ossification in the raphe of the dorsal side of the arch of the atlas.[7,15,16] Results of the present study indicated that there is a difference between the cranialmost, central, and caudalmost portions of the dorsal arch of the atlas in the process of fusion of the raphe of the dorsal side of the arch, although the mechanism is unknown. Because incomplete ossification appeared most often in the cranialmost portion, the development process may be affected by other dysplasias (occipital dysplasia, atlantooccipital instability, or atlantooccipital overlapping) at the junction of the occipital bone and the atlas. Dogs with AAI with concomitant IODA generally have a smaller physique than those with AAI without concomitant IODA. Further, dogs with AAI and concomitant DAs underwent surgery for AAI at a younger age than did those with an ID; this finding was similar to an observation in a previous report,[2] but no correlation between DAs and IODA was found in the present study. Among the dogs of the present study, it was interesting that IODA and DAs were present in approximately 50% and only IODA or DAs were present in approximately 20%. Atlantoaxial instability was present with neither concomitant DA nor concomitant IODA in 12 dogs (11.3%). Other factors, such as defects of the transverse ligament of the atlas, are also known to cause AAI,[8] suggesting that factors other than DA and IODA were responsible for AAI in those dogs.

In the present study, the presence of IODA and the severity of spinal cord injury (as determined by assignment of a subjective neurologic grade) did not appear to be related. With or without concurrent IODA, implementation of ventral fixation of the atlantoaxial joint resulted in postoperative improvement in the severity of spinal cord injury in dogs with AAI. However, the non-IODA group had a lower postoperative recovery percentage, which suggested that other cranial junction abnormalities—including hydrocephalus or syringomyelia—may have been involved. For example, the dog in the non-IODA group that died of status epilepticus 1 month after surgery had the greatest dilatation of the lateral ventricles of all the study dogs, as revealed by preoperative MRI. When marked dilatation of the ventricles is identified, as in that dog, a ventriculoperitoneal shunt might be effective.[29]

Results of the present study indicated that in dogs with AAI (predominantly TBDs) that were evaluated, concomitant DA or concomitant IODA was common. In particular, for dogs with incomplete ossification in the central part of the dorsal neural arch of the atlas, time of surgery was at middle to advanced age; IODA is inferred to be a cause of AAI onset in middle to advanced ages. The omission of a control group of TBDs without AAI to confirm that IODA was more frequent in dogs with AAI than in unaffected TBDs was a limitation of this study. In addition, histologic evaluation of the portion of the dorsal arch of the atlas where ossification was incomplete was not carried out because the study was a retrospective assessment of clinical cases. Various factors are involved in the pathogenesis of cranial junction abnormalities, including AAI, but these remain poorly understood. Additional research is necessary to investigate not only DAs and IODA but also the relevance of other factors in the development of cranial junction abnormalities in dogs.

Acknowledgments

No third-party funding or support was received for this study or the writing or publication of this manuscript. The authors declare that there were no conflicts of interest.

The authors thank Editage (www.editage.jp) for English language editing.

Footnotes

a. Aquilion PRIME (TSX-303a) 80- and 160-slice CT scanner, Toshiba Medical Systems Corp, Tochigi, Japan.
b. OsiriX DICOM Viewer, Pixmeo SARL, Geneva, Switzerland.
c. SPSS Statistics for Windows, version 23.0, IBM Corp, Armonk, NY.

References

1. Shores A, Tepper LC. A modified ventral approach to the atlantoaxial junction in the dog. *Vet Surg* 2007;36:765–770.
2. Beaver DP, Ellison GW, Lewis DD, et al. Risk factors affecting the outcome of surgery for atlantoaxial subluxation in dogs: 46 cases (1978–1998). *J Am Vet Med Assoc* 2000;216:1104–1109.
3. Denny HR, Gibbs C, Waterman A. Atlantoaxial subluxation in the dog: a review of thirty cases and an evaluation of treatment by lag screw fixation. *J Small Anim Pract* 1998;29:37–47.
4. McCarthy RJ, Lews DD, Hosgood G. Atlantoaxial luxation in dogs. *Compend Contin Educ Pract Vet* 1995;17:215–226.
5. Thomas WB, Sorjonen DC, Simpson ST. Surgical management of atlantoaxial subluxation in 23 dogs. *Vet Surg* 1991;20:409–412.
6. Geary JC, Oliver JE, Hoerlein BF. Atlantoaxial subluxation in the canine. *J Small Anim Pract* 1967;8:577–582.
7. Parry AT, Upjohn MM, Schlegl K, et al. Computed tomography variations in morphology of the canine atlas in dogs with and without atlantoaxial subluxation. *Vet Radiol Ultrasound* 2010;51:596–600.
8. Watson AG, de Lahunta A. Atlantoaxial subluxation and absence of transverse ligament of the atlas in a dog. *J Am Vet Med Assoc* 1989;195:235–237.
9. Downey RS. An unusual cause of tetraplegia in a dog. *Can Vet J* 1967;8:216–217.
10. Zaki FA. Odontoid process dysplasia in a dog. *J Small Anim Pract* 1980;21:227–234.
11. Johnson SG, Hulse DA. Odontoid dysplasia with atlantoaxial instability in a dog. *J Am Anim Hosp Assoc* 1989;25:400–404.
12. Ladds P, Guffy M, Blauch B, et al. Congenital odontoid process separation in two dogs. *J Small Anim Pract* 1971;12:463–471.
13. Forterre F, Precht C, Riedinger B, et al. Biomechanical properties of the atlantoaxial joint with naturally-occurring instability in a toy breed dog. *Vet Comp Orthop Traumatol* 2015;28:355–358.

14. Takahashi F, Hakozaki T, Kanno N, et al. Evaluation of the dens-to-axis length ratio and dens angle in toy breed dogs with and without atlantoaxial instability and in healthy Beagles. *Am J Vet Res* 2017;78:1400–1405.
15. Warren-Smith CM, Kneissl S, Benigni L, et al. Incomplete ossification of the atlas in dogs with cervical signs. *Vet Radiol Ultrasound* 2009;50:635–638.
16. Owen MC, Davis SH, Worth AJ. Imaging diagnosis — traumatic myelopathy in a dog with incomplete ossification of the dorsal lamina of the atlas. *Vet Radiol Ultrasound* 2008;49:570–572.
17. Watson AG, Evans HE, de Lahunta A. Ossification of the atlas-axis complex in the dog. *Anat Histol Embryol* 1986;15:122–138.
18. Evans HE. The skeleton. In: *Miller's anatomy of the dog.* 3rd ed. Philadelphia: WB Saunders Co, 1993;166–170.
19. Currarino G, Rollins N, Diehl JT. Congenital defects of the posterior arch of the atlas: a report of seven cases including an affected mother and son. *Am J Neuroradiol* 1994;15:249–254.
20. Gangopadhyay S, Aslam M. Posterior arch defects of the atlas: significance in trauma and literature review. *Eur J Emerg Med* 2003;10:238–240.
21. Schulze PJ, Buurman R. Absence of the posterior arch of the atlas. *AJR Am J Roentgenol* 1980;134:178–180.
22. Schrödel MH, Braun V, Stolpe E, et al. Coincidental deficiency of the posterior arch of the atlas and thalassaemia minor: possible pitfalls in a trauma victim. *Emerg Med J* 2005;22:526–528.
23. Sharma A, Gaikwad SB, Deol PS, et al. Partial aplasia of the posterior arch of the atlas with an isolated posterior arch remnant: findings in three cases. *Am J Neuroradiol* 2000;21:1167–1171.
24. O'Sullivan AW, McManus F. Occult congenital anomaly of the atlas presenting in the setting of acute trauma. *Emerg Med J* 2004;21:639–640.
25. Rivero MA, Vázquez JM, Gil F, et al. CT-soft tissue window of the cranial abdomen in clinically normal dogs: an anatomical description using macroscopic cross-sections with vascular injection. *Anat Histol Embryol* 2009;38:18–22.
26. Sogo M, Ikebe K, Yang Tsung-Chieh, et al. Assessment of bone density in the posterior maxilla based on Hounsfield units to enhance the initial stability of implants. *Clin Implant Dent Relat Res* 2012;14:e183–e187.
27. Stalin C, Gutierrez-Quintana R, Faller K, et al. A review of canine atlantoaxial joint subluxation. *Vet Comp Orthop Traumatol* 2015;28:1–8.
28. Schulz KS, Waldron DR, Fahie M. Application of ventral pins and polymethylmethacrylate for the management of atlantoaxial instability - results in nine dogs. *Vet Surg* 1997;26:317–325.
29. Hoerlein BF, Gage ED. Hydrocephalus. In: *Canine neurology.* 3rd ed. Philadelphia: WB Saunders Co, 1978;733–760.