Skeletal Aspects of the Atlanto-Occipital Fusion in a Japanese Brown Calf

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ABSTRACT. Atlanto-occipital fusion in a Japanese Brown calf was examined morphologically, paying special attention to skeletal changes. At the craniovertebral junction, the basal occipital bone fused to the cranial extremity of the ventral arch of the atlas with the rudiment of the atlantal centrum. The dens was not formed at the axis. These changes suggest that a hypocentrum and a centrum of the atlas derived from the first cervical sclerotome had failed to separate the occipital base from the proatlantal sclerotome including the apical element of the dens. Although a developmental disturbance at the cervical and thoracic vertebrae was also associated, critical neurological signs such as ataxia and paralysis were absent.

NOTE Anatomy

KEY WORDS: atlanto-occipital fusion, congenital anomaly, Japanese Brown calf.

Atlanto-occipital fusion is a developmental disorder due to abnormal segmentation and development of the causal occipital and cranial cervical sclerotomes [14, 16]. Although its incidence has been estimated to be very rare in the domestic animals, the affected animals often show some critical upper motor neurological signs and quadrilateral proprioceptive deficits that accompany the defect [14, 17]. There is little pertinent information available about skeletal abnormalities in cattle except a few case reports in the following breeds: Angus-Hereford cross, Ayrshire-Hereford cross, Charolais, Devon, Holstein, Japanese Black, and Polled Hereford [2, 3, 6, 9, 11, 19, 20]. In this paper, we report the morphological changes at the caudal parts of the occipital bone and the cervical vertebrae in atlanto-occipital fusion in a Japanese Brown calf were examined using a maceration specimen, and its pathogenesis was discussed from an embryological point of view.

The anomalous calf was a six-month-old female that weighed 132 kg at the time of the postmortem examination. At the physical examination prior to sacrifice under xylazine sedation, only brachycervical and growth disturbances were recognized. However, the calf was alert and responsive, and its vital signs were normal. Data on the dam, including her health conditions and medical treatment during pregnancy, were insufficient. Its sire was suspected to be the carrier of the mutant gene for chondrodysplastic dwarfism [13, 21]. Antibodies to some viruses (Aino, Akabane, BVD-MD, Chuzan), which would induce abnormal bovine birth [12], were negative in the calf serum.

At the postmortem examination, morphological abnormalities were seen in the craniovertebral junction and cervical vertebrae, such as firm attachment and incomplete articulation between the occipital bone and the atlas and scoliosis in the cervical regions, respectively. The dens of the axis was not observed. From maceration specimens, the wider caudal end of the basal occipital bone fused to the cranial extremity of the ventral arch with the rudiment of the atlantal centrum at the caudal articular surface of the atlas (Fig. 1A). Since the fused portion at the craniovertebral junction did not fully synostose, the occipital bone and the atlas could be separated intentionally without damage. The fused surface of the atlas was composed of the unseparated ventral arch and rudiment of the atlantal centrum (Fig. 1B). Although the lateral parts of the occipital bone, including the occipital condyle and the jugular process, were in a state of hypoplasia, no other area was fused to the cranial articular surface of the atlas. The cranial extremity of the axis was wider and depressed due to failure of dens formation (Fig. 1C). From the fifth cervical to the first thoracic vertebrae, the compartments of the vertebrae, such as the arch, spinous process, articular process, body, and transverse process, were irregularly fused; furthermore, the spinal axis curved to the left. From the second to the fifth thoracic vertebrae, the spinal processes showed left-side inclination.

Atlanto-occipital fusion in cattle is assumed to be congenital [2, 3, 9, 11, 20]; however, congenital defect, trauma, infection, and neoplasm are supposed to be causes of incompetence of the dens, which is often observed in craniovertebral disorders in man [5]. Resorption of the dens might also follow injury in man [4]. In the present case, morphological skeletal changes at the craniovertebral junction and in the region from the fifth cervical to the first thoracic vertebrae may have been congenitally induced because there was no evidence of postnatal fracture, inflammation, or trauma.

The structure of the craniovertebral junction is formed as a part of a very complex process in the early fetal period. The basal parts of the occipital bone arise from the parachordal cartilage and the first three sclerotomes. The marginal bone around the foramen magnum, the atlas, and the axis, originate from each corresponding segment after the sclerotomal reorganization of the original fourth to sixth sclerotomes [5, 14]. The fusion of the basal occipital bone to the ventral arch with a rudiment of the atlantal centrum in
the present case suggests that the failure of each sclerotomal junctional area to separate and the developmental error of the dens would probably occur in the early embryonic stage. In the normal atlas, a hypocentrum develops into the ventral arch, and a centrum joins the axis to form a part of its cranial articular surface and the base of the dens [8, 10, 18]. It has been reported that the apical part of the dens is derived from the proatlantal sclerotome, which forms the basioccipital part around the foramen magnum [15, 18]. Therefore, it is reasonable to assume that a hypocentrum and a centrum of
the atlas derived from the first cervical sclerotome had failed to separate the occipital base from the proatantal sclerotome, including the apical element of the dens. The dysgenetic process of bony compartments at the craniovertebral junction is illustrated in Fig. 2. Simultaneously, the separation of the sclerotomal segments between the fifth cervical and first thoracic vertebral regions would be incomplete. The bovine sclerotome-differentiating process starts in the first somite during the 23rd day and progresses caudally through somite pair 36 by the 28th day of development [7]. It may be possible that the disturbance of morphogenesis in the craniovertebral bony segments due to a dysgenetic agent occurs in the early embryonic period.

Since spinal cord compression and injury, which were induced by narrowing of the foramen magnum and vertebral canal at the atlanto-axial level and by an incomplete articulation due to the atlanto-axial subluxation, were not detected, neurological signs such as ataxia and paralysis would not occur.

Judging from the antibody titer to arthropod-borne viruses in the calf serum, it is difficult to evaluate the intrauterine infection because the early bovine embryo had no ability to induce antibodies and because the maternal antibodies had disappeared in the calf serum at six months after birth [1]. The pathogenicity of the viruses against the skeletal tissues was not demonstrated [12]. Therefore, there is less relation between these viral infections and the skeletal changes. Because of absence of the characteristic morphological changes of the long bone at the limbs [13] and useful information regarding dam pedigree, the present case cannot be categorized as bovine chondrodysplastic dwarfism.

In conclusion, the present anomaly is defined as an atlanto-occipital fusion due to separate error between the altantal centrum and hypocentrum and the occipital base.

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REFERENCES