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CLINICAL PRESENTATION OF PROSENCEPHALIC HYPOPLASIA IN A LIVE HOLSTEIN DAIRY CALF

PARHAM MOTTAGHIAN[1*], ADEL GHAREBAGHY[1], FATEMEHETEMADI[1], AMIN ANOUSHEPOUR[2] AND AHMAD ASGHARI[2]

1: Department of Clinical Sciences, Faculty of Veterinary Medicine, Tehran University, Tehran, Iran

2: Department of Clinical Science, Faculty of Veterinary Sciences, Science and Research Branch, Islamic Azad University, Tehran, Iran

***Corresponding Author E Mail:mottaghian@vetmed.ut.ac.ir; Phone Number: +989125492113 – Postal Code: 1487794643**

ABSTRACT

In this report, a case of prosencephalic hypoplasia in a live Holstein dairy calf is described. The emphasis is on the explanation of the clinical signs based the anatomic defects presented. The most significant malformations were cranioschisis and cerebral aplasia. The calf survived for approximately 4 days but was hypothermic, unable to stand, unable to suckle and blind. The possible cause of this congenital defect could not be ascertained.

Keywords: Congenital defect, Prosencephalic hypoplasia, Holstein dairy calf

INTRODUCTION

Congenital defects are structural or functional abnormalities that are present at birth, and may affect a single structure or function, parts of various systems or an entire system **(Dennis and Leipold, 1979)**. Congenital defects of the central nervous system (CNS) can affect only the CNS or the CNS and the craniofacial skeleton **(Leipold, 1986)**. Prosencephalic hypoplasia (cerebral aplasia) is a rare defect in forebrain induction and is characterized by the complete absence of telencephalon and diencephalon. The defect is associated with a small opening in the calvaria on the midline at the level of the

orbits where fluid, presumably CSF, emerges. There have been few reports of the defect in calves and lambs **(Cho, 1978; Washburn *et al*, 2004)**. **Cornillie *et al* (2004)** described a case of prosencephalic hypoplasia in a stillborn calf. The objective of this report is to explain a case of prosencephalic hypoplasia in a live Holstein dairy calf in Tehran, Iran **(February, 2014)** and in addition, to explain the observed clinical finding on the basis on the anatomic defect.

Case Report

The calf presented with inability to stand and defect in calvaria **(Figures 1 and 2)**. Complete examination showed that the calf was hypothermic (body temperature < 36°C). Heart rate and respiratory rate were 140 bpm and 66 bpm, respectively. The calf was blind, mydriatic and pupillary light reflexes were negative. The eyeballs were well developed. Jerk (slow-quick) nystagmus movements involving both eyes could be seen. Rigidity and dorsiflexion of the neck and extension of the head was obvious. The calf periodically showed paddling movements and vocalization. Spinal reflexes of all four limbs were intact. The facial sensation was normal. The calf was able to suckle and to void urine and feces normally. Keeping in a warm place, the calf survived for nearly 96 hours.

The presence of Bovine Viral diarrhea (BVD) virus was assessed by Polymerase Chain Reaction (PCR), in addition,serum samples taken before colostrum consumption was assessed for antibodies against BVD virus by ELISA, however, the result of both test were negative.

Obtained radiographs were also confirmative of the presence of the defect in the frontal bone.

Necropsy findings included cranioschisis on the midline at the level of the orbits, abnormally small cranial cavity and the absence of telencephalon (cerebral cortex, hippocampus, and striatum), diencephalon (globus pallidus, thalamus, hypothalamus, hypophysis), and mesencephalon. The cerebellum and caudal parts of the brainstem were present. No significant malformations were apparent in spinal cord and vertebral column.

Figure 1: Calf with prosencephalic hypoplasia

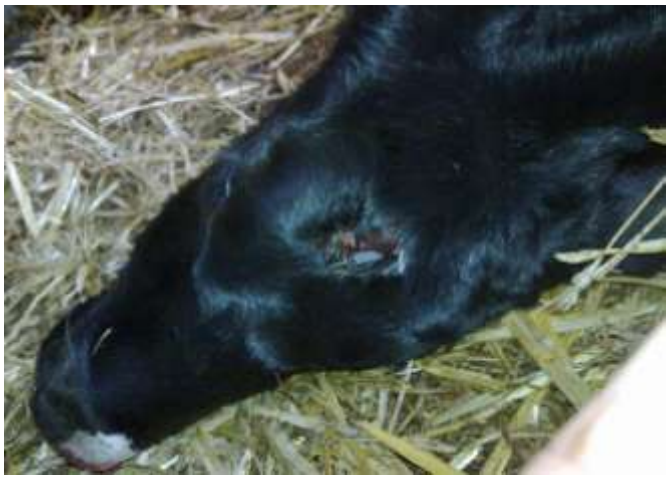

Figure 2: Cranioschisis. Defect in the center of calvaria

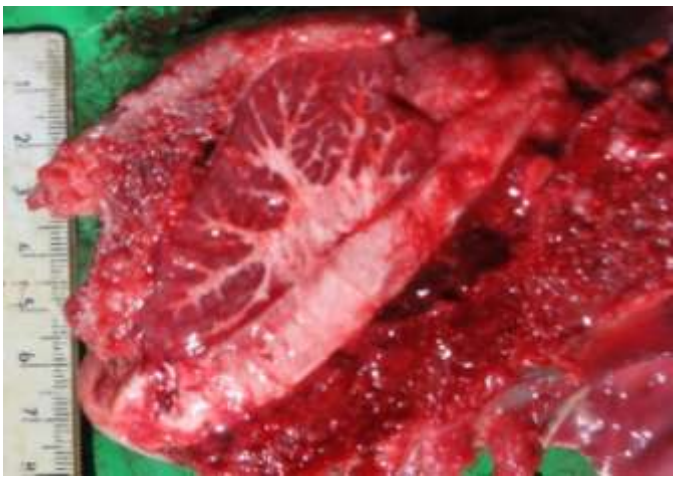

Figure 3: Mid-sagittal view of the cranium and affected brain. Complete absence of Prosencephalon and mesencephalon; Only Cerebellum and caudal parts of the brainstem are visible.

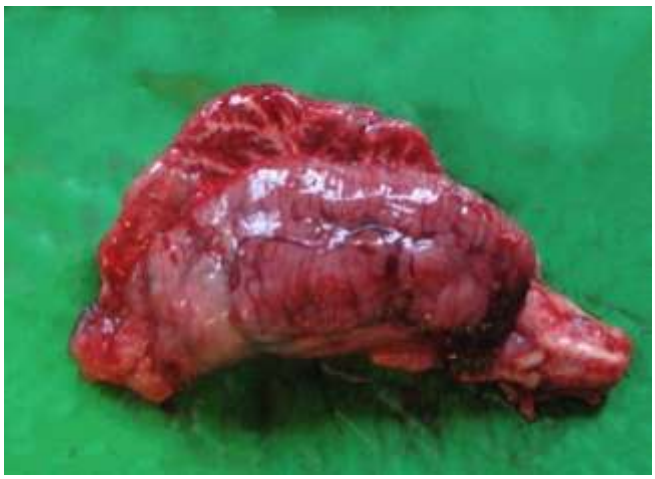

Figure 4: Dorsal view of the affected brain; Abnormally shaped cerebellum.

DISCUSSION

Prosencephalic Hypoplasia or cerebral aplasia is characterized by cranioschisis, absence of the diencephalon with the cerebral hemispheres and rostral midbrain, various forms of eye defects, and relatively normal development of caudal brain stem, cerebellum and spinal cord. True anencephaly, which means absence of brain, is a rare event, and the term has been misapplied to cases of cerebral aplasia, or prosencephalic hypoplasia **(Maxie, 2007)**.

Anomalies present vary considerably from case to case in the details of their expression, various combinations of typical malformations occur, and there is considerable variability in the development of morphological sequelae **(Maxie, 2007)**.

In the case presented, there were complete absence of prosencephalic (forebrain) and mesencephalic (Midbrain) Structures.The only part visible but abnormally shaped was the metencephalon (hindbrain) including thecerebellum and caudal parts ofthe brainstem (pons and medulla oblongata).

The eyeballs were normally sized andwell developed, but the calf was completely blind. The pupillary light reflexes were absent and the pupils were mydriatic. The ascending visual pathway runs from the retina via the optic nerves, midbrain and internal capsules to the visual areas in the occipital lobe of the cerebrum. The pupillary light reflex (PLR) measures the integrity of the retina, optic nerves, optic chiasm, pretectal and oculomotor nuclei in the midbrain, oculomotor nerve, ciliary ganglia, and constrictor pupillae muscle **(Smith *et al*, 2009)**. All the anatomic regions inside the cranium which are related to the visual

pathway and the pupillary light reflex arc (Telencephalon, diencephalon and the midbrain) were thoroughly absent in this case. Pathologic jerk (slow-quick) nystagmusis usually of vestibular origin. Nystagmus may occur in cerebellar disease but is usually more of a tremor of the globe than the jerk movements associated with vestibular disease **(DeLahunta, 2009)**. Animals affected by bilateral vestibular diseases do not show nystagmus. In the case presented, jerk nystagmus was obvious, but due to the extent and bilateral nature of the defect, it is unreliable to determine and localize the origin of the nystagmus.

Normal facial sensation and motor function in the calf were reflecting the intact functionality of both the trigeminal and facial nerve, respectively. In addition, sucking reflex as a primitive reflex which is under control of the brainstem was intact in this case.

The calf was unable to stand and to maintain the sternal recumbency. However the spinal reflexes were intact and the calf was capable of moving the limbs. In addition, rigidity and dorsiflexion of the neck and extension of the head was obvious. These signs may be associated with the defect and dysfunction of brain stem. It is necessary to mention that cerebral lesions do not cause abnormalities in posture and gait **(Radostits, 2007)**. No significant malformations were apparent in spinal cord and vertebral column.Normalspinal reflexes and the ability to void urine and evacuate feces could reflect the intact functionality of the thoracolumbar and sacral segments.

Hypothermia (body temperature less than 36˚ C) and bradycardia presented in this case could be associated with the absence of diencephalon including the hypothalamus and/or inability to consume sufficient amount of colostrum and energy deficiency.

One hypothesis about the mechanism involved in this brain malformation is as follows. If some factor caused the neurectoderm of the prosencephalon to fail to separate from the skin ectoderm at the level of the rostral neuropore, that could prevent the outgrowth of the telencephalic vesicles and create a defect in the closure of the calvaria, resulting in cerebral aplasia and cranioschisis, respectively. CSF could leak from the third ventricle of the diencephalon, which remains attached to the surface skin **(DeLahunta, 2009)**.

The cause of this malformation in animals is unknown but is one of the neural tube defects thought to result from a deficiency in folic acid in the diet of the human mother very early in development, before pregnancy may be diagnosed. Hyperthermia has also been

implicated as a cause of this neural tube defect. As mentioned earlier, the negative PCR and antibody ELISA results for BVD virus in this case proposed that the defect was not associated with intrauterine infection with BVD virus.

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