BILATERAL UNCOMPLICATED ANOPTHALMIA IN A BUFFALO CALF

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ABSTRACT

The present case reports the bilateral anopthalmia in a five day old male graded Murrah buffalo calf. The eyelids were closed and the eyeballs were not visible. The eyelids were undersized, upper eyelid had long cilia but the lower eye lid lack cilia. The palpebral fissure was so small as to prevent retraction of the eyelids at five days old. At three months old the size of the right palpebral fissure was 1.9×0.8 cm and that of left was 2.1×0.8 cm. Dissection of skull and brain at three months age revealed the abnormal tissue masses in the orbit which could not be easily identified as ocular. Orbital cavity showed remnants of ocular muscles, fibrous tissue and some fat. There was a small conjunctival sac. Cornea, sclera, choroid, iris, ciliary body, retina and lens were not found grossly. The nictitating membrane was not detectable. Optic foramen was pin head sized. The ventral aspect of the brain revealed hypotrophied optic chiasma and the optic tracts and absence of optic nerves. History and the bull semen register revealed that the specified animal and its dam were produced by the same bull's semen. Even though it was not confirmatory it was helpful to suggest hereditary involvement.

Keywords: anopthalmia, congenital defect, buffalo calf, inbreeding

INTRODUCTION

Anophthalmia means absence of one or both eyes. Eye balls arise from the out pouching of the diencephalon called optic vesicle (Chandrasekhara and Jagapathiramayya, 2013). During development of the eye, there is ample opportunity for developmental defects to occur (Leipold and Huston, 1968). Anophthalmia may arise embryologically in different ways. First, suppression of optic primordia during the development of fore brain, but this condition was unassociated with other defects of the nervous system. Such localized failure of the optic primordia resulted in a primary or uncomplicated anophthalmia. Second, in addition to failure of the optic primordial to develop from the forebrain, there was abnormal development of fore brain. The resulted anophthalmia appeared secondary to a generalized disorder of the anterior neural tube and termed as secondary or complicated anophthalmia. Third, the optic vesicles develop normally for a time and then at some stage of its development, or that of its successor, the optic cup, ceases to develop and undergo hypoplasia or atrophy. Such cases were classified as degenerative anophthalmia (Ron Ofri *et al*., 2008).

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HISTORY AND OBSERVATIONS

The calf when observed at five day old had normal vigor, appetite and body size. In the clinical examination, it was found that the calf did not have eyeballs bilaterally. The calf showed no other abnormalities both morphologically and clinically. The eyelids were undersized and closed and eye balls were not visible (Figure 1). Upon retraction of the lids manually a tissue which was not recognized as orbital was noticed. History reveals the inbreeding for two generations with same bull's semen, but the other calves born with that semen as well as the dam of the specified animal were quite normal.

RESULTS AND DISCUSSION

In most of the cases reported it was the white cattle that affected (Bahr, 2003; Julian, 1960; Leipold and Huston, 1968; Korkmaz and Ritas, 2012; Morimoto *et al*., 1995). No reports were found regarding this condition in black cattle. Morimoto *et al*. (1995) stated that the cattle breed in which anophthalmos was seen as a relatively common disorder was the Japanese brown cow. In this breed, animals were born with a remnant of pigmented tissue deep in the orbit and also with caudal sacral and tail abnormalities. Townsend. (2008) stated that in most cases it is associated with other defects, particularly those of posterior spinal column like absence of tail or posterior vertebral deformities.

There were no other vertebral column defects in this case (Figure 2). Morimoto *et al*. (1995) stated that the defects of the vertebral body such as wedge vertebra, hemivertebra, and sagittal cleft vertebra seen in the lumbar, sacral, and coccygial regions and the meandering of the axial line of abnormal vertebrae may suggest the failure of notochord formation in the early fetal period. The proportion of bilaterally anophthalmic calves having other defects was somewhat greater than for unilaterally affected calves (Leipold and Huston, 1968).

At three months age the animal was sent to the slaughter house and the head of the animal was collected. At three months old the size of the right palpebral fissure was 1.9×0.8 cm and that of left was 2.1×0.8 cm (Figure 3). The orbit was small because the normally enlarging globe regulates the development of the surrounding bony structures. Orbital cavity showed remnants of ocular muscles, fibrous tissue and some fat. There was a small conjunctival sac. Cornea, sclera, choroid, iris, ciliary body and lens were not found grossly. Lacrimal gland tissue was detected and large pea sized. The nictitating membrane was not detectable. Pin head sized optic foramen was found. The ventral aspect of the brain revealed hypotrophied optic chiasma and the optic tracts and absence of optic nerves (Figure 4). The intraorbital parts of the optic nerves were missing and no trace of nerve tissue was found in the stenotic optical canals.

A monogenic autosomal recessive inheritance may have caused bilateral anophthalmia (Bahr, 2003; Leipold, 1984). Chromosomal aberrations could not be detected in the affected calves (Bahr, 2003). History of the present case revealed the inbreeding for two generations but the other calves born with that semen were quite normal. The evidence was inadequate to evaluate any possible genetic basis for the anopthalmia. It was helpful to suggest hereditary involvement. Millemann *et al*. (2007) suggested the hypovitaminosisA as the cause for the birth of blind calves. The defects reported in this calf could

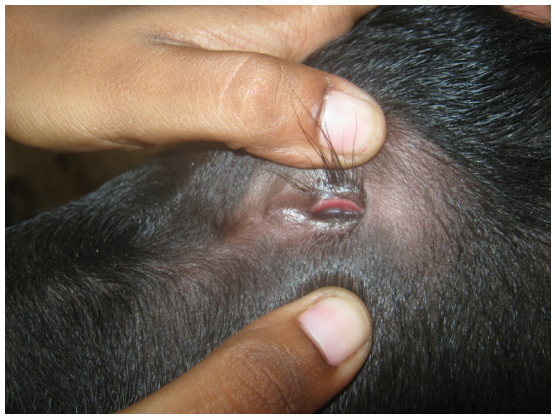

Figure 1. Five day old buffalo calf upper eye lid showing.

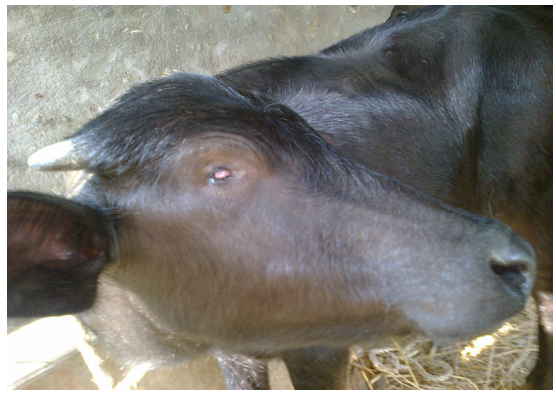

Figure 2. spinal column and tail well developed long cilia and lower eye lid shows no cilia.

Figure 3. Eye at 3 months age.

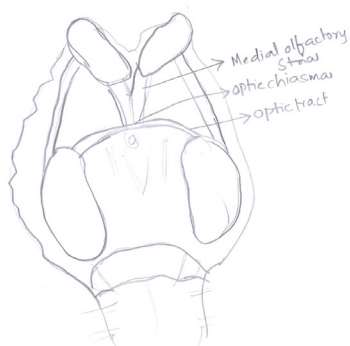

Figure 4. Line diagram showing ventral aspect of brain-absence of optic nerves and hypotrophied chiasma.

have resulted from a hereditary susceptibility in a predisposing environment.

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