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Congenital anophthalmia in a goat kid: A case report

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Abstract
A normally kidded telicherry doe was presented. It had delivered two normal live kids and one abnormal dead kid. That dead kid was identified as a monster under the very rare category of congenital bilateral anophthalmia (animals born without both eyes). The post-mortem examination of that monster kid was carried out and the findings are discussed.

Keywords: Bilateral anophthalmia, congenital defect, telicherry doe

Introduction
Abnormalities that occur during developmental stages of fetus are numerous and anophthalmia is one among them. It can affect all species of domestic animals Sofanda *et al* (2010) [9]. Anophthalmia is a greek word and it can be medically defined as absence of ocular tissue in the orbit. Mann (1953) [5] classified anophthalmia into three categories namely primary, secondary and consecutive or degenerative anophthalmia. In primary anophthalmia optic pit becomes enlarged and inside which optic vesicles are formed. Secondary anophthalmia mostly occurs in the early gestation period and there is failure of development of the anterior neural tube. In consecutive/degenerative anophthalmia optic vesicles degenerate and disappear subsequent to formation. Then there is regression of the partially developed eye rather than aplasia of the optic vesicle in an apparently anophthalmic orbit of extra ocular muscle insertion into a fibrous mass representing an aborted eye.
Anophthalmia can be caused by gene mutation, chromosomal abnormalities and environmental factors. Genetic mutation of SOX_2 gene is considered a major reason for phenotypic bilateral anophthalmia/microphthalmia. Mutation of some other genes like RBP_4 gene, OTX_2 gene, CHX_{10} general and RAX gene resulting in anophthalmic condition have been reported by Ragge *et al*., (2005) [8] & Wiliamson (2006) [10]. Chromosomal abnormalities like deletion, duplication and translocation have also resulted in an ophthalmic as reported by Brooks and Traboulsi (2005) [2]. Environmental factors like exposure of the mother to X-rays during gestation period, exposure to thalidomide, nutritional deficiency diseases like hypovitaminosis A have also ended up in both unilateral/bilateral anophthalmia in the prenatal stage as reported by Mason *et al*., (2003) [6]. Brown *et al*., (1975) [3] has reported some viral infections resulting in retinal dysplasia, cataract and globe developmental abnormalities.
In the present study a rare condition of bilateral primary anophthalmia noticed in a telicherry goat kid is discussed.

Case history and observation
A telicherry doe that kidded normally was presented in the morning hours with her two male kids that were alive and one female kid that was found dead at birth. Careful gross examination of that dead kid revealed absence of both eyes characteristic of bilateral primary anophthalmia. History further revealed that the flock didn't have any antecedents of similar malformations or abnormalities.

Treatment and discussion
Abnormalities present at birth result mostly due to the altered frequency of breed, geographical area, year, sex, parental age, level of nutrition. Exposure of the doe to adverse environmental factors can result in defect of a single body part or it's functional alteration thereby increasing prenatal mortality (Dennis and Leipold, 1979) [4]. The dead kid showed a single phenotypic defect namely bilateral anophthalmia.

This study revealed that this condition was usually associated with holoprosencephaly, a cephalic disorder in which the prosencephalon (the forebrain of embryo) fail to develop into two hemispheres causing facial deformities that may affect the eye, nose and upper lip (Paolo *et al*., 2005) [7]. In this case, no specific predisposing factor could be identified. Authur *et al*., (1993) [1] also reported that the anomalies observed in the off springs can also be due to infectious agents like viruses which can also be a factor in this case too. The presented dead kid showing primary form of bilateral anophthalmia has occured due to enlargement of optic pit and formation of optic vesicles because of the absence of optic tissue. In summary a rare case of bilateral anophthalmia in a dead telicherry kid was reported.

Fig 1: Foetus with Congenital anophthalmia

Reference

1. Arthur GH, Noakes DE, Pearson H. 6th edn. Veterinary Reproduction and Obstestrics (Theriogenology). Bailliere Tindal. 1993, 105-126.
2. Brooks BP, Traboulsi EI. Congenital malformations of the eye. In Duane's Clinical Ophthalmology on CD-ROM edition foundation Chapter 40 Edited by: Tasman W, Jaegar EA. Philadelphia: Lippincott. 2005, 1.
3. Brown TT, Bistner SI, De Lahunta A *et al*. Pathogenetic studies of infection of the bovine fetus with bovine viral diarrhoea virus II. Ocular lesion. Vet Pathol. 1975; 12:394-404.
4. Dennis SM, Leipold HW. Ovine congenital defects. The veterinary bulletin. 1979; 49(4):233-237.
5. Mann I. The developmental basic of eye malformation Philadelphia. JB Lippincott. 1953.
6. Mason CS, Buxton D, Gartside JF. Congenital ocular abnormalities in calves associated with maternal hypovitaminosis A. Vet Rec. 2003; 153:213-214.
7. Paolo T, Andrea R, Roberta B. Brain malformations. In: Paolo T, Andrea R and Raybaud B. Padiatric Neuroradiology: Brain, Head, Neck and spine. Springer. 2005, 92-95.
8. Ragge NK, Lorenz B, Schneider A, Bushby K, De Sanctis L, De Sanotis U, Salt A *et al*. SOX_2 anophthalmia syndrome Am J. Med Genet A. 2005; 135:1-8.
9. Sonfada ML, Sivachevan MN, Haruna Y, Wiam IM, Yahaya A. Incidence of congenital malformations in ruminants in the North Eastern region of Nigeria. International Journal of Animal and veterinary Advances. 2010; 2(1):1-4.
10. Williamson KA, Hever AM, Rainger J, Rogers RC, Magee A, Fiedler Z *et al*. Mutation in SOX_2 cause anophthalmia-esophageal-genital (AEG) syndrome. Hum Mol Genet. 2006; 15:1413-1422.