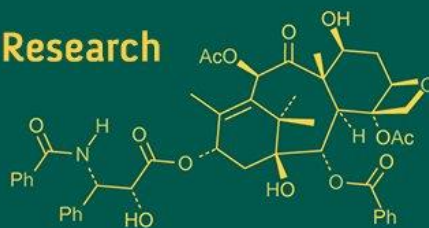


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Congenital bilateral anophthalmia in a Mecheri lamb: A case report

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Abstract

Congenital ocular malformations are uncommon developmental anomalies in sheep and may arise from genetic, nutritional, infectious, environmental, or teratogenic factors that affect embryogenesis. This report describes a rare case of congenital bilateral anophthalmia in a Mecheri lamb, one of the most populous indigenous meat sheep breeds in Tamil Nadu. A newborn lamb is delivered after a normal gestation period of approximately 150 days at the Mecheri Sheep Research Station, Pottaneri, exhibited complete absence of both eyes with markedly underdeveloped orbital regions, fused eyelids, and shortening of the facial bones and a compressed muzzle. No gross abnormalities were observed in the limbs, external genitalia, or body morphology of the offspring. The dam remained clinically healthy throughout gestation, with no history of nutritional deficiency, infectious disease, drug administration, or exposure to known teratogenic agents. Based on the gross clinical findings, the condition was diagnosed as congenital bilateral anophthalmia. Although the precise etiology could not be established, the anomaly most likely resulted from the disruption of optic vesicle development during early embryogenesis. This case represents one of the few documented reports of congenital bilateral anophthalmia in the Mecheri breed and highlights the importance of documenting congenital anomalies, maintaining breeding records, and undertaking pathological and genetic investigations to better understand their etiology and support the conservation and genetic improvement of indigenous sheep breeds.

Keywords: Anophthalmia, congenital anomaly, craniofacial malformation, Mecheri sheep, lambs, developmental defects

Introduction

Congenital malformation in sheep occurs as a result of genetic or environmental influences, which very often overlap to a greater or lesser degree (Angus, K. 1992) ^[1]. It may be structural or functional defects that occur during prenatal development and are present at birth. The abnormalities may result from genetic defects, chromosomal aberrations, nutritional imbalances, infectious diseases, environmental toxins, or exposure to teratogenic agents during critical stages of embryogenesis. (Noakes *et al.*, 2019; Constable *et al.*, 2017; Noden and de Lahunta, 2017) ^[6, 2, 8]. In livestock species, congenital defects can lead to neonatal mortality, reduced productivity, and economic losses for farmers. The Anophthalmia and Microphthalmia (A/M) are rare errors of eye development (with a combined prevalence of 3:100,000 to 30:100,000). Whereas true anophthalmia refers to the histological absence of ocular tissue in the orbit, microphthalmia ranges from extreme cases with only tiny or no visible remnants of globe in the orbit (also called “clinical anophthalmia”) to simple microphthalmia with structurally normal small eyes (axial length at least 2 SD below the mean for an individual of that age) (Verma, A.S and Fitz Patrick, D.R, 2007) ^[11]. The Ocular developmental anomalies reported in sheep include anophthalmia, microphthalmia, cyclopia, coloboma, and retinal dysplasia. The Anophthalmia is characterized by the complete absence of ocular tissue due to the failure of optic vesicle formation during embryonic development. The Bilateral anophthalmia is particularly rare and is frequently associated with abnormalities in adjacent craniofacial structures because ocular and facial tissues develop concurrently during embryogenesis (Dennis, 1975) ^[3]. Mecheri sheep are one of the most populous and economically important indigenous sheep breeds in Tamil Nadu and renowned for its adaptability to semi-arid conditions, hardiness, disease resistance, and superior meat production and these breed plays a vital role in the

livelihoods of small and marginal farmers across its breeding tract. Despite its economic and genetic importance, reports on congenital ocular anomalies in the Mecheri breed are exceedingly scarce. The present study describes a rare case of congenital bilateral anophthalmia in a Mecheri lamb, thereby contributing to the limited literature on congenital developmental anomalies in this valuable indigenous sheep breed.

Case History and Clinical Findings

A new born Mecheri lamb was observed at the Mecheri Sheep Research Station, Pottaneri, Salem District, Tamil Nadu, India. The dam was maintained under standard farm management conditions and naturally mated on January 12, 2026. The ewe delivered a single lamb on June 11, 2026, corresponding to a gestation length of 150 days, which falls within the normal gestation period for sheep. No history of disease, drug administration, vaccination-related complications, nutritional deficiency, or exposure to known teratogenic agents was reported during the pregnancy. The dam appeared clinically healthy throughout gestation, but the lamb died at birth and exhibited severe craniofacial abnormalities and generalized weakness. Gross examination revealed the complete absence of visible ocular structures on both sides of the head. The orbital regions were markedly underdeveloped, and the eyelids appeared to be fused over the orbital area. Facial bones are shortened and malformed, resulting in an abnormal craniofacial appearance. The muzzle appeared compressed, and the facial symmetry was distorted. No obvious abnormalities were observed in the limbs, external genitalia, or body morphology. Based on the gross clinical findings, the condition was diagnosed as congenital bilateral anophthalmia associated with craniofacial malformation.

Discussion

Congenital bilateral anophthalmia is an uncommon developmental anomaly resulting from the disruption of ocular morphogenesis during the early stages of embryogenesis. The eye development begins with the formation of the optic grooves from the developing forebrain, followed by evagination of the optic vesicles, induction of the lens placode, and subsequent differentiation of the retina and other ocular structures (Maxie *et al.*, 2016)^[5]. Failure of optic vesicle formation or interruption of these sequential developmental events results in the complete absence of the globe (true anophthalmia), whereas partial disruption may lead to varying degrees of microphthalmia (Noden and de Lahunta, 2017; Verma and FitzPatrick, 2007)^[8, 11]. In the present case, the complete bilateral absence of visible ocular structures, indicates that the developmental insult likely occurred during the early phase of organogenesis. (Figure 1). The neural crest cells contribute extensively to the formation of the orbit, facial bones, and connective tissue, as well as portions of the anterior segment of the eye. Therefore, disturbances in neural crest cell migration, proliferation, or differentiation can produce combined ocular and craniofacial defects, as observed in the present lamb (Noden and de Lahunta, 2017 and 1985)^[8, 7]. The aetiology of congenital anophthalmia is multifactorial and frequently involves interactions between genetic

susceptibility and environmental factors. The mutations affecting key developmental genes, including SOX2, PAX6, RAX, OTX2, VSX2, and BMP4, have been associated with anophthalmia and microphthalmia in humans and several domestic animal species. Although molecular investigations were not performed in the present case, a hereditary basis cannot be completely ruled out. As a congenital ocular defect have occasionally been reported in sheep and cattle, careful pedigree evaluation and monitoring of related animals are recommended before retaining the breeding stock. The environmental factors should also be considered in the differential diagnosis of congenital ocular anomalies (Leipold and Dennis, 1987)^[3]. Maternal vitamin A deficiency during early gestation is a well-recognized cause of ocular and central nervous system malformations in ruminants because retinoic acid plays a critical role in embryonic patterning and organogenesis (Noakes *et al.*, 2019)^[6]. Similarly, ingestion of teratogenic plants, exposure to toxic chemicals or certain pharmaceuticals, hyperthermia, and other environmental stressors during the critical period of foetal development may interfere with normal eye formation. In the present case, however, no history of nutritional deficiency, drug administration, or exposure to known teratogens was obtained from flock records (Singh *et al.*, 2014)^[10].

Congenital infections should also be considered when investigating foetal malformations in sheep. The viruses capable of crossing the placenta, including the border disease virus and bluetongue virus, have been associated with congenital abnormalities affecting the central nervous system and ocular tissues (Constable *et al.*, 2017)^[2]. Although the dam remained clinically healthy throughout gestation and no evidence of infectious disease was observed, laboratory investigations such as histopathology, virological examination, and molecular diagnostic assays are required to definitively exclude infectious etiologies (Radostits *et al.*, 2007)^[9]. The lamb in the present report completed a normal gestation period of 150 days, suggesting that foetal growth and maintenance of pregnancy were not adversely affected, despite the severe developmental defect. This observation supports the hypothesis that the anomaly originates during early embryogenesis rather than as a consequence of late gestational injury. Furthermore, the absence of gross abnormalities in the limbs, external genitalia, and other body systems indicates that the developmental disturbance was largely confined to the ocular and craniofacial regions of the body.

Reports describing the congenital bilateral anophthalmia in indigenous Indian sheep breeds are extremely rare. To the best of our knowledge, this is the first documented case in Mecheri lambs. The documentation of such congenital anomalies contributes to the understanding of developmental disorders in indigenous breeds and provides valuable baseline information for veterinary clinicians, pathologists, and breeding programs. Continued surveillance, detailed pathological examination, genetic investigations, and maintenance of accurate breeding records are essential to determine the underlying cause and minimize the recurrence of similar congenital defects in conservation and breeding herds.



Fig 1: Frontal view of the Mecheri lamb showing bilateral absence of ocular structures



Fig 2: Lateral view demonstrating malformed orbital regions and shortened facial bone

Conclusion

A rare case of congenital bilateral anophthalmia was observed in a Mecheri lamb born after a normal gestation period. The defect was characterized by the complete absence of both eyes and marked facial deformities. Although the exact aetiology could not be determined, the anomaly likely resulted from disturbances during the early embryonic development. Further studies involving pathological examination, genetic analysis, and epidemiological investigation are required to elucidate the underlying causes of congenital defects in Mecheri sheep.

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Conflict of Interest

The authors declare no conflicts of interest regarding the publication of this case report.

References

1. Angus K. Congenital malformations in sheep. In Practice. 1992;14(1):33-38.
2. Constable PD, Hinchcliff KW, Done SH, Grünberg W. Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs and Goats. 11th ed. St. Louis (MO): Elsevier; 2017.
3. Dennis SM. Congenital abnormalities of sheep and goats. Veterinary Bulletin. 1975;45:160-180.
4. Leipold HW, Dennis SM. Causes, nature and effect of bovine congenital defects. Advances in Veterinary Science and Comparative Medicine. 1987;31:197-271.
5. Maxie MG, editor. Jubb, Kennedy and Palmer's Pathology of Domestic Animals. Vol. 1. 6th ed. St. Louis (MO): Elsevier; 2016.
6. Noakes DE, Parkinson TJ, England GCW, Hewitt DA. Arthur's Veterinary Reproduction and Obstetrics. 10th ed. London: Elsevier; 2019.
7. Noden DM, de Lahunta A. The Embryology of Domestic Animals: Developmental Mechanisms and Malformations. Baltimore (MD): Williams & Wilkins; 1985.
8. Noden DM, de Lahunta A. The Embryology of Domestic Animals: Developmental Mechanisms and Malformations. 2nd ed. Chichester: Wiley-Blackwell; 2017.
9. Radostits OM, Gay CC, Hinchcliff KW, Constable PD. Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs and Goats. 10th ed. Edinburgh: Saunders Elsevier; 2007.
10. Singh AP, Pandey AK, Sharma A. Congenital anomalies in domestic animals: A review. Indian Journal of Veterinary Anatomy. 2014;26(1):1-8.
11. Verma AS, FitzPatrick DR. Anophthalmia and microphthalmia. Orphanet Journal of Rare Diseases. 2007;2:47.