

## CONGENITAL ANOPHTHALMOS IN A CROSSBRED MALABARI KID

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Congenital anophthalmos or anophthalmia in goats is scantily reported. It is the unilateral or bilateral absence of eye at birth and may be accompanied by other cranial abnormalities (Duplessis et al., 1982 and Sadler, 2004). The present report describes a rare case of bilateral anophthalmia in a kid.

A cross bred Malabari doe was brought to the Veterinary Dispensary, Avoly in Ernakulam District, Kerala. The owner reported that the animal has passed fluids about 1 hour 30 minutes back, but not yet kidded. They told that in previous kidding the animal kidded a dead foetus. They used to send the animal for grazing in areas where plenty of greens are available. The animal showed signs of abdominal straining and was clinically normal in other aspects.

The perineal region of the animal was thoroughly cleaned with water and 1 in 1000 potassium permanganate solution. The animal was examined per vaginally and the dead foetus in anterior presentation was exteriorized by manual traction. The foetus weighed 1.5 kg and showed developmental defects of the head region. Both eyeballs were not developed (anophthalmos) and the orbit did not contain any fibro- fatty tissue unlike the observations of Ashturkar et al. (1996). Eyelids were present, but eyelashes were not developed. The upper and lower lips of the kid also showed incomplete development.

The eye, which is a sensitive detector of light, develops from the wall of brain as the result of a series of inductions in a step- by-step manner. It develops precociously and will be three fourth of its final diameter at birth (Arey, 1965). PAX6 is the master gene for eye development (Sadler, 2004). Eyeball is most susceptible to terratogens during the 4<sup>th</sup> to 8<sup>th</sup> week of gestation and can get affected till the end of pregnancy (Singh, 1996). Exposure of embryo to terratogens during 5<sup>th</sup> or 6<sup>th</sup> week of embryonic life can cause lip defects.

Congenital anophthalmos has been reported in pig, horse, dog, cat, rat and guinea pig. In pig it is associated with maternal Vitamin A deficiency (Radostits et al., 2000 and Jones et al., 1997). According to Jones et al. (1997) in guinea pig this condition is inherited. Smith and Sherman (1994) observed that various chemical or viral terratogens may interfere with embryonic development of eye ball. They quoted that anophthalmia occurred in a lamb whose mother was exposed to apholate, an insect chemosterilant during pregnancy. Noden and Lahunta (1984) cited that viruses like Bovine Viral Diarrhoea Virus, Herpes Virus-2 and Rubella Virus can cause eye defects in domestic animals. They have reported that ingestion of the plant *Veratrum californicum* can cause eye defects like cyclopia in sheep. In the present case, anophthalmic condition was accompanied by upper and lower jaw defects and eyelids were developed similar to the findings

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of Duplessis et al. (1982). Congenital defects may occur due to genetic or environmental defects or by a combination of both. Maternal, placental and foetal factors influence the intra-uterine growth of foetus. For normal growth of the foetus, mother should get adequate amount of nutrients and the same should be transferred through the placenta. Since the animal was sent for grazing, the chance of deficiency of Vitamin A is very less, but chance of ingestion of any toxic plant or insecticide can not be overlooked. Since the foetus was dead, there might be a chance of viral infection. Since the animal kidded a dead foetus for the second time, there may be some underlying genetic factors also, contributing to the congenital defects.

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