ACHONDROPLASTIC CAPRINE FOETAL MONSTROSITY WITH ANURY, ATRESIA ANI AND SCROTAL HERNIA

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ABSTRACT

Bull Dog foetal monster or achondroplasia along with scrotal hernia, atresia ani and anury in Black Bengal goat is reported.

Key words : Achondroplasia, Bull dog foetus, Monster-kid, Black Bengal doe.

INTRODUCTION

The occurrence of achondroplasia has been reported rarely in dogs, cats, sheep and goats by Roberts (1971). The present communication reports an incidence of Bull Dog foetal monstrosity with anury, artesia ani, scrotal hernia, foetal ascites in a Black Bengal goat.

CASE HISTORY AND OBSERVATIONS

A full term pluriparous Black Bengal doe with a history of unsuccessful attempt to relieve dystocia by a Para veterinary staff was attended as a referral case. General condition of the doe was fair revealing kidding signs to have occurred four hours earlier before being referred. The birth passage was completely dilated and after sufficient lubrication and thorough obstetrical mutation and forced extraction, a dead foetal monster along with oedematous placenta was delivered per-vaginum. The fetal placenta was adventitious with diseased cotyledons.

The foetus, recovered in the present case was a full grown monster with extensive congenital deformities throughout its body. It weighed about 2.5 kg and its whole body was covered with short glossy hairs. The monster had pot belly, disproportionate dwarfism, a short vertebral column, abnormal short legs and relatively large, round head with cleft palate and protruding tongue with associated developmental defects like anury, scrotal hernia and atresia ani. The neck appeared to be short and thick. Eyes were small and ears were big. The neck appeared to be short and thick. Eyes were small and ears were big. The muscles of the trunk and ventral abdominal region were disproportionally developed leading to a enlarged abdomen with the accumulation of ascitic fluid. Sloss and Dufty (1980) suggested that obstruction by lymphatics prevents the circulation of peritoneal fluid and ascites could be due to diminished urinary excretion.

Limbs of the foetus were too short, as if vestigeal limbs represented as pelvic limbs and short, deformed, stumpy pectoral limbs with marked digital ankylosis and joint contracture. The genetic defects that caused the limb deficiencies could be associated with autosomal recessive genes and chromosomal aberrations (Lenz, 1980). Scrotal sac was thin and membranous and was filled with sticky fluids with herniation in which omentum passed down the inguinal canal in contact with spermatic cord lying in the cavity of tunica vaginalis. Widening of rima oris, agenesis of lips and dental pad devoid of teeth eruptions were evident. The monster revealed typical features of achondroplasia as described by Roberts (1971) and Ravindra Reddy *et al.* (2008). Achondroplasia of varying degrees have been attributed to lethal autosomal genes where line or inbreeding is practiced (Roberts loc. cit, Bakshi *et al.*, 1987).

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