DYSTOCIA DUE TO FOETAL HYDROCEPHALUS IN A BLACK BENGAL DOE

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ABSTRACT

A rare case of caprine foetal dystocia due to congenital hydrocephalus monster in a black Bengal doe and its successful delivery per vaginum has been reported.

Key words: Hydrocephalus, Dystocia, Doe

INTRODUCTION

Hydrocephaly can be defined as dropsical condition of the brain owing to abnormal accumulation of cerebrospinal fluid (CSF) in the cranial cavity and has been encountered as an infrequent congenital developmental anomaly in mammals causing foetal dystocia (McEntee-1990). In severe form there is marked thinning of cranial bones (Noakes et al., 2001). Further a variety of malformations may occur causing constitutional disorder of mouth, eyes and even vertebral column along with hydrocephalus due to genetic reasons owing to autosomal dominant genes with incomplete penetrance (Greene et al., 1973). It may also be caused by infectious etiology, nutritional and numerous environmental factors besides the genetic reasons (Kalman, 1989). Foetal hydrocephaly causing dystocia in bovine is very common but report on the incidence of hydrocephalus kid (Monster) causing foetal dystocia in goat is very sparse. This communication records an incidence of hydrocephalus full grown foetus leading to dystocia in a black Bengal doe for the first time in the locality and its successful delivery per vaginum.

CASE HISTORY AND OBSERVATION

A five years old pluriparous Black Bengal doe in its sixth gestation at full term was presented to the clinic after a lapse of 12 hrs with the onset of straining and kidding signs. The water bag was ruptured and the doe was not able to deliver the kid on its own. It was

straining intermittently. No foetal parts were seen outside.

TREATMENT AND DISCUSSION

The caudal epidural anesthesia was given using 2 ml of 2% Lignocaine hydrochloride and under strict asepsis per vaginal examination was made that revealed full cervical dilation and a dead foetus with enlarged skull, double the size of normal head obstructing the birth canal with extended fore limbs in anterior longitudinal presentation.

Applying pressure and on palpation by finger tips on the foetal head it revealed marked swelling and tenderness of the skull. The soft fluctuating consistency of foetal head had led to the possible diagnosis of congenital hydrocyphalus or foetal head abnormality with thinning of bones sculpture of the brain. After profuse lubrication of the birth canal with liberal application of home made coconut oil, foetal mani pulation and subsequent synchronous traction was applied which proved it an unsuccessful attempt. Hence stab incision was given on the cephalic region and instantly profuse release of amber colour fluid measuring about 475 ml took place. The foetal head then collapsed to a marked extent and foetal delivery was made with the help of small animal's orbital hook, and simultaneous traction from the traction points of foetal extremities. The dead foetal monster with enlarged head and faciomandibular abnormality weighing 1.8 kgs was opened for internal organs exploration specially of the brain. The placenta was manually recovered. No other foetus was lying in utero and a vaginal passery Lixen ® IU (Glaxo) was kept in utero for maintaining uterine hygiene. The doe immediately received intravenous infusion of parenteral rehydration fluid, Intalyte ® (Intas) 500 ml along with 1 ml Dexamethasone Sodium ® (Ranbaxy), intramuscular antibiotic coverage by Injection C-flox ® (a ciprofloxacin preparation of Intas) 2 ml and parenteral non-steroidal anti-inflammatory drug (NSAID) by Meloxicam injection (Inj. Melonex ® Intas) 2 ml. Both antibiotic and antinflammatory drugs were administered for another 4 days at the same dose rate.

It was hydrocephalus dead, male foetus with facio mandibular defects giving a grotesque look. Internal organs exploration revealed no abnormalities other than brain tissues. Abnormalities were detected in brain tissues and meninges along with marked thinning of the frontal, temporal and parietal bones. The ventricles of the head were distorted with the accumulation of excessive fluid and possibly due to autolytic changes as started in the dead foetus, the fluid turned into an amber colour as released on stab skull incision. Roberts (1986) described the malady as congenital hydrocephalus that could be attributed to a single autosomal recessive gene. McEntee (1990) described the malady in mammals as hydrocephaly owing to genetic reasons. There is a great variation in the observation of hydrocephalic fetuses which appeared fortuitous and is of definite scientific interest. It is generally classified as being either internal or external hydrocephaly based on the cerebrospinal fluid (CSF) accumulation inside or sometimes outside the ventricles causing pressure atrophy of the cerebral tissues (Gilman, 1956). This malady is assumed to arise from disturbances in normal circulation of CSF resulting from its altered production and or absorption (Fride, 1975). An autosomal dominant gene with incomplete penetrance (Greene et al., 1973) have been reported to be linked with hydrocephaly in bovine foetal monstrosity whereas it is known to be inherited in cattle and exacerbated in its malformation by co-existing hypo vitaminosis-A (Jubb and Kennedy 1970, Blood and

Henderson, 1971). However, Sane et al. (1994) opined that the cause of hydrocephaly is not definitely known but it is usually ascribed to the dearrangement of foetal circulation. Hereditary basis of this congenital malady does not hold good in the present case i.e. in hydrocephalic caprine foetal monstrosity as occurred in a black Bengal doe based on the observations, breeding history and analysis of all births in the whole goat flock of the animal owner. This was the first incidence in the locality and reported because of both scientific and obstetrical interest.

REFERENCES

- Blood, D.C. and Henderson, J.A. (1971) Veterinary Medicine 3rd edn. Bailliere, Tindall and Castle Ltd. London.
- Fride, R.L. (1975). Developmental Neuropathology, Springer Verlag, New York.
- Gilman, J.P. W. (1956) Congenital Hydrocephalus in domestic animals, Cornell Vet., 46: 487.
- Greene, H.J. Leipold, H.N., Huston, K. and Dennis, S.M. (1973). Congenital defects in cattle, *Irish Vet. J.*, **27**:37.
- Jubb. K.V.F. and Kennedy, P.C. (1970). Pathology of Domestic Animals, Academic Press, NYC, Vol. I. 2nd Edn. (c.f. by Roberts S.J., 1986).
- Kalman, T.S. (1989). Congenital Malformations in Laboratory and Farm Animals, Academic Press, Inc. P. 101-111.
- McEntee, K. (1990). Reproductive Pathology of Demestic Mammals, Academic Press, 6th Edition, Williams and Wilkins, Baltimore, New York.
- Noakes, D.E., Parkinson, T.J. and England, G.C.W. (2001) Arthur's Veterinary Reproduction and Obstetrics, 8th Edn. W.B. Saunder's Company, Philadelphia.
- Roberts, S.J. (1986). Veterinary Obstetrics and Genital Diseases, 3rd Edn. Edwards Brothers, Michigah, PP. 77.
- Sane, C.R., Deshpande, B.R.; Kaikini, A.S., Velhankar, D.P., Kodagali, S.B., Luk tuke, S.N., Hukeri, V.B. and Deopukar, V.B. (1994) A Text Book of Reproduction in Farm Animals (Theriogenology), 2nd Edition, Varghese Publishing House, Bombay, P. 95.