FETAL MUMMIFICATION IN A COW -A CASE REPORT

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ABSTRACT:

A case of mummification of fetus in a crossbred cow which is a rare gestational disorder has been placed on record.

INTRODUCTION:

Fetal mummification is one of the rare gestational disorder in livestock where fetus dies in uterus and remain in situ beyond normal length of gestation (Chaudhary et al., 2005). It has been reported to occur in several domestic species (Roberts, 1986) and common in swine and feline, less common in cattle and mare but rare in canines (Arthur et al., 1989). Fetal mummification has been observed in most of the cattle breeds of world but its hereditary character is still unknown (Sane et al., 1982). In contrast Roberts (1986) reported it to be caused by single recessive autosomal gene. Conditions necessary for bovine fetal mummification to occur include maintenance of dead fetus within the uterus by persistence of corpus luteum of pregnancy or uncommonly a viable fetus. Following death of fetus in bovine the fetal fluid and placental fluids are absorbed and maternal placenta involutes and occur mostly in middle or late trimester of gestation. The present study put on record a rare case of bovine fetal mummification.

CASE HISTORY:

A pleuriparus cross bred cow (Holstein X Kankrej) aged 7 yrs was presented in the field with a history of non developed udder and reconfirmation of pregnancy. The animal was inseminated artificially 8 months back and pregnancy was confirmed after 2 months but there were no development of physical characters of pregnancy. Feeding, watering and other activities of the animal were normal. Rectal temperature was 101^{0F}.

Clinical observation:

The animal was completely normal. Vulvar mucous membrane was pinkish yellow and dry indicating completely normal texture. Vaginal examination appeared dry and had thick mucous shreds. Rectal palpation revealed hard bony mass with no fetal fluid in the uterus.

Cervix was completely closed and uterus contracted over the fetus. There were no palpable structures like curuncles and cotyledons. Apart from fetal fluid, pregnancy looked as it was of approximately 6 months. On further palpation left ovary had no palpable structures while right had well developed corpus luteum.

Treatment:

Since animal was completely normal and healthy hence Lutalyse 25 mg was given intramuscularly and owner was advised to observe at least for 72 hours. After 48 hours owner complained of slight discomfort in the animal, as animal was straining slowly and intermittently.

Vaginal examination revealed presence of dry fetus along with placenta on the vaginal passage which was extracted out with little force. Cervix was partially dilated with 4 fingers. Dirty mucous paste from vaginal passage was completely evacuated and 4 furea boluses were put intrauterine. Lixen powder 25 gm was smeared in the vaginal passage to safeguard from infection. Gentamicin 30ml intramuscular was given for 3 days to control the infection. On 11th day 5 ml Lutalyse was again injected intramuscularly to enhance uterine involution and evacuate infection from uterus if any.

The fetus and placenta were completely smeared with thick dark chocolate coloured mucus paste. Umbilicus was attached with placenta. Careful observation of the fetus observed no abnormality in any part of body. After a month animal came into oestrus with dirty flakes in mucus. It was infused with Lixen solution 50

ml and metronidazole 50 ml intrauterine. Animal again came into oestrus with clear mucous discharge after 20 days. It was inseminated on 2nd and 3rd day post oestrus and pregnancy was confirmed 45 days post insemination. Owner was advised for reconfirmation of pregnancy after every 2 months.

Discussion:

In the present study Lutalyse 25 mg was given intramuscularly to expel the fetus which was similar with the treatment applied by Tamuli et al.(2003) and Chaudhary et al. (2005) whereas Saxena et al. (2001) tried Valethamate bromide, Betamethasone and Estradiol Valerate for expulsion of fetus with variable success. In another study Phogat and Gupta (1992) delivered a mummified fetus from cross bred cow through cesarian where the cow was over 4 months of its gestation period and had shown symptoms of parturition at its normal time.

Several explanations have been propounded to explain the etiology and pathogenesis of hematic mummification but exact mechanism is still obscure. It is assumed that fetal death might have occurred due to inter placental hemorrhage and thus failure of placental function (Roberts, 1986). The present case looked similar but neither torsion of uterus nor abnormal location of umbilical cord was noticed which may postulate the death of fetus at 6 months pregnancy as Arthur et al., (1989) reported that a portion of hematic fetus show torsion or abnormal location of umbilical cord which might cause placental congestion and fetal death. In his opinion it might result from exaggeration of normal fetal mobility within or after 5 months of gestation when fetus takes its final gestational presentation or a primary placental lesion might causes hypoxia and consequent alteration in fetal mobility. Morrow (1986) explained that fetal mummification occurs when a fetus died without concomitant luteolysis and adequate cervical dilatation. Generally mummification of bovine fetus affects single fetus but occasionally involve one or both fetuses in twin pregnancies (Roberts, 1986).

In another explanation some cases appear to have genetic basis and lethal producing (Van Rensburg, 2001). In Channel Island breeds where high rate of incidence was seen and intense breeding was practiced, the cause was attributed to an autosomal recessive gene (Arthur et al., 1989). A sex linked lethal gene affecting male fetus only was suggested by Deaton et al.(1959) while Molly (1961) postulated that environmental influences might induce this condition and some alteration in uterine environment, including minor degree of anoxia might set the stage for a harmful gene normally of low penitrance to express itself and exert its undesirable effects. It might be probable that mummified fetus resulting from a mutant genotype retained beyond term because of its endocrine pathology.

REFERENCES:

Arthur,G.H., Noakes, D.E. and Pearson, H.(1989).Veterinary Reproduction and Obstetrics. 6th Edition. Bailliere Tindal London: 24-28.

Chaudhary, S., Siddiquee, G. M. and Patel, J.S.(2005). IJFV **1**(1):62-63.

Deaton,O.W. Odds, D. and Seath, D.M.(1959). J. Dairy Sci. **42**: 312.

Molly, E.T.(1961). J. Obst. Gynec. **68**:303.

Morrow D.A. (1986). Current Therapy in Theriogenology. W. B. Saunders Company, Philadelphia. pp 208-209.

Phogat,J.B. and Gupta, S.L.(1992). Indian J. Anim.Reprod. **13**:92.

Roberts, S.J. (1986). Veterinary Obstetrics and Genital Diseases (Theriogenology) 3rd Edn. Jthaca New York.

Sane,C.R., Luktuke, S. N., Deshpande, B. R., Kaikini, A. S., Velhankar, D. P., Hukeri, V.B. and Kodagali, S.B.(1982). "Reproduction in farm animals"(Theriogenology).Varghese Publishing house. Lucknow .

Saxena, A., Mishra, S.K., Chandra,R. and Tiwari, H.A.(2001). Indian Vet.J. **78**: 750-751.

Tamuli, M.K., Rajkonwar,C.K. and Borgohain, B.N. (1987). Indian Vet.J. **64**:608-609.

Van Rensburg,S.W.J.(2001). Breeding Problems and A.I. Green Wood publication, Lucknow.

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