



CASE REPORT

Canine Fetal Mummification

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ABSTRACT

This paper reports on a case of canine fetal mummification which is very rare. A Japanese Spitz aged 5 year was brought to the clinic of Mount Everest Kennel Club. Breeding history of dog was obtained from the owner. X-ray confirmed the presence of fetus. Exploratory laparotomy was done under general anesthesia. Five mummified fetuses were observed. Ovariohysterectomy and corrective surgery were performed. Bitch recovered successfully through therapeutic management after surgical approach.

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INTRODUCTION

Death of fetus and fetal fluid are reabsorbed by the uterus causing dehydration of fetal tissue and associated membranes with persistence of the corpus luteum so that the products of conception are retained within the uterus is called fetal mummification (Noakes, 1986). Fetal mummification has been reported in several species but it is more common in cattle. In canine, it is uncommon and sporadic in nature (Roberts, 2004). Fetal mummification is a sterile process due to morphological changes of the retained dead fetus after the first third of the pregnancy, in the presence of a mature fetal skin resistant to autolysis (Johnston and Raksil, 1987; Johnston *et al.*, 2001a,b; Jackson, 2004b; Grunert *et al.*, 2005; Linde-Forsberg, 2010). Fetal death occurring in the late gestation, not associated with abortion or maceration, may be followed by fetal mummification. Fetal mummification does not occur during the first half of pregnancy because embryonic or fetal death prior to the development of fetal bones usually is followed by unobserved discharge or tissue resorption (Lorenz *et al.*, 2009). In the dog, fetal mummification is a characteristic of canine herpes virus (CHV) infection (Arthur *et al.*, 1996). Canine fetal mummification is apparently similar to swine where one to three mummified fetuses are expelled with normal pups at the time of whelping (Roberts, 2004). A case report of canine fetal mummification and its successful treatment has been reported in this paper.

History and clinical observations

A 7-year-old female Japanese spitz with a history of mating 4 months before examination was brought to the clinic of Mount Everest Kennel Club. The owner reported anorexia, fever, vomiting, polydipsia, abnormal vaginal discharge since a week. The rectal temperature was 104°F and the animal was weak and lethargic. The perineum of the animal was soiled with vaginal discharge. Hematological examination revealed leukocytosis, neutrophilia and low PCV count which may be due to blood loss subsequent to uterine rupture and passage of red blood cells into the uterine lumen by diapedesis (Vorwald *et al.*, 2012). Biochemical test show hypoproteinemia and hypoalbuminaemia. Hyperproteinemia may be resulting from dehydration or increased synthesis of acute phase proteins and antibodies in response to bacterial infection and inflammation. Hypoalbuminemia was due to a lack of protein ingestion consequent to anorexia as a result of decreased liver synthesis of albumin, hemorrhages and protein loss (Vorwald *et al.*, 2012). Radiographic examination of lateral view of the abdomen indicated the presence of skeleton, suggestive of autolytic changes following death.

Surgical management

Explorative laparotomy was performed under the general anesthesia using Xylazine (1-2mg/kg BW I/M) and Ketamine (10mg/kg BW I/M). The uterine tissue was found scarred, which may have allowed an accumulation of purulent material in the uterine cavity. The membrane

was closely adhered to the fetuses. The horn of uterus was filled with serosanguinous content. The fetuses were dehydrated, surrounded by dark capsules with wet surface. Ovariohysterectomy was performed to remove the fetus and its adhered membrane.

Neosporin[®] powder was sprinkled inside the abdominal cavity. The abdominal wall was closed using 0-0 catgut suture and the skin was closed with silk. Dicrysticin[®] (1ml/5kg BW) and Meloxicam (0.2-0.3mg/kg BW) was given intramuscularly for five consecutive days post-operatively. Intravenous fluid therapy was given for 3 days. Daily dressing was done for 5 days with Wokadine[®] ointment and Neosporin[®] powder. The suture was removed on 7th post-operative day. The bitch recovered uneventfully.

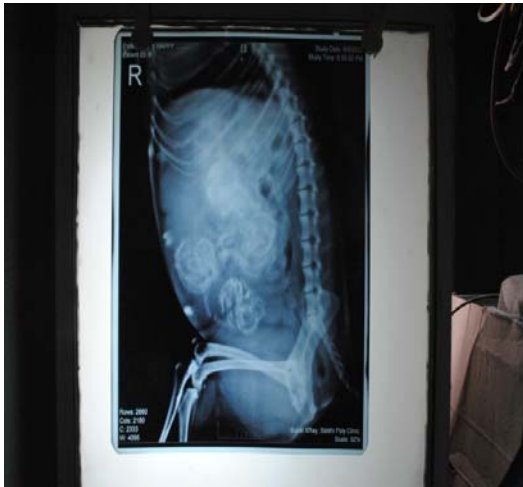


Fig. 1: X-ray of the abdominal cavity (lateral view)



Fig. 2: Mummified fetuses

DISCUSSION

The fetal mummification is common problem in polytocous and rare in montocous species (Perumal and Srivastava, 2011). In this study, all the fetuses were mummified and surrounded by dark capsules with wet surface. The placental fluids are absorbed and the fetal

membranes adhere to the dehydrated fetus, allowing the formation of a dark tissue with a wet surface without odor or secretions (Johnston and Raksil, 1987; Johnston *et al.*, 2001a; Nascimento and Santos, 2003; Jackson, 2004a; Grunert *et al.*, 2005; Kennedy and Miller, 2007). The main reason for the lack of expulsion of dead mummified fetus in present case may be primary uterine inertia which is common in canine species (Romagnoly *et al.*, 2004). Walett and Linde (1994) also reported uterine inertia as main cause of dystocia in bitches. The owner reported that the bitch did not show any clinical manifestations of approaching whelping. In elderly female dogs, due to poor abdominal muscle tone there is difficulty in producing uterine contraction in second stage of labor (Jackson, 2004b). This primary uterine atony may have been cause for the maternal dystocia (Vorwald *et al.*, 2012). It was also believed that fetuses fail to produce sufficient ACTH and cortisol to initiate the birth process (Johnston *et al.*, 2001a; Linde- Forsberg, 2010).

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