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Fetal maceration due to primary (complete) uterine inertia in a bitch-a case report

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ABSTRACT

Primary complete uterine inertia is an important cause of dystocia in dogs and may result in fetal death and subsequent fetal maceration when diagnosis and treatment are delayed. A 1.3-year-old mongrel bitch was presented with a history of mating approximately 70 days earlier, abdominal enlargement, and reddish-black vaginal discharge for five days. Ultrasonography revealed dead fetuses with degenerative changes, while radiography confirmed the presence of fetal skeletons within the uterus. Based on the clinical findings, primary uterine inertia with retained dead fetuses was suspected. Initial medical management with oxytocin and supportive therapy failed to induce fetal expulsion. Consequently, ovariohysterectomy was performed. During surgery, greenish uterine exudate and markedly autolysed fetal material with only skeletal remains were observed, confirming fetal maceration. The bitch recovered uneventfully following postoperative antibiotic, *anti*-inflammatory, and analgesic therapy. This case highlights the importance of early diagnosis and timely surgical intervention in managing complete uterine inertia complicated by fetal maceration.

Keywords: Dystocia, Fetal maceration, Primary uterine inertia, Retained fetus, Ovariohysterectomy

Introduction

Uterine inertia is defined as the lack of ability of the uterine muscles to contract sufficiently to expel the fetus right from the onset of whelping. Primary complete uterine inertia is one of the principal causes of dystocia. It is characterised by the failure of uterine muscles to expel normal-sized fetuses through birth canal which is normal, and by contraction which are either completely absent, weak or infrequent (Singh *et al.*, 2020) [1]. Foetal maceration occurs as a consequence of failure of expulsion of an aborting foetus at the late gestation, probably due to uterine inertia, failure of the genital tract to contract and dilate sufficiently or due to abnormal disposition of the dead foetus (Drost, 2007) [2]. Before foetal maceration occurs, bacterial cells invade the uterus through the dilated cervix, infect the dead foetus, and cause putrefaction and autolysis of the soft tissues; in some cases, they leave the foetal bones within the uterus (Long, 2009) [3]. The extent of putrefaction and tissue lysis depends on the time elapsed and the timing of intervention.

Materials and Methods

A 1.3-year-old mongrel bitch was presented to the Veterinary Clinical Complex (VCC), Nagpur Veterinary College, Nagpur, with a history of mating approximately 70 days earlier. The owner reported that the dog had developed abdominal enlargement along with reddish-black vaginal discharge for the past five days. Since the owner was unaware of the normal gestation period in dogs, veterinary attention was not sought initially, as the condition was considered to be a normal part of pregnancy.

On clinical examination, the rectal temperature, respiratory rate, and pulse rate were within the normal physiological range. Transabdominal ultrasonography revealed fetuses without detectable cardiac activity along with hyperechoic areas within the uterus, indicating fetal death and possible degenerative changes. Per-vaginal examination did not reveal any fetus in the birth canal. Abdominal radiography subsequently confirmed the presence of fetal skeletons within the abdomen.

Based on the history and findings of ultrasonography (Fig. No. 1) and radiography (Fig. No. 2), the case was tentatively diagnosed as primary uterine inertia associated with retained dead fetuses. As an initial therapeutic approach, medical management was attempted with oxytocin and calcium supplementation to stimulate uterine contractions and facilitate expulsion of the retained fetuses (Mahla *et al.*, 2016) [4].

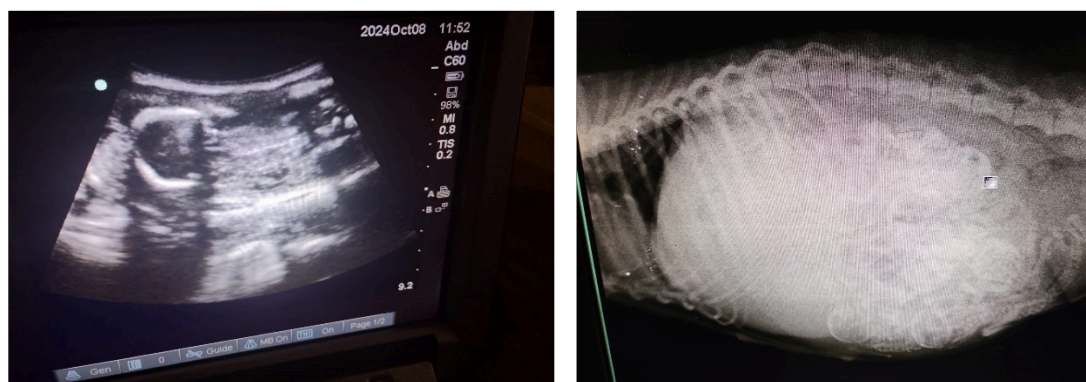


Figure 1. Ultrasonographic image of fetus Fig No.2 Radiographic image with multiple fetus

Results and Discussion

The bitch did not respond to the initial medical treatment administered on Day 1, which included Inj. oxytocin @ 10 IU intravenously, Inj. dextrose 5% @ 5 ml/kg intravenously, and Inj. Monocef @ 15 mg/kg intravenously.

As there was no response to medical management, surgical intervention was planned on Day 2. The bitch was initially stabilized with 150 ml of Ringer's lactate administered intravenously. Atropine @

0.04 mg/kg subcutaneously was given as a pre-anaesthetic, followed by sedation with ketamine @ 10 mg/kg and diazepam @ 0.5 mg/kg. An ovariohysterectomy was then performed through a mid-ventral abdominal approach. On exteriorization and incision of the uterus, the uterine horns were found to contain a greenish exudate, along with markedly autolysed fetal material. Only skeletal remains of the fetuses were present, which was suggestive of fetal maceration. (Fig No. 3 & 4) Based on the surgical findings, a confirmative diagnosis of fetal maceration associated with complete primary uterine inertia was made.

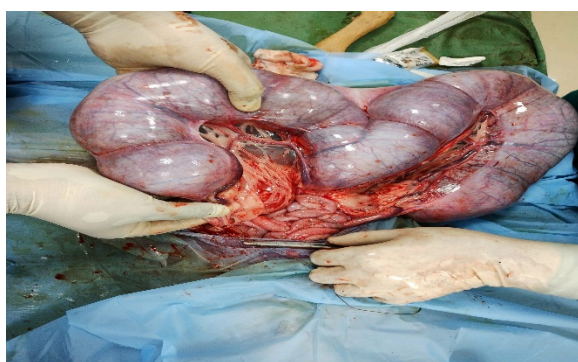


Figure 3. Gravid uterus Fig. No. 4 Macerated Fetuses

Postoperatively, the bitch was administered Inj. Toxomox @ 9 mg/kg intramuscularly, Inj. Pantop @ 0.5 mg/kg, Inj. Avilin @ 1 ml, Inj. Tramadol @ 2 mg/kg, and 150 ml of DNS intravenously. Antibiotic, anti-inflammatory, and analgesic therapy was continued for seven days. The bitch recovered uneventfully, and the skin sutures were removed after 14 days.

Fetal maceration in the uterus is a relatively uncommon condition in canine practice. Failure of fetal expulsion, which may occur due to uterine inertia or intrauterine infection, can lead to fetal death followed by emphysema or maceration (Johnston *et al.*, 2001) [5]. In cases of complete primary uterine inertia, where there may be little or no obvious signs of active whelping, determining the stage of parturition can be challenging. Delayed recognition and treatment may result in fetal death and subsequent maceration, which can further compromise the health and survival of the dam (Verma *et al.*, 2021) [6].

In the present case, timely presentation along with appropriate diagnostic evaluation enabled early identification of the condition and allowed surgical intervention through ovariohysterectomy. Ovariohysterectomy has also been reported as an effective treatment option in similar cases (Bodh *et al.*, 2014; Sagar *et al.*, 2017) [7, 8].

Conclusion

Primary uterine inertia is an important cause of dystocia in dogs and, when left untreated, may result in fetal death, fetal maceration, uterine complications, and potentially life-threatening consequences for the

dam. In the present case, medical management with oxytocin failed to induce effective uterine contractions, and surgical intervention was subsequently performed. Ovariohysterectomy successfully resolved the condition and ensured the survival of the bitch. Therefore, in cases of complete primary uterine inertia, particularly when retained or dead fetuses are present, surgical treatment should be considered as a primary treatment option rather than medical management, as timely surgical intervention can prevent further complications and improve the chances of maternal survival.

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