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Evaluation of treatment protocols for complete primary uterine inertia in female dogs

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Abstract

The common cause of maternal dystocia is uterine inertia and the most common form of uterine inertia in bitches is primary uterine inertia. Studies were carried out on 40 clinical cases of dystocia in female dogs, diagnosed to be due to complete primary uterine inertia, when presented to the obstetrical clinics in Veterinary College Hebbal, Bengaluru. Many of the medical protocols used for treatment of primary uterine inertia have centred on intravenous infusion of oxytocin, glucose and calcium, either alone or in combination. This study was conducted to evaluate the efficacy of these protocols on the pattern of uterine contraction in the cases of primary uterine inertia in female dogs. Uterine tocodynamometric studies revealed that complete primary uterine inertia was associated with weak and infrequent uterine contractions therapeutic intervention using oxytocin, oxytocin and dextrose or calcium and oxytocin failed to significantly augment the uterine contractions. It was concluded that cases of complete primary uterine inertia are best treated using the surgical option.

Keywords: Canine dystocia, treatment protocols, uterine inertia, oxytocin, calcium, glucose

1. Introduction

Dystocia has conveniently been described as being maternal or foetal in origin and there is overwhelming evidence that in the bitch, maternal dystocia is encountered more frequently [1, 2]. There is also evidence that the most common cause of maternal dystocia is uterine inertia, representing 40 per cent [1] to 75.3 per cent [2] of all dystocia attributed to the dam. The most common form of uterine inertia in bitches is primary uterine inertia, which has been further classified as complete or partial [3]. In complete primary uterine inertia, the bitch does not start labour. In partial primary uterine inertia, the bitch starts to deliver her puppies, but the labour ends prematurely, despite the presence of a patent birth canal [4].

The cause of primary uterine inertia is unknown, although a disturbance in the sequence of hormonal events required for normal labour may represent one possible cause [5]. Labour is the physiological process by which a foetus is expelled from the uterus through the vagina and is characterized as regular uterine contractions accompanied by cervical effacement and dilatation [6] and it is commonly believed that uterine inertia is associated with complete absence, weak or asynchronous uterine contractions.

The suggested causes for primary uterine inertia include a deficiency of Oxytocin [5], Serum Calcium [1, 7] and Blood Glucose [7, 8]. Therefore, many of the medical protocols used for treatment of primary uterine inertia have centered on intravenous infusion of oxytocin, glucose and calcium, either alone or in combination [4, 9, 10]. The efficacy of many medical protocols commonly used by the veterinarians to relieve dystocia due to uterine inertia has not been subjected to a thorough scrutiny.

Therefore, the present study was conducted to evaluate the efficacy of certain treatment protocols for augmenting uterine contractions in confirmed cases of primary uterine inertia. Specifically, this study was undertaken to determine the effect of intravenous supplementation of oxytocin, calcium and glucose on the pattern of uterine contraction in cases primary uterine inertia female dogs.

2. Materials and Methods

Studies were carried out on 40 clinical cases of dystocia in female dogs, diagnosed to be due to complete primary uterine inertia, when presented to the obstetrical clinics in Department of Veterinary Gynaecology and Obstetrics, Veterinary College Hebbal, Bengaluru. A diagnosis that dystocia due to Complete Primary Uterine Inertia was made, if the animal presented had a history and ultrasonic evidence of completion of pregnancy term, complete absence or the

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presence of very weak signs of first stage of labour, and the presence of greenish or blackish-green lochia on the perineum, vulva or vestibule for at least two hours. In some cases when the history was suggestive of complete primary uterine inertia, but there was no evidence of lochia in the perineum, the anterior vagina was further examined using a rigid vaginoscope to identify the presence of discharges or cervical opening [11]. The uterine contraction patterns were recorded in these cases using a tocodynamometer (SONICAID TEAM).

Medical management of cases of complete primary uterine inertia under uterine tocodynamometric monitoring was carried out using oxytocin, dextrose and oxytocin or calcium and oxytocin. Each treatment was evaluated on a group of 10 animals to determine the most effective treatment for the relief of complete primary uterine inertia.

2.1 Efficacy of oxytocin

Ten animals diagnosed as cases of dystocia due to complete primary uterine inertia were injected with a single injection of one unit of oxytocin (Syntocinon®, Novartis India Limited) intravenously. Following intravenous injection of oxytocin, the animal was given thirty minutes time to deliver a puppy. If a puppy was not delivered within thirty minutes, the treatment was considered a failure and no further injection of oxytocin was given. Uterine tocodynamometric studies were carried out up to 30 minutes after oxytocin injection.

2.2 Efficacy of dextrose and oxytocin

Ten animals with complete primary uterine inertia were infused with 25% dextrose at a dose of 1 g/kg body weight. Following completion of dextrose infusion, the animals received 1 unit oxytocin injection intravenously. If a puppy was not delivered within thirty minutes after oxytocin infusion, the treatment was considered a failure and no further injection of oxytocin was given. Uterine tocodynamometric studies were carried out up to 30 minutes after oxytocin injection.

2.3 Efficacy of calcium and oxytocin

Ten cases of complete primary uterine inertia were infused with a solution containing 50 mg/ml of calcium gluconate and 87.5 mg/ml of calcium lactobionate equivalent to 9 mg of elemental calcium (Calcium-Sandoz®, Novartis India Limited). Calcium was slowly infused intravenously over a period of ten minutes at the rate of 1 ml/kg body weight, not exceeding 20 ml. This was immediately followed by intravenous infusion of oxytocin at the rate of 1 unit/kg body weight. If a puppy was not delivered within thirty minutes after oxytocin infusion, the treatment was considered a failure and no further injection of oxytocin was given. Uterine tocodynamometric studies were carried out up to 30 minutes after oxytocin injection.

3. Results and Discussion:

3.1 Efficacy of oxytocin

Various workers have frequently employed oxytocin either as posterior pituitary extract or as a synthetic form to augment uterine contractions. In such cases, oxytocin has been commonly injected in doses of 5- 20 units subcutaneously or intramuscularly [11, 12]; 3- 20 units intramuscular [13], 5-15 units intramuscular [9] or 0.5- 1 unit per lb body weight intramuscularly [10]. However, Davidson [14] suggested that doses as low as 0.5- 2 units are more effective in increasing

the frequency and quality of contraction and that higher doses may bring about sustained contractions which may endanger the life of the foetus. In the present study therefore, oxytocin was used at a very low dose (1 unit).

In the present study all the animals with complete primary uterine inertia failed to respond to an intravenous infusion of oxytocin carried out in an effort to augment uterine contractions (Table 1). Uterine tocodynamometric studies revealed that infusion of oxytocin in a case of complete primary uterine inertia did result in uterine contractions, but the strength of uterine contraction in any of the animals did not exceed more than 50 per cent. Further, only one or two uterine contractions lasting less than a minute was identified following oxytocin infusion and the contractions returned to base line levels thereafter. It has been recommended by Gropetti [15] that physiological labour is possible when there are 12 contractions per hour, with each one of 2-5 minutes duration.

The absence of clearly detectable uterine contractions following oxytocin injection explained why all cases of complete primary uterine inertia did not respond to oxytocin therapy in the studies of Vibha [16]. It is possible that complete primary uterine inertia is associated with inherent defective oxytocin receptors in the uterus.

3.2 Efficacy of oxytocin and dextrose infusion

There are several reports in the literature suggesting hypoglycaemia as a cause of uterine inertia in bitches [7, 8, 12, 17, 18, 19] and this forms the basis of using dextrose infusions as a line of medical treatment in cases of uterine inertia [20, 21].

In the present study, animals diagnosed with uterine inertia were first infused with dextrose, followed by intravenous infusion of oxytocin at the end of dextrose infusion. Dextrose was infused primarily with the objective of correcting hypoglycaemia if any and as a source of energy, and oxytocin to augment uterine contractions. It was observed in the present study that this line of treatment did not evoke any response in animals with complete uterine inertia (Table 1). Further, uterine tocodynamometric studies also revealed that infusion of dextrose prior to oxytocin infusion also did not augment the uterine contractions. Further, uterine tocodynamometric studies also revealed that infusion of dextrose prior to oxytocin infusion also did not augment the uterine contractions.

3.3 Efficacy of calcium and oxytocin infusion

There are a number of reports which have documented successful treatment of uterine inertia with intravenous infusion of calcium [7, 17, 22, 23, 24, 25]. Generally, the administration of calcium increases the strength of myometrial activity and Davidson [26] stated that when ineffective or weak uterine contraction are detected, calcium Gluconate as 10% solution should be given subcutaneously at the rate of 1ml/ 4.5 kg body weight. In the present study therefore, animals with complete primary uterine inertia were first treated with calcium in an effort to strengthen the uterine contractions followed by oxytocin to increase the frequency of uterine contractions.

In the present study, infusion of calcium and oxytocin failed to relieve uterine inertia in any of the cases where the inertia was diagnosed as complete (Table 1). Johnston [27] concluded that there was no indication that serum calcium deficiency was a cause of uterine inertia. Bergstrom [5] also opined that in many cases of uterine inertia, calcium was not needed to initiate the labour and that oxytocin alone may be sufficient.

4. Conclusion

Uterine tocodynamometric studies revealed that complete primary uterine inertia was associated with weak and infrequent uterine contractions. Therapeutic intervention using oxytocin, oxytocin and dextrose or calcium and oxytocin failed to significantly augment the uterine contractions. It was concluded that cases of complete primary uterine inertia are best treated using the surgical option.

The present study did not try to elaborate the various causes of uterine inertia. In a clinical setup, it is perhaps also extremely difficult to pin point any one single cause as responsible for uterine inertia. This perhaps explains the reasons as to why a majority of the cases of complete primary uterine inertia failed to respond to medical protocol. The medical treatment aimed at augmenting uterine contractions is unlikely to be successful if there is a hereditary weakness of

the uterine musculature. Similarly, complete uterine inertia due to toxic degeneration of uterine muscle or hydro allantois is also unlikely to respond to medical approaches. Therefore, a poor response obtained to medical therapy in cases of complete primary uterine inertia was on the expected lines.

Further, when obstetrician decides to try medical therapy in cases of complete primary uterine inertia, he should keep in mind that a high percentage of animals may have to be subjected for caesarean section. Further, in trying medical therapy, an obstetrician may be losing valuable time in terms of life of the puppy as oxytocin injections may induce enough contractions of the uterus to cause separation of the foetal membranes, but not the expulsion of the foetus, it therefore seems reasonable to suggest that caesarean section should be the first line of treatment for all cases of complete uterine inertia to maximize the foetal survival rate.

Table 1: Efficacy of different treatment protocols in cases of complete primary uterine inertia

S. No	Treatment Protocols	No. of cases treated	No. of animals responding to treatment	% efficacy
1	Oxytocin Inj	10	00	00
2	Dextrose and Oxytocin Infusion	10	00	00
3	Calcium and Oxytocin Infusion	10	00	00

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