THERAPEUTIC MANAGEMENT OF PRIMARY UTERINE INERTIA IN BITCHES

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The present study therapeutic management of uterine inertia in bitches of Nagpur city was conducted at Veterinary College, Nagpur. Prospectively a complete clinical evaluation was done in total 24 cases of primary uterine inertia by randomly dividing into two groups. Group I was treated with 5% dextrose, 10% calcium gluconate by intravenous route followed by oxytocin intramuscularly, while Group II was treated with 5% dextrose only by I/V route. Blood samples were collected aseptically to estimate serum calcium, glucose, phosphorus, sodium, potassium, and magnesium before treatment and after whelping. Successful induction of whelping was observed in 58.33 and 25.00 per cent treated bitches from group I and group II, respectively. In the non responded bitches caesarean section was performed.

Key words: Bitches, Blood biochemical parameters, Primary uterine inertia, Whelping.

Dystocia occurs in about 5% of all parturitions in dogs (Linde-Forsberg and Eneroth, 2000). The cause may be either maternal or fetal. The most common form of maternal dystocia in bitches is primary inertia which can be classified as complete or partial (Van der Weijden and Taverne, 1994). Primary uterine inertia is the most common cause (75%) dystocia in the bitches (Darvelid and Linde-Forsberg, 1994). The condition is characterized by the failure of uterine muscle to expel normal sized fetuses through birth canal which is normal, except perhaps for an incompletely dilated cervix and characterized by contraction which are either completely absent, weak or infrequent. In primary uterine inertia bitches fails to contract the abdominal muscle but the cervix dilates and the placenta can’t detach from the uterine wall.

The cause of uterine primary inertia is not clearly known, although a disturbance in the sequence of hormonal events required for normal labour may represent one possible cause (Bergstrom et al., 2006). The suggested causes for primary uterine inertia include deficiency of oxytocin (Bergstrom et al., 2006) serum calcium (Gaudet, 1985) and blood glucose (Linde-Forsberg and Eneroth, 2000). Therefore, many medical protocols used for treatment of primary uterine inertia have centered on intravenous infusion of Oxytocin, glu-
cose and calcium either alone or in combination (Bergstrom et al., 2006). This study focuses on administration of calcium, glucose and oxytocin in different regimen for treatment of primary uterine inertia in bitches.

Materials and Methods

In a prospective study at TVCC, Veterinary College, Nagpur, for a period of 3 years from April 2013 to March 2016; a total 24 bitches suffering from primary partial uterine inertia were randomly divided into two groups having 12 bitches in each. Group I was treated with 10 per cent calcium gluconate @ 0.5 ml/kg bwt I/V, Dextrose 5 % I/V and oxytocin @ 2 IU/kg bwt I/M. While group II was treated with Dextrose 5 % infusion only. A diagnosis of dystocia due to partial primary uterine inertia was made, if the bitches started to deliver puppies, but the labour ends prematurely, despite the presence of a patent birth canal with a history and ultrasonic evidence of completion of pregnancy term, presence of signs of first stage of labour along with the presence of greenish or blackish-green lochia on the perineum, vulva or vestibule and absence of response to feathering stimulation of vagina.

Blood samples were collected aseptically before treatment and after completion of whelping in both the treatment groups to know and record changes in serum calcium, glucose, phosphorus, sodium, potassium and magnesium. Caesarean section was done immediately in those cases which failed to deliver fetus with

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in 30 min of oxytocin therapy in group I and absence of contraction to feathering response after completion of dextrose infusion in Group II. The collected data was statistically analyzed using Student’s t-test.

**Results and Discussion**

**Blood biochemical parameters in uterine inertia**

Serum sodium concentration levels before the start of the treatment was 151.17±2.84 and 150.69±3.04 mEq/L in group I and group II, respectively. While the respective values after whelping were 149.86±3.07 and 149.96±3.05 mg/dl (Table-1). Serum sodium values statistically were reduced significantly after whelping in group I and II, but were still within the normal physiological limit.

Serum glucose concentration levels before the start of the treatment was 69.71±5.0 and 63.35±5.42 mg/dl in group I and group II, respectively. While the respective values after whelping were 96.84±8.93 and 97.73±9.85 mg/dl (Table-1). All the values of glucose increased significantly after whelping in between the two treatment groups.

Serum calcium concentration levels before the start of the treatment was 8.51±0.28 and 9.44±0.33 mg/dl in group I and group II, respectively. While the respective values after treatment were 9.06±0.26 and 9.00±0.30 mg/dl (Table-1). Significant increase in values was found in group I.

<table>
<thead>
<tr>
<th>Biochemical Parameters</th>
<th>Treatments</th>
<th>Group –I</th>
<th>Group –II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>Before</td>
<td>8.51±0.28</td>
<td>9.44±0.33</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>9.06±0.26*</td>
<td>9.00±0.30**</td>
</tr>
<tr>
<td>Glucose</td>
<td>Before</td>
<td>69.71±5.07</td>
<td>63.35±5.42</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>96.84±8.93**</td>
<td>97.73±9.85**</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>Before</td>
<td>5.06±0.43</td>
<td>4.48±0.19</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>4.82±0.41</td>
<td>4.34±0.22</td>
</tr>
<tr>
<td>Sodium</td>
<td>Before</td>
<td>151.17±2.84</td>
<td>150.69±3.04</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>149.86±3.07*</td>
<td>149.96±3.05*</td>
</tr>
<tr>
<td>Potassium</td>
<td>Before</td>
<td>4.61±0.22</td>
<td>4.39±0.28</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>4.40±0.25</td>
<td>4.12±0.24</td>
</tr>
<tr>
<td>Magnesium</td>
<td>Before</td>
<td>2.14±0.01</td>
<td>2.17±0.04</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>2.13±0.01</td>
<td>2.12±0.01</td>
</tr>
</tbody>
</table>

Note: *indicate at 5% level of significance; ** indicate at 1% and 5% level of significance

In between the two groups there was no significant difference in the values of serum calcium, phosphorus, sodium, potassium, magnesium concentration in bitches diagnosed as cases of primary uterine inertia (Table-1). These serum biochemical values were within normal reference range with no signs of abnormality. It is unlikely that the serum concentration of the electrolytes is a cause to uterine inertia in the bitches. But during the present study serum glucose level was low in all bitches having primary uterine inertia while increased significantly after treatment in both the groups. Similar findings are reported by Linde Forsberg and Eneroth (2000) who reported hypoglycaemia as a cause of uterine inertia, especially in canine dystocia. Where as Bergström et al. (2006) reported hyperglycemia secondary to high cortical concentrations during dystocia in the bitches.

While serum calcium level was towards lower side in all bitches having primary uterine inertia which increased significantly after treatment in group I. They were all in the physiological range. Despite serum calcium often being within the normal reference range, resolution of dystocia caused by primary uterine inertia with administration of calcium indicates its potential role in uterine inertia as also reported by Hollinshead et al. (2010). This suggests that the benefit of calcium administration is at cellular or sub cellular level.
Phosphorus is the second most important electrolyte regulating myometrial contraction -s after calcium. In the present study, none of the 24 bitches with primary uterine inertia had any evidence of hypophosphatemia. Likewise Serum magnesium levels were also within the normal physiological range in all bitches with primary uterine inertia.

**Efficacy of treatment protocols in uterine inertia**

The efficacy of intravenous infusion of calcium gluconate @ 0.5 ml / kg bwt I/V, Dextrose 5 % I/V and oxytocin @ 2 IU / kg b.w. I/M dextrose in 12 bitches with primary uterine inertia from group I and response was observed in 7 (58.33 %) bitches. While in group II bitches infused with dextrose 5 percent only the response to therapy was seen in only 3 of 12 (25.00%) bitches. During the present study, caesarean section was done in all the non responding cases from both the groups having primary uterine inertia.

The success rate obtained during the present study could be due to the inclusion of dextrose and calcium as a part of medical management of primary uterine inertia. It can be seen that bitches with primary uterine inertia was having hypoglycemia and hypocalcemia. Thus, it was seen that the treatment protocol in group I had better outcome i.e. treatment with intravenous calcium with 5% Dextrose and Oxytocin. Buckner (1979) has also recorded that the hypoglycemia has a role in causing uterine inertia in bitches. The successful correction of uterine inertia due to hypoglycemia by intravenous administration of glucose solution has also been reported by Greiner (1974) and Jones and Joshua (1988).

In the hypocalcemic bitches, calcium infusion is not always effective. One reason for this may be that it is the intracellular calcium which is involved in myometrial contractions and the calcium ions in serum have to pass into the cell via channels. Both the transport of extracellular calcium into the myometrial cell as well as the release of calcium from the sarcoplasmic reticulum is regulated by hormones as also reported by Sanborn (2000) but where ever the response to calcium therapy is observed is because of the action of calcium at cellular and sub cellular level.

**References**


