Postpartum complications in large domestic animals

Prof G N PUROHIT

Head, Department of Veterinary Gynecology and Obstetrics, College of Veterinary and Animal Science, Rajasthan University of Veterinary and Animal Sciences, Bikaner, Rajasthan, India
Post partum complications

Retention of after birth
Post partum bleeding
Post partum prolapse
Paralysis
Post partum metritis
Lacerations of the birth canal
Metabolic disorders Milk fever, Eclampsia
Retention of after birth (Retained placenta)

A placenta should fall within 8-12 hours in cattle, buffaloes, sheep and goat and within 2 hrs in the mare if it is not expelled within this time it is considered retained. The retention is considered primary if there is lack of detachment from maternal caruncles in ruminants and secondary if there is mechanical difficulty in expelling the already detached placenta.

Incidence: from 2-50% more in summer and more in selenium and vitamin E deficient areas.

Mechanism of placental detachment in cows

↑ collagenase during postpartum causes massive breakdown of collagen during uterine involution. ↑ serotonin in fetal blood during late gestation cause ↑ collagenase by uterine cells. This also decreases blood to placenta. Uterine distension also causes separation. Increases in PG metabolites favor separation.
Etiology of RAB

Premature parturition
Failure of cotyledon caruncle detaching mechanisms
Hormonal imbalances ↑ progesterone
Trauma causes ↑ heparin which ↓ proteolysis
Low calcium and deficiency of Se and Vitamin E
Metabolic imbalances
Post partum disease Normal mechanism of placental separation
<table>
<thead>
<tr>
<th>Factor</th>
<th>% Retained Placenta</th>
<th>Relative Risk*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Obstetric</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abortion</td>
<td>62</td>
<td>10.3</td>
</tr>
<tr>
<td>Multiple birth</td>
<td>37</td>
<td>8.3</td>
</tr>
<tr>
<td>Two previous retentions</td>
<td>25</td>
<td>6.0</td>
</tr>
<tr>
<td>Cesarean delivery in hospital</td>
<td>62</td>
<td>6.0</td>
</tr>
<tr>
<td>Stillbirth</td>
<td>19</td>
<td>4.4</td>
</tr>
<tr>
<td>Fetotomy</td>
<td>26</td>
<td>4.1</td>
</tr>
<tr>
<td>Advanced age of cow</td>
<td>10</td>
<td>3.3</td>
</tr>
<tr>
<td>Cesarean delivery</td>
<td>26</td>
<td>3.2</td>
</tr>
<tr>
<td>One previous retention</td>
<td>12</td>
<td>3.0</td>
</tr>
<tr>
<td>Difficult calving</td>
<td>13</td>
<td>2.1</td>
</tr>
<tr>
<td><strong>Physiologic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Short gestation plus low calf weight</td>
<td>12</td>
<td>3.0</td>
</tr>
<tr>
<td>Summer calvings</td>
<td>11</td>
<td>1.6</td>
</tr>
<tr>
<td>Sex of calf (male)</td>
<td>12</td>
<td>1.05</td>
</tr>
<tr>
<td><strong>Hormone Imbalance</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prepartum ovariectomy</td>
<td>100</td>
<td>15.1</td>
</tr>
<tr>
<td>Prepartum corpus luteum ablation</td>
<td>100</td>
<td>15.1</td>
</tr>
<tr>
<td>Abnormal (high/low) prepartum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Progesterone</td>
<td>90</td>
<td>13.6</td>
</tr>
<tr>
<td>Estrogens</td>
<td>34</td>
<td>5.1</td>
</tr>
<tr>
<td><strong>Induced delivery</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prostaglandin F₂α</td>
<td>80</td>
<td>12.1</td>
</tr>
<tr>
<td>Dexamethasone + prostaglandin F₂α</td>
<td>79</td>
<td>12.0</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>67</td>
<td>10.1</td>
</tr>
<tr>
<td>Dexamethasone + estrogens</td>
<td>67</td>
<td>10.1</td>
</tr>
<tr>
<td>Dexamethasone + relaxin</td>
<td>15</td>
<td>2.2</td>
</tr>
</tbody>
</table>
Normal mechanism of placental separation

Fetal Cortisol Increase

Change from Progesterone to Estrogen production

Increased Estrogen

Increased PGF2α

Lysis of CL

Increased Oxytocin Receptors

Relaxin Secretion

Increased Myometrial Contraction

Progesterone Decline

Increased Collagenase Activity

Breakdown of Fetal Cotyledon-Maternal Caruncle interface

Delivery of Detached Placenta

Development/Recognition of fetal MHC Class 1 molecules

Maturation of Fetal MAO

Maternal Immune Response

Leukocytes and Cytokines

Serotonin Levels

High

Low

Corpus Luteum
PGF2α- Prostaglandin F2 Alpha
MHC- Major Histocompatibility Complex
MAO- Monamine Oxidase Enzyme System
Cotyledon caruncle interface
Pathophysiology of retained fetal membranes in cows

Parturition ← Prepartum disturbed onset of cotyledon proteolysis

Partal trauma > mast cells

Failure of cotyledon proteolysis

RFM

Metabolically active RFM continues to grow under metabolic stress

Bacterial colonization Release of inflammatory molecules RFM Mass

Toxins Uterine effects Systemic effects

> Immunosuppressors Hypothalamus &

> permeability neuro endocrine systems

> lysozymes

> chemotaxis

↑ endometrial damage

Metritis

↓ Appetite & milk delayed involution ← ← ← ← ← ← ← ← ← ↓

↓ Fertility
**Therapies**

**Manual removal**

**Uterine motility stimulants Oxytocin**

Collagenase injections of 200,000 U of bacterial collagenase in 1,000 ml of physiologic saline solution via umbilical arteries (1 or 2) between 24 and 72 hours of retention caused release of retained fetal membranes

**Antibiotics**

Ozone treatments 30 ml ozone flush (Riger Spray; Novagen, Parendzana, Italy) infused directly intrauterine, coupled with 4500 mg of cephalexin

Electrical stimulation of uterine contractions by stimulator (Elegant) (Russia) that works on 9 V current and has a thread to remove it. It sends 7 electrical impulses of 0.1 sec
Policresulenum 2.16 g (Polycondensate of m-cresol-sulfonic acid and formaldehyde at the mass ratio of 14:1)
Electrical stimulator for Retained placenta

The electro-stimulator for uterine contractions in cows "Elegant". General view.
Effects of RFM

- Decreased appetite, delayed involution, Delayed post-partum estrus, increased incidence of metritis, increased services per conception.
Retained placenta in a mare, sheep and goat.
Retained placenta in buffalo
Retained placenta mare:

More in aged draft mares and in dystocia and this can lead to laminitis because of release of vasoactive components. The peripheral blood circulation increases post partum because of decrease in uterine size and increased uterine tone. The fibrogen degradation products of uterus are thus carried to peripheral circulation thus causing laminitis.

Therapy: 30-80 IU oxytocin in saline IV.
- Infusion of normal saline intrauterine
- Manual removal
- Cold water baths, antihistaminic and antibiotics
  if laminitis develop
• One well-known technique is the “Burns Technique” involving the introduction of large volumes of lavage fluid into the allantoic cavity followed by the manual occlusion of its entrance for 10–15 min.

• Laminitis prevention includes ice boots, NSAIDs, and pentoxifylline administration 4.4 mg/kg, orally every 8 hours but avoid concurrent use of NSAIDs.
Uterine lavage
Vaginal ruptures can be located by careful palpation. Fluid accumulations can be seen lateral to the vulva subsequent to vaginal ruptures during or after parturition. Surgical repair is possible.
Post partum hemorrhage
Hemorrhage from a uterine artery is common in older mares and is a cause of death in a significant number of aged broodmares.
Mechanical trauma to the blood vessels of the pelvic region as a result of parturition, usually following dystocia or assisted calving.
Accidental rupture of cotyledons during manual removal of placenta.
Subsequent to torsion correction.
Vulval hematoma common in gilts.
Rupture of broad ligament, peritoneal hemorrhage common in mares with a copper deficiency and symptoms of vaginal pain, dysuria, colic and difficult defecation are seen.
• **Signs**: can range from scant venous ooze through to profuse arterial haemorrhage.

• **Diagnosis**: diagnosis of the location and severity of the laceration and haemorrhage is important. It may be possible to visually inspect the outer walls of the vagina and vulva, however generally manual examination of the uterus, cervix and vaginal walls is necessary to identify any tears or bleeding vessels.

• **Treatment**: where possible, by clamping and ligating affected vessels. Occasionally packing the vagina in order to control bleeding may be necessary. Blood transfusion is indicated in some severe cases.

  Cold packs are suggested.

NSS + 10 cc formalin, oxytocin, blood transfusion, ergonovine maleate if bleeding occurs after several days of parturition.
Postpartum hemorrhage, vulvar abscess and vulvar laceration in buffalo

Vulvar abscess can occur a few days after parturition and can be treated by the administration of antibiotics or by surgical excision.
Vulvar Edema Can occur post partum

Vulvar edema is the result of dystocia handling or a consequence of truss application for prevention of a vaginal prolapse. The edema usually resolves spontaneously, however, cold fomentation, diuretics and anti-inflammatory drugs are suggested for 3-5 days if the edema is extensive.
Hematomas of the birth canal or vulva are rare in large animas. They are usually left untreated with overall supplementation of anti-inflammatory and analgesic drugs. These local injuries are absorbed generally within 5 to 10 days. However, if too large and cumbersome, they can be incised evacuating clots and suturing the site.
Post partum hemorrhage in mares

Common complications included fever, leucopenia, retained fetal membranes, increased digital pulses, thrombophlebitis, and cardiac arrhythmias.

Treatment should be aimed at restoring cardiovascular volume, enhancing coagulation, controlling pain, and reducing the effects of endotoxemia. Haemostatic agent of choice is aminocaproic acid (40 mg/kg or 20 g/500 kg) diluted in 1 l of isotonic saline and administered through the IV catheter.

Formalin: The current recommended dose is 30–150 ml of 10% buffered formalin in 1 l of isotonic fluids.

Administration of hypertonic saline (3–5 ml/kg or 2 l/500 kg) is suggested along with corticosteroids.
Cervical and vaginal lacerations
Common in cow, mare and goat and repaired by suture and or surgery at a later time.

Perineal lacerations
Common in mares the term *Gill flirter* is used when the rectum and vulva become continuous
Surgeries are suggested

Prolapse of bladder
Common in mares as the urethra is large and the birth process is violent. The prolapsed bladder is replaced under epidural anesthesia.
Rupture of uterus

Common in cow, goat and ewe rarely in the mare. In the goat uterine rupture may occur due to fight or ring womb

- Uterine rupture is a complication that may be associated with fetotomy, excessive manipulation during a dystocia, fetal malposition, uterine torsion, uterine lavage, or with a seemingly normal delivery.

- Complications that may be associated with uterine rupture include visceral herniation, peritonitis, haemorrhage, shock, and death.
If a full-thickness tear or rupture has occurred, there will likely be contamination of the abdominal fluid and an increase in white blood cell counts. Abdominocentesis may be of benefit for a diagnosis and is indicated in any mare that has abdominal pain after foaling, especially after dystocia or fetotomy.
Conservative management in mares includes stall rest in a quiet, dark area with minimal disturbance. It is often best to leave the foal in the stall to keep the mare as quiet as possible. Sedatives should be used with caution (especially acetylpromazine) because they may exacerbate hypovolemic shock.

- Administration of oxytocin and shock management is suggested.
- Emergency laparotomy may be performed in some cases
- Prognosis is poor
Damage to the lumbosacral plexus

Gluteal paralysis is reported in mare.

There is difficulty in getting up and atrophy of gluteal muscles. Animals should be kept on slings.
Obturator paralysis

Obturator paralysis is common in hiplock condition. There is paralysis of adductor thigh muscles and the limbs become stiff. Nervine tonics and slings are suggested.
Rupture of gastrocnemius muscle

There is inability to support weight and the gastrocnemius tendon is flaccid. The condition is more in selenium deficient areas.
Sciatic/Peroneal nerve paralysis

Peroneal paralysis Cows with dystocia and milk fever are commonly affected. There is knuckling of the fetlock and dropping of the hock joint. Bandage over the affected part and nervine tonics are suggested.
Vulvar Hematoma in sows

• This is a condition where shortly after farrowing blood vessels inside the vulva rupture, due to stretching, pressure or trauma to the tissues. The vulva fills with blood. When this occurs the tissues become very fragile and if they are crushed the vulva splits with severe hemorrhage. Vulval hematoma can also arise where a gilt has to be assisted at farrowing and damage occurs from a large arm.

• Clinical signs

• The vulva becomes swollen and very dark blue. If it ruptures it may bleed continuously. Blood clotting is poor and the animal becomes anemic and ultimately bleeds to death.
Treatment

- The animal should be sedated and local anesthetic is injected around the tissues nearest the body of the sow just forward of the bleeding area. Three methods are then used for control:
  - A piece of band or bandage is placed between the lips of the vulva and behind the bleeding tissues. It is then tightened to produce a tourniquet. This should be left for 24 hours.
  - If this does not stop the bleeding then a series of mattress sutures should be passed through the vulva and tied to the exterior.
  - If the hemorrhage still does not stop, the hematoma must be opened by a veterinarian who will use a pair of artery forceps to clamp the ruptured blood vessels and tie them off.
Postpartum Dysgalactia Syndrome in Sows

- Postpartum dysgalactia syndrome (PPDS) is a primary cause of neonatal problems (eg, diarrhea, crushing, runting, inanition, poor growth) but is challenging to characterize because of its multiple manifestations and the difficulty in making an etiologic diagnosis.

- Evidence suggests that lipopolysaccharide (LPS) endotoxins, a portion of the cell wall of all gram-negative bacteria, play a central role. Bacterial endotoxins can be absorbed from the uterus (eg, endometritis or metritis), mammary glands (eg, acute multiglandular mastitis), or gut (eg, constipation as a consequence of feeding finely ground feed to sows can result in bacterial overgrowth and subsequent absorption of endotoxins from the intestines) and lead to endotoxemia.
MMA complex is essentially a subtype of PPDS, probably the most severe clinically but also the least common.

• LPS endotoxins exert their effects even before farrowing and act with the intervention of the innate immune system (macrophage activation). These changes adversely affect production and secretion of colostrum and milk.

• PPDS is seen almost exclusively within the first 3 days after farrowing.

• Piglet losses are due to emaciation or diarrhea (or both), as a consequence of poor nutrition during the first few days postpartum. Diagnosis is difficult and based on clinical signs.
Treatment and Control:

- Systemic or local therapeutic intervention (antibiotics, NSAIDs) can sometimes be helpful but only on a short-term basis. Flunixin meglumine may help to counteract the effects of endotoxins. Antimicrobial treatment is usually prescribed before susceptibility can be tested. A broad-spectrum antibiotic is therefore recommended. However, if antibiotics are used long-term, a dependence on them for puerperal fevers, acute mastitis, vaginitis, endometritis, or neonatal diarrhea can develop rapidly and lead to multiresistant bacterial infections. Oxytocin or prostaglandins (or both) can be useful in cases of prolonged farrowing or postpartum endometritis. By far the most effective method is to cross-foster the piglets from affected to healthy sows, as long as the health status of the litters are equivalent. Oxytocin (5–10 U/sow) is occasionally effective in reestablishing lactation if used 4 or 5 times at 2- to 3-hr intervals. In herds in which PPDS is a significant problem, incidence may be reduced by inducing parturition with prostaglandin F₂α; this results in rapid induction of labor and dilatation of the teats for a shorter period of time.
Thank You

Kindly share the video and subscribe to my You tube channel **Govind Narayan Purohit** if you like them