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How I Evaluate the Peri-Partum Mare with Colic: Special Considerations

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Take Home Message

Many mares show signs of abdominal pain at some stage during gestation or around the time of parturition. The most serious causes of colic during the peri-partum period (excluding colic associated with stage III labour) include rupture of the uterine, utero-ovarian or external iliac arteries, damage to the small colon and its mesentery, perineal injuries, large colon displacements and torsion, uterine rupture and inversion of the uterine horn. Consideration must be given to all these possibilities during the examination.

Introduction

Colic in the broodmare presents both diagnostic and treatment challenges. In the pregnant mare, we are faced with not only one but potentially two patients simultaneously, and the best course of treatment for one may not necessarily be what is best for the other. The goals of treating the pregnant mare with colic are to identify and correct whatever abnormality is present as soon as possible, and to support placental function to maintain fetal viability throughout the remaining period of gestation. The goal for the foal is to keep the maternal environment optimal to maintain the pregnancy so it may be born with a reasonable chance of survival outside of the uterus.

Although many different causes of colic can be encountered in the peri-parturient mare, there are certain specific diseases that are more commonly encountered at this time. This review will focus on some of these conditions.

Arterial Rupture

Rupture of the middle uterine, utero-ovarian, or the external ileac arteries at or around the time of foaling is a significant cause of colic and death in older (>11 years) foaling mares. Rupture of the middle uterine artery or utero-ovarian artery may result in the formation of a large, painful haematoma in the ipsilateral broad ligament that may dissect below the serosal uterine surface if the hemorrhage is contained within these structures. Pain results from the stretching of, and pulling on, these structures as the hematoma forms. If the broad ligament or serosa subsequently rupture and the haemorrhage is no longer contained, then the mare will rapidly bleed out into her abdominal cavity. Rupture of the external ileac artery, due to its anatomic location, results in the mare directly and fatally bleeding into her abdomen. Fatal bleeds are most common in aged mares (>18 years), and unfortunately the first occurrence of this disorder may be fatal.

The right middle uterine artery has been reported to be the most frequently affected of the susceptible vessels. There may be increased stress placed on this artery as a result of stretching...
of the right broad ligament due to pressure from a full cecum. Uterine artery rupture may also occur secondary to uterine torsion. Findings of aneurisms and intimal fibrosis in affected middle uterine and utero-ovarian arteries on post mortem examination have been reported, and support the theory that age related vascular changes predispose older mares to arterial ruptures.

Mares with uterine artery rupture demonstrate signs of colic, sweating, tachycardia, tachypnea, pale mucous membranes, thready pulse, cool extremities, depression, weakness and collapse. Initially, affected mares demonstrating signs of colic may be mistaken for mares sensitive to “after cramps”, but the signs of persistent pain and deterioration in the mares’ cardiovascular status and mucous membrane appearance readily distinguish them. Affected mares may also have swelling and edema in their vulva and perineal region more than that typically observed in a normal post foaling mare.

During the early course of a hemorrhage, the PCV usually remains normal. After 8-24 hours post acute hemorrhage, retention of sodium and water by the kidneys in an effort to correct hypovolemia result in a decrease in the PCV that will more accurately reflect the mare’s actual circulating red blood cell mass. Therefore, it is best to monitor affected mares with serial PCV measurements in order to accurately assess the actual degree of the mare’s anemia.

Mares whose hematomas rupture or who directly bleed into their abdomen from the start demonstrate rapid signs of shock and death. Mares whose bleeds are initially contained within the broad ligament may still die if the blood loss has been severe enough, or if the clot is disturbed and subsequently ruptures during the recovery phase (up to 2 weeks later).

Rectal palpation and ultrasound identifies a painful swelling of the haematoma within the broad ligament and over the uterus. Direct blood loss into the abdomen may be identified via transabdominal ultrasound. Free blood in the abdomen appears as a hypoechoic fluid with swirling echogenicity. Abdominocentesis will confirm these findings; abdominal fluid with a PCV >5% and a total protein > or = 3.5 g/dl supports a finding of frank haemorrhage into the abdomen.

Treatment of mares with extensive or uncontained uterine artery rupture is often unsuccessful. Attempts at surgical ligation are typically unsuccessful due to poor surgical access and the difficulty in finding the damaged arterial site within the volume of the haematoma. Also affected mares are very poor anaesthetic risks due to their poor cardiovascular status.

There are two approaches to managing affected mares that survive the initial stages of the hemorrhage – one conservative, the other more aggressive. Regardless of which therapeutic option is chosen, the mare must be kept as quiet as possible so as to minimize excessive increases in her arterial pressure. The conservative approach to treatment involves minimizing stress or excitement of the affected mare. The mare is kept in a quiet, darkened stall with or without her foal (depending on which is least stressful to the mare). Transportation of the mare is generally contraindicated. Tranquilizers are used judiciously as needed to help keep the mare calm, and, in the case of acepromazine, to help reduce arterial pressure directly. Naloxone (8-32mg/500 kg iv) has been anecdotally reported to be helpful in some mares. Aminocaproic acid (Amicar) (20 g in intravenous fluids as loading dose, followed by 10 g every 6 hours) has also
been recommended; this drug acts by inhibiting fibrinolysis. Analgesics (butorphanol 0.01-0.04 mg/kg IM) are also used as needed to control the mare’s pain. Attempts at volume re-expansion with fluids or whole blood transfusions are indicated to preserve cardiac output and perfusion, but may increase arterial pressure and disturb haemostasis.

The more aggressive therapeutic approach involves utilizing all of the above treatments as well as the careful application of subtotal volume re-expansion with crystalloid fluids to support tissue perfusion and whole blood transfusions or synthetic oxygen-carrying fluids as indicated to support tissue oxygenation. Care must be taken to keep mean arterial pressure below normal. Whole blood transfusion is not necessary until the mare’s PCV is <20%.

The prognosis for mares with uterine artery rupture is guarded. For those that survive the acute episode, it is important that they be kept quiet for several weeks as the clot resolves and the vessels slowly repair as increases in arterial pressure during this period can cause renewed bleeding. Final resolution of the hematoma may take months depending on the initial size. Mares that have survived their first episode have a high risk of recurrence of uterine artery rupture with subsequent pregnancies and foaling.

**Mesocolic Rupture**

Mesocolic rupture and subsequent segmental ischemic necrosis of the small colon occur as a complication of foaling and are the result of direct trauma caused by the foal as it positions itself for delivery. During first stage labour, movements of the fetus cause the small colon of the mare may become trapped between uterus and dorsal body wall, causing the mesocolon to tense and tear.

Mesocolic rupture can also result from type IV rectal prolapse, a condition sometimes associated with parturition. The vascular arcade of the mesocolon may stretch and tear when more than 30-cm of the rectum and small colon prolapses through the anus.

Following mesocolic rupture, infarction of the intestine results, causing functional obstruction and progressive signs of colic. Segmental ischemic necrosis of the small colon caused by disruption of the mesocolonic vasculature should be considered when examining post-parturient mares that show signs of abdominal pain, particularly when the cardiovascular health of the horse deteriorates slowly and concentration of protein and the nucleated cell count in the peritoneal fluid increase. Affected horses usually fail to pass feces and present with large amounts of impacted feces in the distal small colon and rectum.

**Perineal Trauma**

Perineal damage (1st, 2nd, and 3rd degree perineal lacerations, vestibular bruising, hematomas, excessive vulvar stretching, etc.) can cause significant pain, and some mares with normal post partum swelling and bruising may experience a reluctance to defecate and secondary constipation. Anti inflammatory drugs (phenylbutazone or flunixin meglumine) as well as local treatment with topical anti-inflammatory ointments are indicated to relieve pain and swelling of tissues. Walking and the administration of oral laxatives (mineral oil) and laxative feeds (bran
mashes, grass, etc) may also be helpful.

**Colonic Displacements and Torsions**

Broodmares are especially at risk of developing large colon displacements and torsions, especially during the first 100 days post foaling. Vital signs and the degree of colic in the affected mare are reflective of the severity of the colonic disorder, i.e. a large colon volvulus will present as a violently painful colic often with a very high heart rate (60-100bpm) whereas a simple colonic displacement may present with mild to moderate signs of colic with a relatively normal heart rate. Diagnosis is made by identification on rectal examination of an abnormally positioned, gas distended colon, and in cases of torsion with intestinal compromise, the abdominal fluid will be seosanguinous. Surgical correction is neccessary.

**Uterine Rupture**

Rupture of the uterus at foaling can cause peritonitis and/or abdominal pain. Diagnosis is made by rectal and ultrasound examination, in addition to abdominocentesis. If the tear is small and dorsal, conservative treatment with antimicrobials, crystalloids, colloids, peritoneal drainage and NSAI drugs may be successful. Surgical treatment may be required in large tears. If there is gross peritoneal contamination, the prognosis is poor.

**Inversion of a Uterine Horn and Uterine Prolapse**

Inversion of a uterine horn post-foaling frequently results in acute pain within the first few hours of foaling that is unresponsive to low-dose analgesics. Pain is the result of the ovary and tip of one horn becoming inverted and entrapped within the uterine lumen. The condition may progress to a complete prolapse of the uterus through the vulvar lips if left uncorrected.

Diagnosis of an inverted uterine horn is based on a rectal palpation finding of a blunted uterine horn with a tense mesovarium disappearing into the centre of the blunted tip. In minor intussusceptions, the ovary may not yet be entrapped and is still palpable at the very tip of the blunted horn. Palpation of this area is often painful to the mare. The inverted horn can also often be felt *per vaginum* within the lumen of the uterus.

Direct treatment and correction of the invaginated uterine horn includes controlling the mare’s straining and pain (sedation, epidural), manual reduction of the inverted horn per vagina (may require the use of uterine relaxants (acepromazine, clenbuterol), or even general anaesthesia to relieve the encircling spasm in the myometrium), and full replacement of the previously invaginated horn and ovary to their normal position. Supportive therapy in the form of intravenous fluids, NSAIDs, antibiotics, tetanus prophylaxis, etc may also be indicated. Careful use of low dose oxytocin (10-20 IU IM) once the horn has been returned fully to its normal position may also aid in rapid normal involution and prevention of a recurrence.

**Further Reading**


