

Complications during gestation in the cow

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Abstract

Uterine torsion and vaginal prolapse are accidents of gestation; this paper first reviews the predisposition, symptoms, diagnosis, and treatment of uterine torsion, and the predisposition, pathogenesis, symptoms, treatment, and prognosis of vaginal prolapse during late gestation in the cow. Other noninfectious complications of gestation are either fetal or placental in origin. Two relatively unusual fetal problems, mummification and maceration, are also discussed, followed by a comparison of two hydropic conditions (hydrallantois and hydramnios). Hydrops allantois, primarily a placental problem, occurs about nine times more frequently than hydrops amnii, which is a result of fetal anomalies. Abnormal offspring syndrome is a more recent phenomenon associated with the transfer of IVF or SCNT embryos. Finally, reference is made to teratology in general and a website of illustrations is cited.

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1. Introduction

There are many unrelated complications that can occur during gestation in the cow. This review will highlight noninfectious abnormal placental and/or fetal development. However, it does not address early embryonic death, which constitutes the largest percentage of pregnancy wastage. Where possible, the prevalence, treatment options, and prognosis of these complications will be discussed.

2. Uterine torsion

The ventral attachment of the broad ligament is along the lesser curvature of the uterus, leaving the greater curvature free, and predisposing the cow to uterine torsion during the third trimester. In *Bos indicus* cattle, the ventral attachment changes from ventral at the body to dorsal at the tip of the horn. As cows get up

on their hind legs first, the (gravid) uterus is temporarily suspended. The broad ligament is looser and longer in pluriparous cows. The abdomen is capacious, especially when the rumen is relatively empty. Strong fetal movements and poor maternal muscle tone further contribute to torsion of the uterus. In 89% of cases of uterine torsion, the weight of the fetus is above the mean [1].

There is evidence of abdominal pain and discomfort due to stretching of the broad ligament. Other signs such as anorexia, rumen stasis, constipation, increased pulse and respiration are usually present. The diagnosis of uterine torsion is based on a history of advanced pregnancy. On transrectal palpation, the orientation of the broad ligaments is distinctly altered; depending on whether the torsion is to the left or the right, the respective broad ligament is pulled tightly across the uterus. Spiral folds can be palpated per vaginam. Most torsions are to the left (counter clockwise); in general, the uterus rolls toward and over the nongravid horn (approximately 60% of all pregnancies in the cow are in the right horn). Thirty-four percent of uterine torsions

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occur anterior to the cervix (no vaginal involvement). Forty-five to 90° torsions are uncommon; 20% are 90–180°, 57% are 180–270°, and 22% are 270–360° [1]. Depending on the degree of torsion, the fetus may be in dorso-pubic presentation. With severe torsion, circulatory embarrassment occurs.

Treatment depends on the degree of the torsion. With rotations of 90° or less, the fetus can frequently be manually rocked into a normal dorso-sacral position. Greater rotations can be corrected by rolling the cow around the fetus, that is held in place by a plank in the flank [2]. Briefly, the cow is cast with ropes to lie on the side of the direction of the torsion. A long plank is placed in the paralumbar fossa of the cow and an adult person stands on the plank above the paralumbar fossa. Next, the front legs of the cow are tied together (as are the hind legs), and they are pulled up and over the recumbent cow. In intractable cases, a cesarean section must be done to deliver the fetus, suture the uterus, and manually untwist the uterus. The prognosis depends on the degree of severity and largely on the extent of vascular compromise; the latter may make the uterus friable, predisposing it to rupture.

3. Vaginal prolapse

The primary predisposition to cervico-vaginal prolapse in cattle is elevated plasma estrogen concentrations during late gestation. Contributing factors are pluriparity, *Bos indicus* breeding, obesity, large calves, and occasionally hilly terrain.

Pathogenesis and development of the prolapse is progressive; it starts with the exposure of some of the vaginal mucous membrane. The prolapsed mass moves in and out as the cow gets up and lies down. Exposed mucous membranes dry out and become irritated, leading to straining and greater exposure of the mass. The prolapsed tissues become edematous, leading to further circulatory impairment and more swelling. Ultimately the cervix and occasionally the bladder may become involved.

The protrusion of an angry-looking soiled mass is an obvious clue; a presumptive diagnosis is frequently made by the owner. However, there are several conditions that can be mistaken for a prolapsed vagina, including fetal membranes containing bloody fluids, vulvar hematoma, cystic vestibular (Bartholin) glands, and tumors.

Treatment depends on the severity and varies from simply elevating the hindquarters of the cow with a platform in a tie stall, to the placement of retention sutures or devices (prolapse pins) in the vulva. In

nonpregnant animals, a deep purse string suture (Buhner) is placed under epidural anesthesia. A disadvantage of the use of prolapse pins is that they may cause fistulas, whereas a deep purse string suture may be difficult to remove as it becomes embedded into the tissues, especially when there is contamination and the interval to calving is >1 week.

The prognosis depends on the severity. Since most of the prolapses occur during late pregnancy, the cow must be observed for signs of impending parturition to allow for timely removal of the prolapse pins or sutures. There is no association with the prolapse of the uterus after parturition. Although vaginal prolapse is likely to recur during the next pregnancy, uterine prolapse usually does not recur [3].

4. Mummification

Mummification of the fetus is a curious, but uncommon event. No specific cause has been identified for the mummification of most bovine fetuses. There are several prerequisites for the process of mummification: (1) the fetus must be dead; (2) there must be no air (oxygen) in the uterus (the cervix must be tightly closed); (3) no bacteria should gain access to the uterus via its blood supply. Therefore, certain viruses (enteroviruses, BVD) are speculated to be involved; they can kill a fetus quickly without causing further contamination and irritation. A cause that is easy to understand, but which rarely occurs, is torsion of the umbilical cord at an early stage of pregnancy.

Mummification is most common at end of the first and the beginning of the second trimester of pregnancy in the cow. After the fetus dies, the fetal fluids are gradually resorbed and the fetus itself becomes progressively more dehydrated. The wall of the uterus shrinks tightly around the fetus, ultimately even into the empty sockets of the eyes (after the eyeballs have completely shriveled). The caruncles disappear completely as well. The fetus slowly compacts and attains a leathery texture. The entire process takes several weeks, depending on the age of the fetus at the time of its death.

Meanwhile the cow is asymptomatic (albeit anestrous). Frequently the first indication that something is wrong is when the cow shows no udder development near the time she is expected to calve, and indeed she fails to calve. Examination at that time by transrectal palpation reveals a uterus that is devoid of fluids and that is drawn tightly around a small firm fetus with a bird-like head. The empty eye sockets are usually readily recognized.

Treatment is relatively simple, although often not economically feasible (as the cow is typically non-lactating and will require a minimum of 10 months before calving); therefore, in most cases, they are simply culled. A single treatment with a luteolytic dose of PGF_{2α} will induce estrus in 3–5 days; the cervix will relax and open, the uterus contract, and the mummy will be expelled. A vaginal exam 3–5 days after treatment is recommended; due to its sticky, dry nature, the mummy may be stuck in the vagina and require assistance to be delivered. The occasional large mummy, inadequate uterine contractions or inadequate cervical dilation may require the mummy to be delivered by cesarean section. After expulsion of the mummy, the uterus quickly regresses in size. Its lumen is not contaminated and the caruncles are already involuted; therefore, the cow may be bred back at the next estrus, with a good chance of conception.

5. Fetal maceration

Although fetal mummification occurs when the fetus dies in the uterus in the absence of air and bacterial contamination, and the cervix remains tightly closed, fetal emphysema and maceration occur when the cervix is open and miscellaneous bacteria invade the uterus from the vagina. Fetal maceration follows incomplete abortion, but it is not common; the latter may be the result of a partially dilated cervix or the abnormal presentation of a fairly dry fetus which causes it to be retained in the uterus.

With the dead fetus incubated at body temperature, bacterial multiplication is rapid and the fetus putrefies. Initially it becomes distended with gas and it subsequently decomposes. The wall of the uterus becomes thick and surrounds the disintegrating fetus like a capsule, as if to wall off an abscess. Consequently, the cow does not display severe systemic illness. After about the 3rd month of gestation, fetal bones resist maceration. Sharp pointed bones (e.g. fetal ribs) may deeply embed themselves in the uterine wall. Occasionally a bone perforates the uterine wall and becomes encapsulated by adhesions. The rare finding of a small bone free in the abdominal cavity of a cow at slaughter can be explained in this manner.

Meanwhile, the cow may display only vague signs of intermittent straining, accompanied by a foul, grayish-red vaginal discharge which may contain small bones. She may be mildly febrile, anorectic and depressed. These changes are usually noted in the lactating cow as her milk production also drops, but they are easily overlooked in heifers, dry cows, or beef cows. The

diagnosis is readily made by transrectal palpation. The uterus feels thick-walled and firm, fluctuation is largely absent, and, in advanced cases, crepitation of the fetal bones can be felt. There is usually also a slight, purulent vaginal discharge.

The prognosis for future fertility of the cow is very poor, due to the extent of endometrial damage. Therefore, treatment is usually not an economically viable option, and the cow should simply be culled. However, the cow may be condemned at slaughter if the foul smelling odor, due to bacterial putrefaction, has permeated the entire carcass. If the individual value of the animal warrants treatment, a luteolytic dose of PGF_{2α} can be given to induce estrus and attempt to evacuate the contents of the uterus. However, some bones may be partially embedded in the wall or lodged sideways, preventing their expulsion. As a last resort, in the case of a few isolated bones, they can be removed surgically. Surgery is best performed via a midline abdominal incision, to provide optimal access to the small, contaminated uterus.

6. Hydrops allantois (hydrallantois)

This condition is seen sporadically in dairy as well as beef cattle. Excessive accumulation of allantoic fluid may be progressive after mid-gestation. As much as a 10-fold increase in allantoic fluid volume, up to 200 L [4], has been reported (normal volume of allantoic fluid near term is 8–15 L). Placental dysfunction is evident by the occurrence of adventitious placentation characterized by a reduced number of placentomes (~75–120) and the development of a more primitive villous placentation. Nutritional deficiencies have also been reported to cause this condition (prevalence, up to 30%) in Criollo cattle owned by small holders in Zacatecas, Mexico and raised on poor soil during the long (December–May), severe dry season [5].

Affected cows have bilateral abdominal distension. They are distressed, anorectic and have no rumen activity (due to compression). Dehydration and constipation follow and eventually the thin cows become recumbent. During a transrectal examination, the uterine wall is very tight and it is difficult to palpate the fetus.

Salvage slaughter is generally recommended, due to the underlying adventitious placentation, which is a permanent alteration of the caruncular structures of the endometrium. Gradual drainage by repeated trocharization is possible, with concurrent oral fluid therapy. However, the allantoic fluid readily re-accumulates. Parturition can be induced if the cow is reasonably

close to term, provided she is concurrently given large amounts of electrolytes *per os*. When the accumulation of fluids is the result of nutritional deficiencies, as reported in Mexico, improved nutrition will help if the condition is diagnosed early. The prognosis for future fertility is guarded in cases of adventitious placentation, but more favorable when due to nutritional causes.

7. Hydrops amnii (hydramnios)

Hydramnios is a rare condition. Excessive accumulation of amniotic fluid can be the result of fetal anomalies such as impaired deglutition or renal dysgenesis or agenesis. The increase of amniotic fluid is gradual. When viewed from the rear, cows with hydramnios have a pear-shaped abdomen. It may affect only one of twin fetuses. Hybrids produced by the mating of an American Bison bull with a domestic cow cause an increased incidence of hydramnios. The prognosis for future breeding life of the dam is good, but the fetus is invariably defective and nonviable.

8. Abnormal offspring syndrome

Hydrops allantois is also a part of the Abnormal Offspring Syndrome (AOS), a negative consequence of the transfer of in vitro produced (IVP) embryos and somatic cell nucleus transfer (SCNT) embryos, in cattle and in other species. Embryos, fetuses, placentae, and offspring can differ substantially in morphology and developmental competence compared with those from embryos produced in vivo. Following the transfer of IVP or SCNT embryos in cattle, the scope of abnormal developmental conditions are more accurately described by the term AOS than the term Large Offspring Syndrome (LOS) [6].

Abnormal phenotypes resulting from IVP and SCNT embryos are stochastic in occurrence; they have not been consistently linked to aberrant expression of single genes or specific pathophysiology. Reliable methods of early diagnosis are not yet available. Abnormal development of the allantoic membranes and amount of fluid has been reported in placentae from IVP embryos [7]. The prevalence of hydrallantois is significantly higher in IVP pregnancies (one case in 200 pregnancies) than for normal pregnancies (one case in 7500 pregnancies) [8].

A fetal death rate of 24% has been reported in dairy cows pregnant following transfer of IVP embryos. Losses were greater between 50 and 80 days than from 80 days to term [9]. Although 50–100% of cloned pregnancies were lost between 30 and 60 days of gestation, placentae from cloned cattle during this period either had evidence of hypoplasia and reduced cotyledonary development or they were normal. Additional pregnancy loss of cloned cattle may occur during the second trimester, due to reduced number of cotyledons, and in the third trimester in association with hydrallantois, placental edema, reduced number of placentomes, and fetal anasarca [10]. Recently, a bovine interspecies model (*Bos taurus* × *Bos gaurus*) was used to identify imprinting status of genes in placental tissues in placental and fetal tissues in control and SCNT pregnancies at day 40 of gestation [11].

9. Teratology

Teratology may be subdivided into congenital anomalies and fetal monsters. Their occurrence is stochastic and relatively rare, which makes it difficult to study their etiology. A list of the more ‘common’ examples includes freemartins, atresia ani, cleft palate,

Table 1
Differential diagnosis between hydrallantois and hydramnios in cattle

Characteristic	Hydrallantois	Hydramnios
Prevalence	85–95%	5–15%
Rate of development	Rapid (within 1 month)	Slow over several months
Shape of abdomen	Round and tense	Piriform, not tense
Transrectal detection of placentomes and fetus	Nonpalpable (tight uterus)	Palpable
Gross characteristics of fluid	Watery, clear, amber-colored transudate	Viscid, may contain meconium
Fetus	Small, normal	Malformed
Placenta	Adventitious	Normal
Refilling after trocharization	Rapid	Does not occur
Occurrence of complications	Common	Uncommon
Outcome	Abortion or maternal death common	Parturition at approximately full term

Adapted from Refs. [2,4].

hydrocephalus, dicephalus, *Schistosoma reflexum*, fetal anasarca, and *Globosus amorphus*.

10. Illustrations

For illustrations for each of the foregoing conditions, go to the Visual Guide to Theriogenology (<http://drostproject.vetmed.ufl.edu>) and select the Bovine Guide. Most images will be located under Accidents of Gestation and Teratology.

11. Conclusions

Only the more common noninfectious accidents of gestation in cattle have been reviewed. Emphasis was placed on clinical manifestations, diagnosis, and resolution (Table 1). Because the prevalence of these complications is low, owners and veterinarians are less familiar with these abnormalities, and diagnosis may present a challenge. In most instances, the etiology is unclear, which makes it difficult to recommend methods of prevention.

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