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Etiology, antenatal diagnosis and therapy of fetal complications of gestation in large and small domestic ruminants

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Abstract

The etiology, antenatal diagnosis and therapy of common fetal complications of gestation in the large and small domestic ruminants are reviewed. The complications include those because of fetal death like abortion, fetal mummification and fetal maceration or because of the fetal developmental abnormality like fetal monsters, fetal dropsical conditions or ectopic location of the fetus. The etiology of most fetal gestational complications (except abortions) continues to be poorly understood. Most fetal complications (except fetal mummification and maceration) are identified at the time of parturition/abortion in the large and small domestic ruminants due to absence of routine evaluation of mid to late pregnancies and the paucity of data on growth patterns of the fetus and its annexes. Ultrasonographic evaluations of cattle and sheep and maternal hormonal and protein estimations have helped in the antenatal diagnosis of a few fetal complications in recent years. Similar descriptions for the buffalo and goat are largely unavailable. When fully developed, such methods can serve as a useful adjunct to the present methods of evaluation of fetal well being. Therapies for fetal complications are not specific and include those involved for the termination of pregnancy and the care of the dam at the time of parturition. It is concluded that the antenatal diagnosis of most of the commonly occurring fetal complications of gestation is possible with ultrasonography in cattle, sheep and goats and such pregnancies should be carefully monitored or terminated.

Keywords: Abortion, antenatal, domestic ruminants, dropsy, fetal mummification, ultrasonography.

Introduction

Many fetal problems can occur during pregnancy in the large (cattle and buffalo) and small (sheep and goats) domestic ruminants including fetal death, (abortion, mummification, maceration, monster) extra uterine pregnancies and fetal dropsical conditions. The etiology of most of these conditions is poorly described. Various diseases of the fetus or dam may have adverse effects on the pregnancy. Although extensively studied in human pregnancies the data are scant

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on the ultrasonographic manifestations of any anomaly in the last trimester of pregnancy (Buczinski, 2009). The objective of ante-natal fetal diagnosis for abnormalities is to offer termination of the pregnancy in humans, for diseases that are potentially fatal and for which there is still no adequate nor specific therapy. Ante-natal fetal diagnostic approaches in humans include ultrasonography, magnetic resonance imaging, chorionic villus sampling, amniocentesis and fetal blood sampling (Wong, 1989). In cattle and sheep data on approaches to detect fetal well being has been generated during the last few years (Scott and Gessert, 2000; Noia *et al*., 2002; Anthony *et al*., 2003; George *et al*., 2004; Jonker, 2004; Breukelman *et al*., 2006; Buczinski *et al*., 2007a; Buczinski, 2009) for establishment of normal fetal parameters (Buczinski *et al*., 2007a) and because of increased incidence of fetal disorders with the use of assisted reproductive technologies. Like conditions are not known for the buffalo and because of a deep capacious abdomen data generated in cattle are less likely to be useful. The termination of pregnancies with known fetal disorders is still not common in the large and small ruminants except under some specific conditions. The continuance of a pregnancy subsequent to fetal death may lead to fetal mummification or maceration which is undesirable and hence necessitate vetero-medical termination. Fetal monsters and dropsical conditions are frequently detected at parturition rather than during a pregnancy. The fetal complications of pregnancy for this review have been reviewed for etiology, ante-natal diagnosis and therapy under the following sub headings:

A. Fetal complication

1. Fetal death

1.1 Abortion

1.2 Fetal mummification

1.3 Fetal maceration

B. Fetal developmental abnormalities

1.4 Fetal monster

1.5 Fetal dropsical conditions

1.6 Ectopic pregnancy

Incidence

The overall incidence of fetal complications during gestation appears to be low in both the large and small domestic ruminants however; the incidence is based on calving data or the proportions of difficult births. Data of an organized Surti buffalo farm from 2001 to July 2011 involving 529 calvings revealed that the incidence of abortions was 3.96% and 4.53% of the buffalo calves were either born dead or died shortly after birth. There was no case of fetal mummification, fetal dropsy or fetal monster. In our previous study involving 156 cases of dystocia in cattle and buffaloes we recorded only 0.9% incidence of fetal dropsical conditions in cattle and nil incidences of fetal complications in the buffalo (Purohit and Mehta, 2006). In a more recent analysis

involving 192 and 112 cases of dystocia (1996 - 2010) we recorded the incidence of fetal dropsical conditions and fetal monsters to be 1.04% and 1.04% respectively in cattle and nil in buffaloes (Purohit *et al*., 2011a). Eight cases of fetal mummification and two cases of hydroallantois recorded during the same period were not included in this study as they did not result into dystocia.

Previous data analysis of 324 cases of dystocia in goats revealed that the total incidence of fetal complications was 1.54% and included cases of hydroallantois and fetal anasarca (Purohit *et al*., 2006a). Again 3 cases of fetal mummification and 2 cases of fetal maceration were not included in this study as they did not result in dystocia.

Fetal death

The *embryo* is termed a *fetus* from day 45 in cattle when the differentiation and cell organization begins in the bovine developing embryo. The fetus along with its membranes is known as the *conceptus*. Death of developing embryo/ fetus can occur any time during its uterine life. An embryonic death occurring before day 45 of gestation is considered early embryonic death (Miller, 1977) and is considered as infertility. Any fetal death beyond this time is usually considered an abortion.

Changes or affections of the fetus during mid or late gestation can result into fetal compromise which would either result into a fetus with altered form, structure or function (*fetal monsters, fetal dropsical conditions*) but such fetuses may continue to grow till complete gestation. When the fetal compromise is extensive fetal death occurs. A dead fetus is either delivered within 24 to 72 hours of its death (*abortion*) or may remain in the uterus with subsequent shrinkage or absorption (*fetal mummification*) or autolysis (*fetal maceration*) depending upon whether or not the cervix opens. The delivery of a dead fetus depends on the extent of fetomaternal signaling mechanisms operative between the uterus, ovaries and pituitary.

Abortion

Abortion has been defined in a number of ways but the most suitable definition would be "The preterm (before completion of gestation period) delivery of a dead/live fetus which is incapable of independent life (day 42 to day 260 of gestation in cattle, Peter, 2000)". Abortion is also defined as the premature expulsion of the fetus and usually occurs because the fetus has died *in-utero*. Bovine fetuses are usually incapable of independent life when they are delivered before completion of eight months of pregnancy. Fetuses delivered beyond this period are therefore termed "Premature births or deliveries".

The causes of abortion could be physical genetic/chromosomal, nutritional, chemical, drug induced, hormonal, infectious (bacteria, viruses and fungi), or miscellaneous. The abortions because of infections from bacteria may result from different sub types of the same bacteria affecting different domestic

animals (For example *Brucella abortus* affecting cattle, *Br. ovis* and *melitensis* affecting sheep and goats) or different species of bacteria, viruses or fungi. An infectious agent may sometimes result into abortion in a large number of animals, or opportunistic bacterial infections may result into sporadic abortions. At other times a generalized disease in an individual animal may have abortion as an unusual outcome.

Ante-natal diagnosis of abortion is frequently not possible in the large and small domestic ruminants and investigations are focused on evaluating the cause of abortion from aborted material or fetuses. Fetal viability or death is classically identified by fetal responses to tactile manipulation via recto-genital palpation in the large domestic ruminants however; currently fetal viability can be evaluated using hormonal, chemical and ultrasonographic parameters (Jonker, 2004). Sonographic parameters that predict fetal death in cattle include absence of fetal movements or fetal heart beat (Buczinski *et al*., 2011), low concentrations of maternal estrone sulfate or placental proteins, including pregnancy-associated glycoprotein (PAG) and pregnancy-specific protein B-60 (PSPB-60) (Jonker, 2004). When observed by ultrasonography fetal death is always preceded by a visible reduction of the amount of allantoic fluid and by segregation of the allantochorionic membrane from the endometrium (Breukelman *et al*., 2005). Examination for fetal development or death during pregnancy is not performed routinely therefore reference curves for normal fetal growth are scarce and there is considerable physiological variation in the normal values (Jonker, 2004). Reports on the use of ultrasonography for fetal well being monitoring in the buffalo are not available. Using ultrasonography early fetal death may be recognized in small ruminants by finding free floating masses along with ribbon like placental membranes. Soon after fetal death the placentomes lose their crisp margins (Haibel, 1990).

Once the process of abortion has started, little can be done to stop it. Clinicians often inject a dose of progesterone on complaint of discharge of vaginal mucoid secretions/blood or constant straining in a pregnant cow, buffalo or goat. It is important to mention that such cases should be first examined thoroughly. Vaginal secretions may originate because of vaginitis and sometimes such secretions may even attract male animals. Moreover, injecting progesterone parentrally to a female in which the process of abortion has started and the cervix has opened could be harmful, as it delays the delivery of a dead or compromised fetus which is undesirable. This may sometimes even result into fetal maceration especially in cows and buffaloes. It is therefore suggested to examine animals showing signs of abortion and initiate therapy only if the cervix is closed. A fetus when it dies in the uterus is usually expelled within 24-72 hours of its death.

Fetal mummification

Fetal death without abortion and without lysis of CL during the end of the first and beginning of the second trimester of gestation in the cow or buffalo results into autolytic changes in the fetus with absorption of placenta and fetal fluids and shrinkage of the uterus and the fetus. This is known as *fetal mummification*. The cervix is closed in this condition and the fetus is sterile (Drost, 2007). Since the corpus luteum is intact, the mummified fetus continues to remain in the uterus and the animal remains anestrus. Fetal death before formation of the fetal bones does not result into mummification as the entire contents may be reabsorbed. One or more mummified fetuses present in the uterus with one or more normal viable fetuses are frequently observed in multiparous species (Hannon, 1981) and rarely in sheep, goats (Markandeya *et al*., 1991; Dadarwal *et al*., 2000), cattle (Gorani *et al*., 1996) and buffaloes (Khar and Nigam, 1971; Saxena *et al*., 2006; Martuciello *et al*., 2009).

Two types of fetal mummification have been described in animals, the *haematic* type in cattle and buffaloes and the *papyraceous* type in the other species. In cattle and buffaloes, when the placental caruncles involute after fetal death, hemorrhage occurs between the endometrium and fetal membranes. This imparts reddish-brown colour to the fetus and hence the mummification is known as *haematic*. Mummification occurs in cattle of all ages and has been recorded in various breeds of cattle and buffaloes (Jalakas, 2000; Drost, 2007). The causes of mummification are poorly described, and it is considered that infectious agents like *Campylobacter fetus*, molds, leptospriosis and BVD-MD virus causing fetal death without abortion may result into mummification in cattle (Drost, 2007) and buffaloes (Martuciello *et al*., 2009). In sheep and goats toxoplasmosis, Blue tongue and BVD are known to result in fetal mummification (Luedke, 1985; Dubey, 1990; Sharma *et al*., 2003; Broaddus *et al*., 2009; Dubey, 2009). Poisoning with toxic plants is a known cause of fetal mummification in goats (Smith, 1979). Torsion of the fetal umbilical cord may sometimes be a cause of its mummification. Accidental prepubic tendon rupture and fetal mummification has been reported in a goat (Singh *et al*., 2008).

The condition can be diagnosed during routine pregnancy examination or sometimes when animals are referred for anestrus. Primiparous heifers are sometimes referred to the clinician with a history of recession of the formerly well developing udder. Transrectal palpation reveals a uterus that is devoid of fluids and that is wrapped tightly around a small firm fetus with a bird-like head. The empty eye sockets are usually readily recognized (Drost, 2007). As the fetus mummifies the uterine wall contracts and tightly enclose the conceptus. The longer the condition exists, the dryer, firmer and more leather-like the tissues of the fetus become. The

uterine wall becomes thick and the animal remains anestrus due to persistence of CL. Sonographically the uterine walls are thickened, no non-echogenic fluid is visible, echogenic areas and hyperechogenic bones may be seen (Kumar and Purohit, 2009). Sometimes animals are not referred for long periods and the mummified fetus inside the uterus may be embedded in the uterine wall. A rare case of mummified fetus reaching the perimetrium of the uterus has been seen by the author and a case of fetal mummification along with uterine torsion has been reported (Moore and Richardson, 1995). Fetal mummies in sheep and goats are generally delivered along with normal fetuses (Kirkbride, 1993) or rarely recognized on abdominal ballottement or vaginal examination. They can be identified by careful sonographic examination (Purohit *et al.*, 2006a). Goat fetal mummies can be identified as hyperechoic areas without any identifiable body parts within a relatively fluid free placentome-less uterus (Haibel, 1990).

The therapy of mummified fetus when diagnosed early is simple. Medicaments that cause CL lysis like estrogens and prostaglandin's are helpful in delivery of the mummified fetuses within 48-72 hours in cattle, buffaloes and goats (Purohit *et al*., 2011b). Clinicians must reexamine cows that are treated with such treatments, as sometimes the fetus may be lodged in the cervix or vagina (Elmore, 1992; de Araujo *et al*., 2006) from where it has to be removed manually. In long standing cases one or many of the medical therapies like prostaglandins, estrogens, isoxsuprine HCl or ritodrine may dilate the cervix but fail to deliver the fetus which is tightly adhered to the uterine wall. Such cases continue to evidence anestrus (Lefebvre *et al*., 2009). In such cases, if the uterine horns are suspended in the abdominal cavity ahead of the pelvic brim, they may be removed surgically through laparotomy under paravertebral nerve block. It is however, not possible to surgically remove a mummified fetus from a uterus that is located largely in the pelvic cavity. When removed surgically large portions of the uterus have to be incised to remove a long standing mummified fetus deeply embedded in the uterus. Colpotomy and hysterotomy is another technique suggested for removal of mummies in invaluable animals (Irons, 1999; Hisbrunner *et al*., 2004). In sheep and goats the presence of mummified fetuses along with a normal fetus is usual and this dictates the continuance of a pregnancy rather than its termination.

The fertility of an animal after medical removal of a mummified fetus is good because the fetus is largely sterile. However, fertility subsequent to surgical removal of a mummified fetus deeply embedded in the uterus is fair to poor. Rarely a goat may deliver one fetus and the second fetus may mummify subsequently (Tutt, 1997).

Fetal maceration

Fetal maceration is the disintegration of

a fetus that has died after formation of the fetal bones (beyond 4 months of pregnancy in large ruminants and beyond 100 days in the small domestic ruminants) and has failed to abort, although the cervix is open. Although uncommon, the reason for the non delivery of a dead fetus could be a partially dilated cervix, uterine inertia, or the abnormal presentation of a fairly dry fetus which causes it to be retained in the uterus (Drost, 2007). Bacterial invasion of the fetus leads to fetal emphysema and maceration (3 to 4 days). There is formation of plenty of (24-48 h) pus and the bones of the fetus separate out.

The animal discharges plenty of foul smelling, reddish gray vulvar discharge and strains frequently. Fever and anorexia may develop due to infection. The condition is noticed by the owners when foul smelling pus is discharged by a pregnant animal. Rarely, it may be diagnosed during pregnancy examination. The condition can be diagnosed by the history, finding of a piece of bone lodged in the cervix, rectal palpation (free fetal bones palpable in crepitating pus and doughy thick uterine wall) radiography (especially is small ruminants) and ultrasonography (finding of hyperechogenic scattered bones in an echogenic or non echogenic fluid with echogenic floating pus) (Kumar and Purohit, 2009). The condition is commonly found in cattle and buffaloes but rarely may be encountered in small ruminants (Mehta *et al*., 2005; Ajit Kumar *et al*., 2007).

The animal should be examined per vaginum and any pieces of bones lodged in the vagina or cervix must be removed manually if the cervix is dilated. Infusion of large quantities of normal saline in the uterus is useful in removing the pus and bone pieces 24 hrs later. When the cervix is not open, prostaglandins or estrogens can be given to lyse the partially lysed CL and/or increase the uterine contractions. Sufficient lubrication and gentle removal of the bones is necessary. Sometimes, it may require several days for the uterus to be cleared of the entire pus and bones. Supportive therapy with antibiotics, fluid replacement and corticosteroids is indicated in severe cases. Laparohysterotomy to remove the macerated fetus is potentially dangerous and must be considered as a last resort (Honparkhe *et al*., 2008; Sood *et al*., 2009). Cows should not be rebred for at least 3 to 4 months after complete removal of a macerated fetus. The prognosis is always poor. Rarely long standing cases may develop peritonitis subsequent to uterine rupture.

Fetal monster

A fetal monster is an individual that has undergone severe damage during pregnancy without death usually changing its appearance (Jackson, 2004).

The etiology of fetal monstrosities continues to be poorly understood. Many of the congenital defects are inherited while others are because of the environment (nutrition, toxins, and

infections). Changes usually do occur in the conceptus during the embryonic period (day 14 to 42 of gestation). Although uncommon in most dairy and beef cattle herds congenital anomalies are found in all breeds of cows and propagated as a result of specific trait selection that inadvertently results in the propagation of the defect (Whitlock *et al*., 2008). The most common inheritance pattern is a simple recessive trait. A large number of fetal monstrosities have been reported in the buffalo species (Purohit *et al*., 2011a) but none has been traced to be of genetic origin. In sheep and goats the incidence of fetal monsters is rare. There are many teratogenic agents that are present in the environment. Each one can affect one to many species of animals. Some of them include tobacco, poison hemlock, lupine, locoweed, Sudan grass and even potato (Mc Ilwraith and James, 1982; Beasley, 1999; Knight and Walter, 2004). Viral infections like Blue tongue, Akabane virus, Border disease, Cache valley virus and BVD can result into congenital defects in cattle, sheep and goats (Luedke, 1985; Chung *et al*., 1990; Stokka *et al*., 2000).

There is paucity of data on the ante-natal diagnosis of fetal monstrosities. Diagnosing the cause of a congenital defect after the birth of a fetus is also difficult and requires constant cooperation of animal owners, veterinarians, technicians, and pertinent diagnostic laboratories (Leipold and Dennis, 1986).

Therapeutic options for fetal monstrosities are oriented towards selected breeding if the origin of the defect can be traced. If a congenital defect can be diagnosed during pregnancy the termination of such a pregnancy is suggested.

Dropsical conditions of the fetal membranes and fetus

Dropsical conditions of the fetal membranes

The term *dropsy* means swelling of soft tissues due to excessive accumulation of fluid. The dropsical conditions of the fetal membranes are *hydrammios, hydrallantois* and *edema of the allantois chorion*. The conditions are usually present singly but rarely may be found together. Hydroallantois is the most common condition observed in cattle compared to other dropsical conditions.

Hydroammios (Hydropsamnii)

Hydroamnios which is a rare condition (Drost, 2007) is characterized by a gradual enlargement or filling of the amniotic cavity and is associated with a congenital defect of the fetus (Roberts, 1985; Troy, 1993), with fetal anomalies such as impaired deglutition or renal dysgenesis or agensis (Troy, 1993; Drost, 2007). The condition is seen commonly in cattle, occasionally in sheep and rarely in buffaloes. The condition develops slowly over several days or months during the latter half of pregnancy, usually the last six weeks of pregnancy in cattle (Zdunczyk and

Grunert, 1999).

Ante-natal diagnostic procedures for diagnosis of hydroamnion in the large and small domestic ruminants are far from perfect and visual diagnosis is still the most frequently used procedure. On viewing from the rear, cows with hydroamnios have pear shaped abdomen (Drost, 2007). With the exception of the difficulties in observing the fetus and smaller placentomes in ultrasonographs no precise ultrasonographic descriptions of the condition have been reported (Heyman *et al*., 2002). The ante-natal diagnosis is thus dependent on the clinical signs.

Pregnancy termination is suggested if the condition is diagnosed early. Often the condition is not recognized until parturition when a large quantity of syrupy, viscid amniotic fluid occasionally containing meconium is released. The fetus is usually small but dies shortly because of defects. Many a times, there may be uterine inertia and a resultant dystocia but this can be handled easily. The prognosis and future fertility of cows is good however, if a genetic predisposition is found it has been suggested that the dam and sire both be discarded from future breeding.

Hydroallantois (hydrops allantois)

This condition is seen sporadically in dairy as well as beef cattle (Drost, 2007). The condition is usually associated with a diseased uterus in which most of the caruncles in one horn are not functional and the rest of the placentomes are greatly enlarged and possibly diseased (Roberts, 1985). The condition may be observed in cows carrying twin fetuses. More recently it has been shown that hydropic conditions are common in clone pregnancies (Hill *et al*., 1999; Heyman *et al*., 2002; Tsunoda and Kato, 2002; Fecteau *et al*., 2005).

The abnormality is probably caused due to structural or functional changes in the allantois chorion including its vessels, and transudation and collection of fluid resembling plasma. The condition usually affects cows of 3 years or more of age (Roberts, 1985). Fetuses may be slightly smaller and show some edema. The condition has been recorded in goats (Morin *et al*., 1994; Purohit *et al*., 2006a) ewe (Milton *et al*., 1989) and buffaloes (Narsimhan and Thangraj, 1968; Gupta *et al*., 1975; Bawa and Sharma, 1977; Srinivas and Sreenu, 2006). Nutritional deficiencies have been described to cause the condition (Flores *et al*., 1977) however; the exact etiology continues to be poorly understood.

Ante-natal diagnosis of the condition is dependent on the clinical signs. The signs of hydroallantois vary depending upon the degree of involvement and the stage of pregnancy. In mild cases where the amount of extra allantoic fluid accumulation is moderate, clinical signs are not appreciable during pregnancy, and increased fluid is only noticed at parturition when uterine inertia may also be present. In severe cases, the fluid

accumulation increases rapidly over a period of 5 to 20 days and is recognizable clinically by bilateral distension of the uterus and abdomen after mid gestation. As much as a 10- fold increase in allantoic fluid volume, up to 200 L (Sloss and Dufty, 1980), has been reported (normal volume of allantoic fluid near term 8-15L). Placental dysfunction is evident by the occurrence of adventitious placentation characterized by a reduced number of placentomes (less than 75) and the development of a more primitive villous placentation in cattle. Dysfunction of fetal kidneys may be present (Roberts, 1985). Affected animals have a bilateral distension and an apple shaped abdomen. They are distressed, anorectic and have no rumen activity (due to compression). Dehydration and constipation follow and eventually cows may become recumbent. During a transrectal examination, the uterine wall is very tight and it is difficult to palpate the fetus. Ultrasonographic findings in large ruminants are inconclusive as there is difficulty in observing the fetus and its annexes although larger sized placentomes are visible (Heyman *et al.*, 2002). The depth of the uterus and the size of the calf may be the limiting factors for fetal ultrasonography in cattle (Jonker, 2004). Similar descriptions in buffaloes and small ruminants are lacking.

When the condition is diagnosed early, parturition must be induced using prostaglandins and corticosteroids (Purohit *et al.*, 2011b). However, care must be taken to supplement sufficient fluid replacements to avoid death of the animal due to shock. Gradual drainage of the fluid by repeated trocharization is possible, with concurrent fluid therapy; however, the allantoic fluid readily re-accumulates (Drost, 2007). The prognosis for future fertility is generally poor. Laparohysterotomy may be suggested in some cases that require immediate termination of pregnancy but, due care must be exercised to avoid sudden withdrawal of allantoic fluid in order to prevent shock. Half of the does suffering from hydroallantois died during or after surgery at our center (Purohit *et al.*, 2006a). Similarly both the cows in which parturition was induced because of a presumptive diagnosis of hydroallantois at our center died during or immediately after induced parturition.

Fetal dropsical conditions

Three fetal dropsical conditions are commonly discussed in the large and small domestic ruminants. In these conditions there is an excess of body fluid, primarily within the fetal interstitial spaces. Nomenclature of the condition depends upon the location of the fluid: hydrocephalus-meninges of the brain; ascites-abdominal cavity and anasarca- generalized all over the body. In humans the conditions are collectively named as “hydrops fetalis” because more than one type may be existent at the same time (Randenberg, 2010).

Hydrocephalus

Hydrocephalus has been reported in virtually all major beef and dairy breeds

of cattle (Gilman, 1956; Baker *et al*., 1961; Greene *et al*., 1974; Christoferson *et al*., 1977; Balasubramaniam *et al*., 1997; Purohit *et al*., 2006; Singh and Brar, 2008), buffaloes (Rao *et al*., 1975; Bhandari *et al*., 1978; Salunke *et al*., 2001; Kumaresan *et al*., 2003), sheep and goats (Dennis, 1974; Majeed *et al*., 1992; Balagopalan *et al*., 1996). Two types of hydrocephalus have been described: the external hydrocephalus- in which the fluid accumulates in the sub-arachnoid space exterior to the brain; and the internal hydrocephalus- in which fluid accumulates in the ventricles of the brain. The fetus dies at birth or soon after birth.

The etiology of fetal hydrocephalus in humans is associated with intra-cranial hemorrhage (Yung *et al*., 2011), chromosomal abnormalities (Nomura *et al*., 2010; Syrios *et al*., 2011), vitamin K deficiency (Kawamura *et al*., 2008). In the bovine species an autosomal recessive gene is considered responsible for many hereditary causes, but infections and nutritional factors may also play a role (Mc Kercher *et al*., 1970) however, the exact etiology of the condition continues to be poorly understood both in animal and human subjects (Yung *et al*., 2011).

Antenatal diagnosis of the condition is important in human medicine because it can result into neurological disorders in newborns. Ultrasonography is a routine to diagnose hydrocephalus in human patients. The ventricles within the head measure larger than normal. Amniocentesis is performed to evaluate chromosomal abnormalities. Fetal magnetic resonance imaging (MRI) can provide additional information about the soft tissues (Blondin *et al*., 2008). Since the routine evaluation of mid to late gestation pregnancies in large and small domestic ruminants is not common the antenatal diagnosis of fetal hydrocephalus is not possible. In one report two cows with a prolonged gestation were studied by trans-abdominal ultrasonography and hydrocephaly was detected in one fetus (Buczinski *et al*., 2007b). The condition is generally found at the time of parturition. Death of the fetus results due to pressure on vital centers of the brain. The frontal, temporal and parietal bones are usually involved which become deformed separated and thin. Diagnosis of the condition subsequent to birth of the fetus is easy. A high dome shaped forehead and downward slant of eyes is characteristic to hydrocephalus.

No specific therapy is suggested during gestation. The role of pro-inflammatory cytokines (present in the cerebro spinal fluid) in the development of hydrocephalus has been recently shown (Sival *et al*., 2008) but the efficacy of anti-inflammatory therapy has not been investigated. When the enlargement is excessive the passage of the fetus through the birth canal at parturition may be difficult and cuts on the head are suggested to reduce the size of the fetus (Purohit *et al*., 2006b). Sometimes, the calf may be born normally (Nandakumar *et al*., 1999) or caesarean section is

required for delivery of the fetus (Rao *et al*., 1975; Balasubramaniam *et al*., 1997; Sharda and Ingole, 2002).

Fetal ascites

Fetal ascites is the accumulation of excess of fluid in the abdominal cavity of the fetus. It is known to be existent in cases of brucellosis, mesotheliomas of the fetal abdomen or fetal death with sterile autolytic changes (Roberts, 1985). The condition has been reported in buffaloes (Honparkhe *et al*., 2003; Palanisamy *et al*., 2007; Srinivas *et al*., 2007; Solanki *et al*., 2010; Vidya Sagar *et al*., 2010) and goats (Purohit, 2006a; Purohit, 2006b; Purohit *et al*., 2006a).

Various etiologies defined recently for fetal ascites in humans are: genetic causes, fetal structural abnormality, congenital syphilis or other infections, fetal environment, placental defects, renal problems of the fetus, teratogens and idiopathic (Zelop and Benacerraf, 1994; Dorairajan, 2010; Boutall *et al*., 2011). Similar causes may probably be existent in the large and small ruminants.

Antenatal diagnosis of fetal ascites has not been described for large and small domestic ruminants. Diagnostic procedures described for horses mention ultrasonography (Reimer, 1997) and those for humans include ultrasonography, Doppler studies, TORCH screening, fetoscopy and amniocentesis (Boutall *et al*., 2011).

Elective cesarean section is suggested for human patients with hydrops fetalis including fetal ascites (Son *et al*., 2010) and for fetuses delivered normally surgical repair is recommended (Hidaka and Chiba, 2009; Hirselj *et al*., 2009). Similar approaches are not described for the large and small ruminants. During parturition the distended abdomen of the fetus is wedged at the pelvis and results in dystocia in cattle (Purohit and Mehta, 2006). Such fetuses seldom survive and hence an incision on the fetal abdominal wall is made to release the extra fluid. The fetus can then be easily removed in cattle and buffaloes.

Fetal anasarca

Anasarca means generalized edema all over the body. The condition is seen commonly in cattle but may affect sheep, (Roberts, 1985), goat (Tamuli *et al*., 1987; Sharma *et al*., 2002; Purohit *et al*., 2006a) and also rarely reported in the buffalo (Devanathan *et al*., 1990). Abortions of affected fetuses are common between 4 to 8 months of gestation in cattle. The condition has been described to affect Ayrshire cattle and is caused by a recessive autosomal gene. Most anasarcous fetuses are expelled dead. When the fetus poses difficulty in its delivery, cuts must be given over many places to release the fluid or fetotomy and/or forced extraction may be used to deliver the fetus. Rarely caesarean section may be indicated. Antenatal diagnostic techniques described for the diagnosis of hydrops fetalis including fetal anasarca in humans include ultrasonography (Having and Bullock,

2011) and magnetic resonance imaging (Nassenstein *et al.*, 2006). Similar descriptions are not available for the large and small domestic ruminants and the therapy suggested is termination of a pregnancy.

Ectopic pregnancies

Although abdominal pregnancies have been described in human and animal species, tubal ectopic pregnancies appear to be restricted to primates.

Other than anecdotal cases, this pathological condition does not occur in domestic or farm animals (Hunter, 2002). In primary (true) ectopic pregnancy the fetoplacental unit forms outside the uterus. This is seen in primates where the placenta is invasive type and uterus is simplex type whereas such conditions are not possible in domestic animals. Although primary abdominal pregnancies have been reported in domestic species (Botcherby, 1980; Hedge, 1989; Davies, 1982; Mitchell, 1989), no viable fetoplacental units outside an intact uterus have been found during the latter half of pregnancy. A secondary extra uterine pregnancy is one which has a fetoplacental unit in the uterus but is located outside the uterus. Some mechanisms which distinguish women from domestic animals are i) in women, the presence of embryo is not required in the uterus for luteal progesterone secretion ii) the human embryo is capable of surviving in either the tubes or uterus iii) the placenta is haemochorial and implantation is invasive. All these characters are not found simultaneously in any domestic animal (Corpa, 2006) and hence a primary ectopic pregnancy is usually not possible in most domestic animals. Accidental rupture of the uterus with resultant ectopic pregnancy has however been recorded in a cow (Krishankumar *et al.*, 2008).

Clinical signs of distress, ultrasonography, peritoneoscopy and other techniques are commonly employed in humans for the diagnosis of ectopic pregnancies, but since no true ectopic pregnancy has been found to occur in the large and small domestic ruminants their antenatal diagnosis are out of question.

An emergency laparo-hysterotomy is routinely practiced in confirmed human ectopic pregnancies but such techniques are seldom required for the large and small domestic ruminants.

Conclusions

It is concluded that the etiology of common fetal gestational disorders continue to be poorly understood in the large and small domestic ruminants. The antenatal diagnosis of most of the commonly occurring fetal complications of gestation is partly possible with ultrasonography and such pregnancies should be carefully monitored or terminated.

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