# UTERINE TORSION IN BUFFALOES: A CRITICAL ANALYSIS

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## ABSTRACT

Critical evaluation of uterine torsion in buffaloes revealed that it is the single largest cause of maternal dystocia in referral cases, its incidence ranges from 52 to 70%, and it affects buffaloes mostly towards terminal gestation. The etiology of the condition continues to be partly understood with weaker broad ligaments, smaller quantity of fetal fluids and decrease in uterine tone and size at terminal stages of gestation coupled with inordinate fetal movements appear to be the precipitating factors. In most studies right sided uterine torsion was prevalent and postulated to be because of the presence of the rumen on the left side and absence of a muscular fold in the right broad ligament of the buffalo. During recent years it has been mentioned that due to uterine circulatory disturbances and muscle exhaustion blood parameters evaluating liver and kidney functions can be used as prognostic indicators for the future outcome of uterine torsion affected buffaloes. Diagnostic evaluation of the condition continues to be transrectal palpation of the broad ligaments which rotate along with the rotating uterus. The success of management of the condition lies in the correct and timely diagnosis and early referral to referral centers. In cases presented within 24 h of the problem, rolling is often successful in correction of the problem failing which laparohysterotomy should be performed immediately. Cervical dilation failure is observed in many buffaloes after correction of the uterine torsion and can be managed by cervical massage with sodium carboxy methyl cellulose and prostaglandin injections. Management of the general condition of the patient is of utmost significance for the dam survival. It was concluded that liver and kidney function tests can prognosticate the outcome of uterine torsion and early presentation to referral centers culminate in successful management of the condition with high dam and fetal survival.

**Keywords:** buffalo, clinical pathology, laparotomy, rolling, uterine torsion

## INTRODUCTION

Torsion of uterus usually occurs in a pregnant uterine horn and is defined as the twisting of the uterus on its longitudinal axis (Purohit *et al*., 2011a, b). The pregnant uterus rotates about its long axis, with the point of torsion being the anterior vagina just caudal to the cervix (Purohit *et al*., 2011a, b) (*post-cervical torsion*). Less commonly the point of torsion is cranial to the cervix (*pre-*

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*cervical torsion*). Uterine torsion during pregnancy (Murty *et al*., 1999), at parturition (Sharma *et al*., 1995; Prasad *et al*., 2000; Matharu and Prabhakar, 2001), or post-partum (Manju *et al*., 1985; Mathijsen and Putker, 1989) is one of the complicated cause of maternal dystocia in buffaloes culminating in death of both the fetus and the dam if not treated early. Because of the rapidity of fetal death that ensues following torsion and the uterine adhesions with visceral organs that develop, uterine torsion must be considered as an emergency. Torsion can result into haemoperitoneum if it results from horn butting between animals (Jadhao *et al*., 1993). A rare case of uterine torsion in a buffalo carrying twin fetuses has been reported (Siddiquee and Mehta, 1992). Uterine torsion is considered to be a more frequent maternal cause of dystocia in buffaloes compared to cattle (Purohit *et al*., 2012). Here we analyze the incidence, etiology, clinical signs, clinical pathology and management of uterine torsion in buffalo.

**Incidence**

The incidence of uterine torsion is considered to be higher in buffaloes although most reported data originate from clinical records and not from actual calvings at organized farms. A retrospective data analysis (2001-2011) of our university farm at Vallabhnagar revealed no incidence at all in 529 calvings of the Surti breed. Data involving clinical cases report that uterine torsion is considered to be the single largest maternal cause of dystocia in buffaloes with an incidence as high as 56 to 67% (Singh *et al*., 1978; Nanda *et al*., 1991; Prasad *et al*., 2000; Purohit *et al*., 2012) and up to 70% (Nanda *et al*., 2003). Uterine torsion cases in buffaloes are 67-83% of the dystocia presented at referral hospitals (Vasishta, 1983; Malhotra, 1990; Singh, 1991; Prabhakar *et al.,* 1994; Srinivas *et al.,* 2007). Uterine torsion has been reported mostly in dairy type buffaloes of India, Pakistan (Ahmed *et al*., 1980) and Egypt (Foud and El-Sawaf, 1964; El-Naggar, 1978), but reports on its occurrence in the swamp buffalo are not seen. The incidence is known to be higher in pleuriparous buffaloes (Singh *et al*., 1978; Pattabiraman *et al*., 1979; Sharma *et al*., 1995; Matharu and Prabhakar, 2001) with maximum frequency during second and third calvings (Nanda *et al*., 1991; Murty *et al*., 1999).

Although a seasonal incidence (Singh *et al*., 1978; Prabhakar *et al*., 1994) has been described in buffaloes, it appears to be because of higher calvings during that season. The usual age (years) of animals that suffer from uterine torsion is 4-12 for buffaloes (Pattabiraman *et al*., 1979; Amin *et al*., 2011).

**Etiology**

The exact etiology of a higher incidence of uterine torsion in buffalo continues to be poorly understood. The most logical explanation for rotation of a pregnant uterus on its axis appears to be the instability of the uterus during a single horn pregnancy and inordinate fetal or dam movements (Purohit *et al*., 2011a). The bubaline amnion is fused at many places to the surrounding allantois, which is attached to the uterine wall (El-Naggar and Abdel-Rouf, 1971) and the uterus lies on the abdominal floor during mid and late gestation with no stabilizing attachments; hence, rotatory fetal movements during the second stage of labor or late gestation would rotate the uterus. A smaller quantity of fetal fluids and an associated decrease in the size of the uterus at the terminal stages of gestation and a lower uterine tone at this time (Ghuman, 2010) increases fetal discomfort and this initiates further fetal movements and a greater degree of torsion. A large number of predisposing causes have been

described (Sane *et al*., 1982; Purohit, 2006) for uterine torsion and include anatomical factors, close confinement, hilly tracts, external injury and wallowing habits of the buffaloes and the lowering of front legs by the animal first, when lying down (Purohit *et al*., 2011a). The higher occurrence of the problem in buffaloes is also hypothesized to be because of the deep capacious and pendulous abdomen of the buffalo (Singh, 1995), inherently weaker muscles of the broad ligaments (Singh, 1991; Singh *et al*., 1992) and the wallowing habits of the buffalo (Singh, 1995). However, none of the uterine torsion affected buffaloes presented at a referral hospital had the history of wallowing or grazing on hills (Nanda and Sharma, 1986) and daily forceful wallowing of pregnant buffaloes failed to induce uterine torsion in one study (Agarwal, 1987). The role of broad ligament musculature in the occurrence of uterine torsion is highlighted from the observation that broad ligaments of bovines suffering from uterine torsion are thin and have less muscle compared to their counterparts with other types of dystocia (Singh, 1991). At least 25% of the females born to uterine torsion affected dams and 11% of the non-pregnant buffaloes have poorly developed muscles in their broad ligaments (Brar *et al.,* 2008a). Additionally, broad ligament musculature is better arranged in cattle compared to buffaloes, thus providing better stability to the pregnant uterus of cattle (Prabhakar *et al.,* 1994, Brar *et al.,* 2008b). A lack of dorsal attachment of the broad ligament during pregnancy (Brar *et al*., 2008a) and a lack of support of the broad ligament in the post cervical area predisposes buffaloes to more occurrence of post-cervical torsion (Brar *et al*., 2008c). Some exciting causes for the occurrence of uterine torsion have been described (Sane *et al*., 1982) and include external injury, lack of exercise and irregular movement of animals. Uterus didelphus has been described as one of the causes of uterine torsion in the buffalo (Singh *et al*., 1995). Slight rotations (below 90°) are symptomless clinically and may be corrected spontaneously but rotations of higher degree usually do not resolve spontaneously.

Among the referred cases of torsion, the pregnancy period is generally complete in 83-85% of buffaloes (Prabhakar *et al.,* 1994, Srinivas *et al.,* 2007). Occasionally, uterine torsion can occur between the 5-8th month of pregnancy (Singh *et al.,* 1979; Purohit *et al*., 2011b). Torsion of the uterus may accompany intestinal obstruction (Dhaliwal *et al.,* 1992), haemoperitoneum (Jadhao *et al.,* 1993), uterine rupture (Amin *et al*., 2011) and formation of adhesions of uterus with surrounding viscera (Siddiquee, 1988).

**Nature of Uterine Torsion**

A pregnant uterine horn may rotate at mid to late gestation, at normal parturition time, or sometimes post partum. The horn may rotate to its right (*clock-wise*) or left side (*anti-clockwise*) with degree of rotation varying between 90° to 720°. The point of rotation can be caudal to the cervix (*post-cervical*) (Deori *et al*., 2009) or just cranial to the cervix (*pre-cervical*). Uterine torsion generally occurs at late pregnancy and at parturition in buffaloes (Ali *et al*., 2011), on the right side of the abdomen at a point just caudal to cervix and usually the uterus rotates to 180° or more from its axis (Amin *et al*., 2011; Ali *et al*., 2011; Purohit *et al*., 2011a, b). Post-cervical uterine torsion is common in buffaloes (87-99%); (Vasishta 1983, Malhotra, 1990; Prabhakar *et al.,* 1994, 1997; Sharma *et al.,* 1995; Srinivas *et al.,* 2007; Amin *et al*., 2011; Ali *et al*., 2011) although in one study pre-cervical torsion was predominant (Purohit *et al*., 2011b). A preponderance of right side uterine

torsion in buffaloes (95-98%); (Vasishta, 1983; Malhotra, 1990; Prabhakar *et al.,* 1994; Srinivas *et al.,* 2007; Purohit *et al*., 2011b) is postulated to be because of the absence of a muscular fold on right broad ligament in the buffalo (Singh, 1991; Brar *et al*., 2008c) and the presence of the rumen on the left side.

**Clinical Signs**

The usual clinical signs are the onset of labor without delivery of fetus and/or fetal membranes and later regression of parturition signs (Singh *et al*., 1978). The animal may show signs of mild discomfort. The animal may adopt a rocking horse stance (Purohit, 2006) and show mild colic pain and constipation (Ali *et al*., 2011). Partial anorexia, dullness and depression may be evident (Sharma *et al*., 1995; Ali *et al*., 2011). Restlessness and arching of back and colic may be seen in some buffaloes (Murty *et al*., 1999). In post-cervical uterine torsion one or both lips of the vulva are pulled in because of rotation of the entire birth canal. Vaginal examination reveals twisting of the vaginal mucous membranes and the hand cannot be passed deeper into the anterior vagina which has a conical end in torsion with a degree of 180° or more. In lesser degree torsions however, the fetus can sometimes be felt. The direction of the vaginal fold twisting is considered the evidence for the direction of torsion. On rectal examination, the twisted uterine horn can be felt and the broad ligament on the side of torsion is rotated downwards sometimes palpable under the uterus and the ligament on the opposite side is tense and stretched and crossing to the opposite side (Purohit *et al*., 2011a). The positive diagnosis of uterine torsion should thus, be based on the location of broad ligaments palpated per rectum. Animals at many locations may be presented to the obstetrician after varying times since the first onset of labour; hence, the clinical signs of shockand toxaemia may be evident depending upon the severity of torsion, previous handling, death of fetus and post-torsion complications.

**Clinical Pathology**

The pathophysiological alterations that occur following uterine torsion have been recently reviewed (Ghuman, 2010). These alterations have been proposed to be used as prognostic indicators for uterine torsion (Amer and Hashem, 2008; Amin *et al*., 2011; Ali *et al*., 2011). A brief mention is made of a few of the studied parameters

*Uterine changes:* Rotation of uterus compresses middle uterine vein which results in disturbances in venous circulation and increases carbon dioxide tension in the fetal blood. Consequently, the uncomfortable fetus makes vigorous movements that may further increase the degree of uterine torsion. With the increase in degree of torsion, there is compression of middle uterine artery and oxygen going to the fetus is decreased (Schultz *et al.,* 1975; Schönfelder *et al.,* 2005). Limited arterial perfusion and venous outflow in the twisted uterus leads to ischemia, hypoxia and cell death causing irreversible damage to the endometrium, myometrium and ultimately death of the fetus. Continued failure of blood supply results in loss of uterine wall elasticity and viability, and hence the uterine wall becomes necrosed, brittle, fragile and prone to rupture (Ghuman, 2010). Inflammatory changes can cause adhesions of uterus with surrounding abdominal tissues. Macroscopically, following rotation of the uterus, the color of the uterine wall changes from rose-pink to blue-purple to grey; indicating the progressive metabolic deterioration of the uterus (Schönfelder *et al.,* 2007a). Congestion,

edema and hematoma are present in the uterine ligaments, mesovarium and ovaries (Schönfelder *et al.,* 2007a). The damage to uterine tissue and its regenerative potential following the rotation of uterus can be accessed from plasma indicators, viz. haptoglobin and creatine kinase (Ghuman, 2010), which increase further following correction of torsion by the rolling of the dam (Ghuman *et al*., 2009).

*Blood components*: Clinical studies on the hematology and blood biochemistry of torsion affected buffaloes have shown marginal differences (Phogat *et al*., 1991; Singla *et al*., 1992). Uterine-torsion affected buffaloes suffer from normocytic normochromic anaemia (decrease in the RBCs, Hb and PCV) due to accumulation of metabolic waste products or relatively large loss of blood during abnormal parturition (Amer and Hashem 2008). The leukogram of these buffaloes reveals lymphocytopaenia, neutrophilia and monocytosis in association with eosinopenia (Pattabiraman and Pandit, 1980; Kaur and Singh, 1993; Malhotra *et al.,*1993; Amer and Hashem, 2008), which continues till the third day postpartum in surgically corrected cases of uterine torsion (Phogat *et al.,* 1991). A huge decrease in the total plasma proteins and albumin is consistently observed in torsion affected buffaloes (Pattabiraman and Pandit, 1980; Manju *et al.,* 1985; Khatri *et al.,* 1986; Singla *et al.,* 1992; Amer and Hashem, 2008). This hypoproteinemia is associated with liver malfunction and negative nitrogen balance because of reduced protein intake. Occurrence of torsion of the uterus is a highly stressful event as revealed by the huge increase in plasma cortisol, which increases further by 15-30% following detorsion of uterus through the rolling of dam (Ghuman *et al.,* 1996, 1997b; Amer and Hashem, 2008). Hyperglycemia in torsion-affected buffaloes is related to activation of stress axis and increased cortisol release, which leads to gluconeogenesis (Ghuman *et al.,* 1996; Amer and Hashem, 2008). In fact, plasma ketones are elevated in these buffaloes due to improper utilization of glucose (Ghuman *et al.,* 1996).

*Liver and renal functions*: Following uterine torsion and after its correction by detorsion or surgical treatment, the activities of aspartate amino transferase (AST), alanine amino transferase (ALT), glutamate dehydrogenase (GLDH), creatine phosphokinase (CK) and gamma glutamyl transferase (GGT) are increased (Pattabiraman and Pandit, 1980; Frazer, 1988; Phogat *et al.,* 1991; Singla *et al.,* 1992; Kaur and Singh, 1993; Kuhad *et al.,* 1996; Amer and Hashem, 2008), which usually becomes stabilized within 10 days after surgical treatment of uterine torsion (Schönfelder *et al.,* 2007b). The increase in plasma AST and muscle specific CK is attributed to great muscular exhaustion produced by strong abdominal contractions following uterine torsion (Kraft and Durr, 2005; Hussein and Abd Ellah, 2008). At the time of presentation of a uterine torsion case, a substantial increase in plasma urea and creatinine indicates poor prognosis (Frazer, 1988; Schönfelder *et al.,* 2007b; Amer and Hashem, 2008). In uterine torsion, ureters lying in the broad uterine ligaments are constricted; thus, the urine output reduces and renal functions may be affected (Schönfelder *et al.,* 2007b). Moreover, the presence of a stress-induced decrease in blood flow to kidneys, shock, dehydration and nephropathy resulting from toxic substances liberated by dead fetus may cause acute or chronic renal insufficiency, leading to decrease in the urea and creatinine elimination (Noakes *et al.,* 2001; Amer and Hashem, 2008).

**Diagnosis**

*History and external signs:* Diagnosis is

easy when the abnormal symptoms appear at the time of parturition. A typical history of a case of uterine torsion will indicate that the animal was about to calve, as exhibited by letdown of milk and relaxation of pelvic ligaments, but adequate time has passed and still there is neither the rupture of fetal water bags nor the appearance of the fetus from the vulvar lips (Prabhakar *et al.,* 1995). External signs of uterine torsion like displacement of upper commissure of the vulva towards inward, left or right, vulvar edema due to compression of the vaginal veins and lymphatic drainage, and a slight depression of lumbo-sacral vertebrae are not the consistent features (Ghuman, 2010).

Post-cervical torsions can be easily diagnosed by vaginal examination. About 66-96% of torsions are post-cervical, in which the twist of the rotated uterus extends caudal to the cervix and involves the anterior vagina in rotation. During rectal examination, attention should be paid to the course of broad ligaments to rule out pre-cervical torsion. In the normal pregnant animal, the broad ligaments can be palpated on the sides of the uterus, whereas in pre-cervical (and post-cervical) torsion, the orientation of broad ligaments is altered and these can be felt by crossed and twisted uterus. Accurate determination of the direction of torsion through rectal examination is necessary prior to making attempts at correction, as detorsion in the wrong direction will worsen the problem.

**Management of Dystocia**

Cases of uterine torsion must be considered an emergency and therapy must be instituted early. It is imperative to precisely evaluate the patient for the general condition before any handling efforts. The patient must be evaluated for the presence of toxemia and shock; as cases presented to the obstetrician after 36 h are likely to have one or more of these conditions. The authors suggest therapy that includes infusion of plenty of fluid along with corticosteroids and antibiotics whenever necessary to combat toxemia and shock before handling of cases presented beyond 36 h of delay. It is also usual to assess the type of previous handling or therapies provided including previous rolling given. In cases presented beyond 36-72 h, toxemia is likely to have set inas well as fetal death coupled with loss of fluid and uterine inertia. The incidence of uterine rupture is fairly high and therefore an examination should be made for this before treatment. Rarely uterine torsion can result in problems like jejunal incarceration (Frazer, 1988).The histamine levels are known to be high in buffaloes suffering from uterine torsion (Matharu and Prabhakar, 1999), hence, antihistaminics should be given. The management techniques in buffaloes suggested for detorting the rotated uterus include *rotation of the fetus and uterus per vaginum, rolling of the animal,* and *laparohysterotomy* (Purohit, 2006; Purohit *et al*., 2011a,b). The choice of the method to be adopted depends on the nature and intensity of the torsion, the viability of the fetus and the time lapse since dystocia onset.

*Rotation of the fetus* per vaginum is possible only in mild degrees of torsion where the obstetricians hand can touch the fetus and sufficient fluids are present in the uterus (Ghuman, 2010; Purohit *et al*., 2011b). The fetus is grasped by a bony prominence such as elbow, sternum or thigh and swung from side to side before being pushed right over in the opposite direction of torsion. If both fetal limbs are palpable they can be tied in the cuffs of *Caemmerer's torsion fork* or a *Kuenhs crutch* and an assistant can rotate them. If the manipulation is successful, the torsion will disappear and the vaginal folds will regain normal shape and the fetus can be delivered with little

difficulty. However, sufficient lubrication must be available in the birth canal and uterus before attempts at rotating the fetus are made using instruments. When sufficient time has passed since the onset of such a problem, the uterus will tightly contract around the fetus and detortion with this method is not possible. The success rate is high if dam is standing, the cervix is sufficiently dilated to grasp the fetus and the fetus is alive (Ghuman, 2010).

*Rolling the buffalo* utilizes the principle of rolling the animal around its uterus while the uterus remains static. It is one of the oldest and simple methods to relieve uterine torsion in buffaloes. The animal must be rolled preferably on grass with its head lower than the rear quarters. Vicious animals must be given a sedative. The animal is laid down in lateral recumbency on the same side to which the torsion is directed. The two hind legs are tied together with a rope (Ghuman, 2010; Purohit *et al*., 2011b). Both the fore legs are also tied together using a separate rope. The animal is rolled suddenly in the same direction as the torsion of the uterus to the other side. The rapidly rotating body of the buffalo overtakes the more slowly rotating gravid uterus. After the animal has been rolled to 180° her body must be brought back to the original position slowly so that she can be rolled once again. After two rollings, the birth canal should be examined to determine whether the torsion is corrected or not. If corrected properly, the spiral folds and stenosis of the birth canal would disappear and if the cervix is dilated, the fetus can be palpated with ease. Plenty of blood stained fluid comes out of the birth canal if the cervix is open, and this is sufficient evidence of torsion correction. If the torsion is not corrected, the rolling procedure should be repeated three or four times. If after four attempts, the torsion is not corrected, then other procedures for correction of torsion must be considered as uterine rupture can result due to violent rolling (Prabhakar *et al*., 1997; Singh *et al*., 1998). Although, torsion may be corrected by rolling in patients of not more than 36 h duration, the potential dangers of uterine rupture with continuous rolling must always be kept in mind. If the vaginal folds are increasing after a rolling, the rolling must be done on the opposite side. Sometimes, after the correction of torsion, it may take 12 h or more for the cervix to dilate and hence one should not take rapid action of removing the fetus after torsion correction without proper cervical dilation. Prostaglandin injections are suggested subsequent to torsion correction if the cervix is not dilated. Fetuses are delivered 12-24 h later in such cases.

Cervical dilation failure is commonly observed in buffaloes subsequent to correction of uterine torsion and is considered a major obstacle in vaginal fetal delivery (Prabhakar *et al*., 2007) especially in the presence of a dead fetus. Thrice hourly massage (15 minutes each) with 1 liter of sodium carboxy methyl cellulose has been suggested (Honparkhe *et al*., 2009) to achieve sufficient cervical dilation for fetal delivery. Prostaglandin injections have also been suggested subsequent to torsion correction when the fetus is alive and the cervix is not sufficiently dilated (Purohit *et al*., 2011b). Cesarean section is indicated if the cervix is found hard and lobulated subsequent to correction of torsion in buffaloes with dead fetus (Honparkhe *et al*., 2009). Histopathological observations on buffaloes affected with uterine torsion revealed progression towards severe inflammation (Singh *et al*., 2010). These inflammatory and necrotic changes in the cervix subsequent to torsion lead to failure of the cervix to dilate (Singh *et al*., 2010).

A modification of the rolling technique called Schaffer's method, has been described by

Arthur, (1966) and recommended widely (Sane *et al*., 1982; Arthur *et al*., 1996; Roberts, 2002) for detorsion of uterus in cows and buffaloes. In this method, a slightly flexible wooden plank of 9 to 12 feet long and 8 to 12 inches wide is placed on the recumbent animal's flank with the lower end of the plank on the ground. An assistant stands on the plank while the buffalo is slowly turned over by pulling the ropes. A slight modification of this method has been suggested for the buffalo (Prakash and Nanda, 1996). The advantages of this technique are that the plank fixes the uterus while the animal's body is turned and that, because the buffalo is turned slowly less assistance is required and it is easier for the veterinary surgeon to check the correct direction of the rolling by vaginal palpation (Purohit, 2006). Usually, the first rolling is successful (Arthur *et al*., 1996). Similar methods have been used with varying degrees of success in the buffalo (Pattabiraman *et al*., 1979; Prasad *et al*., 2000; Matharu and Prabhakar, 2001). In Egyptian buffaloes, Schaffer's method was described as the best method for rolling (El-Naggar, 1978; Samad *et al.,* 1981). However, it has been mentioned that the thick skin of Indian buffaloes causes skidding of the plank at the time of rolling. Moreover, pendulous abdomen of Indian buffalo warrants greater pressure for the fixation of the pregnant uterus. Therefore, modifications were made in Schaffer's method and the method is termed as Sharma's modified Schaffer's method (Singh and Nanda, 1996). It is considered that since buffaloes have a capacious abdomen more pressure is required on the free end of the plank that is being modulated by an assistant resulting in better detorsion compared to Schaffer's method (Singh and Nanda, 1996). Using this method, the detorsion rate in Indian buffaloes was 90% in comparison to 40% success rate achieved by Schaffer's method (Singh and Nanda 1996, Srinivas *et al.,* 2007). When torsion is pre-cervical and the durations were <36 h, 36-72 h or >72 h, the animals successfully detorted were 82, 100 or 67%, respectively (Prabhakar *et al.,* 1997). In buffaloes, detorsion followed by vaginal delivery is easily accomplished when torsion is <180°. With an increase in the degree of torsion, a greater number of rolls is required to detort the uterus and the likelihood of vaginal delivery is decreased (Amer and Hashem 2008). It is suggested that if the torsion is not relieved after three rolls, then failure should be admitted and surgery is indicated (Nanda *et al.,* 1991). Buffaloes subjected to injudicious rolling (>3 rolls) have least survival rate as compared to those where rolling was well planned (Dhaliwal *et al.,* 1991). Depending upon the prior handling of a case of torsion, buffaloes subjected to one to three rolls had a 44-78% survival rate, whereas those subjected to more than three rolls had a 35-56% survival rate (Dhaliwal *et al.,* 1991). In another study, the survival rates of buffaloes requiring 1-2 rolls and 3-4 rolls for complete detorsion of uterus were 85 and 43%, respectively (Ghuman *et al.,* 1997b). Excessive adrenal stimulation due to one to two extra rolls causes a slower decline in plasma cortisol during the post-detorsion period (Ghuman *et al.,* 1997b). In long standing cases of torsion (>72 h), with apparent reabsorption of milk and tightened pelvic ligaments, attempts to achieve detorsion of the uterus are usually unsuccessful due to development of adhesions between the uterus and the adjoining abdominal organs (Dhaliwal *et al.,* 1991). Detorsion of the uterus in these cases is not possible even after detachment of adhesions (Sharma *et al.,* 1995; Luthra and Khar, 1999). Both tissue anoxia and serosal injury following the torsion of uterus are important factors in promoting adhesion formation in the abdominal cavity (Henderson, 1982).

The number of rolls required in buffaloes is more (2.5) compared to cattle (1.0) (Pattabiraman *et al*., 1979) and vaginal delivery takes a longer time after detorsion. Buffaloes with fully dilated cervices at detorsion had maximum survival (Nanda *et al*., 1991; Matharu and Prabhakar, 2001) and detorsion failure occurs in 20% of the cases (Nanda *et al*., 1991). In the authors' experience, detorsion failure is common in cases presented beyond 36 h of delay and in animals where a dead emphysematous fetus is present or uterine adhesions or uterine rupture is present. Similar views have been expressed by other workers (Dhaliwal *et al*., 1993; Prasad *et al*., 1998). It is known that detorsion is difficult in the presence of a dead fetus (Prabhakar *et al*., 1994). Myometrial degeneration and endometrial damage is greater in cases of uterine torsion in which treatment has been delayed (Mandal *et al*., 2002).

*Laparohysterotomy* is suggested in cases of uterine torsion that fail to be corrected by rolling or in long standing cases where fetus is dead and uterine adhesions/ruptures are likely. The outcome of a caesarean when the fetus is dead and emphysematous can be grave. It is advisable to take care of the patient for the general condition before deciding to operate. Caesarean is a method of choice in cases presented with a closed cervix and dead fetus with subsided symptoms of parturition (Singh *et al*., 1978). It is better to administer plenty of fluid therapy, antibiotics and corticosteroids before starting the operation. Different operative sites suggested for caesarean include the right (Dhaliwal *et al*., 1988; Dhaliwal *et al*., 1993) or left flank, midline (on or parallel to linea alba), horizontal incision above arcus cruralis (Dhaliwal *et al*., 1993), or appropriate incision in the right (Sharma *et al*., 1995) or left lower flank (Singh and Dhaliwal, 1998; Murty *et al*., 1999; Prasad *et al*., 2000) or an oblique ventrolateral approach with the animal in right lateral recumbency (Purohit, 2006). The authors and a few other workers (Saxena *et al*., 1989; Varshney *et al*., 1992) consider the left oblique ventro lateral approach with the animal in right lateral recumbency as a better operative site as it results into minimum post operative complications. The anesthesia usually required is mild sedation with local infiltration. Many anesthetic combinations have been suggested for epidural analgesia including xylazine and medetomidine (Singh *et al*., 2009), xylazine and ketamine (Singh *et al*., 2006) or xylazine and detomidine (Tiwari *et al*., 1998); however, xylazine is used as a sedative during laparohysterotomy. Doses of 0.05 mg/kg of xylazine produces mild sedation (Peshin and Kumar, 1979; Alshara *et al*., 2000) in buffaloes; however, lower dosage is suggested as this drug induces marked salivation and bradycardia. Alternatively combining 0.04 mg/kg IM of atropine followed by 0.04 mg/kg of xylazine is suggested to minimize the side effects of xylazine (Khan *et al*., 2007).

During the laparohysterotomy, the uterus is brought to the site of incision by holding a fetal extremity and incised. The fetus is removed with due care. Because of the fetal death and the consequent uterine adhesions that develop in cases operated beyond 36 h, many times it is not possible to detort the uterus before the removal of the fetus. In rare instances, if the animal had uterine torsion, rupture of uterus can occur subsequent to attempts at correction of torsion by rolling. Such ruptures must be searched for during the operation and if possible repaired.

If the tear is not within approach, the best option is to inject 20-40 I.U oxytocin within the uterine wall at 3-4 or more locations to contract the uterus. The abdominal wound is closed routinely.

The major complications that can occur following uterine torsion are fetal and maternal

death (Nanda *et al*., 1991), uterine rupture, vaginal rupture (Dhaliwal *et al*., 1991; Singh and Dhaliwal, 1998) and poor fertility following correction of a long-standing case of torsion. Sometimes fatal peritonitis or expulsion of fetus in the abdominal cavity from a uterine rupture is possible. Uterine torsion affected buffaloes are under acute stress as suggested by elevated plasma cortisol and suppressed ruminal, liver and kidney functions (Ghuman *et al.,* 1996,1997b, 1998a). In addition, the process of detorsion further enhances adrenocortical activity and potentiates the existing stress on the dam. Attempts made to alleviate stress of uterine detorsion by administration of tranquilizers, viz. acepromazine, chlorpromazine or diazepam, before subjecting the dam to detorsion process have failed to achieve desired success as revealed by the regular monitoring of plasma cortisol (Ghuman *et al.,* 1997a). However, dexamethasone administration during the immediate post-partum period of successfully detorted animals decreases stress and thus increases the chances of survival of the dam (Sathya *et al.,* 2005). Moreover, stress-induced oxidative damage is reduced and post-dystocia convalescence is improved when antioxidants like vitamin E and selenium are administered to uterine torsion affected bovines (Sathya *et al.,* 2007). As revealed by plasma concentrations of liver enzymes in uterine torsion affected bovines, the first post-operative week should be critically observed and a liver protection therapy should be instituted during this period (Singh, 1991). Uterine torsion cases suffer from respiratory alkalosis without much metabolic alterations which stabilizes following detorsion (Ghuman *et al.,* 1998b). Determination of blood-gas and acid-base parameters followed by appropriate fluid and electrolyte therapy in torsion affected bovines prognosticate the chances of survival of dam as well as the calf. As a matter of critical care, monitoring blood-gas and acid-base status as well as electrolytes should be a mandatory procedure in torsion affected bovines (Ghuman *et al.,* 1998b). With the increase in duration of uterine torsion, plasma and blood volume decrease and the animal progresses towards dehydration and toxaemia; this suggests the requirement of immediate fluid therapy (Dhindsa *et al.,* 2005, 2007). Torsion cases can be resuscitated successfully using intravenous administration of a small amount of hypertonic saline and dextran-40 followed by oral administration of a large amount of fresh water. This is a quicker, more practical, easier and more effective method compared to intravenous administration of huge amounts of normal saline (Kumar *et al.,* 2009). Dextran-40 can maintain elevated plasma and blood volume for longer durations and thus decreases the degree of dehydration. In fact, alterations in plasma cortisol, albumin and glucose following administration of hypertonic saline, dextran-40 and oral fluids lead to alleviation of stress (Kumar *et al.,* 2009). Thus combating stress appears pivotal to the successful management of uterine torsion affected buffaloes.

## CONCLUSIONS

Based on published data it appears that uterine torsion is the single largest cause of dystocia in buffaloes during terminal gestation. It appears to originate because of inherently weaker broad ligaments, smaller quantity of fetal fluids and decrease in uterine tone and size coupled with inordinate fetal movements. Liver and kidney function tests can prognosticate the outcome of uterine torsion and early presentation to referral centers culminate in successful management of the condition with high dam and fetal survival.

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