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Blood Constituents of Buffaloes in Response to Prognosis and Duration of Uterine Torsion

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Abstract: The present study aimed to evaluate the changes in the total antioxidants capacity, biochemical and haematological constituents in relation to the duration and prognosis of Uterine Torsion (UT) in buffaloes. A total number of 16 buffaloes (*Bubalus bubalis*) were presented to the Veterinary Teaching Hospital, Assiut University, Egypt with a history of dystocia. Animals were classified according to the duration of uterine torsion into two groups: group I (Torsion was lasted for 12-24 h, n = 7) and group II (Torsion was lasted for 24-48 h, n = 9). Buffaloes also were classified according to the prognosis of uterine torsion into three groups: very good prognosis group (n = 4), good prognosis group (n = 7) and fair prognosis group (n = 3). Results revealed that there were significant decreases in serum total proteins, albumin and globulins levels in buffaloes that suffered from UT till 24 and 48 h and also in buffaloes with very good prognosis compared with the control group. Serum Aspartate aminotransferase (AST) and Creatinine phosphokinase (CK) activities were significantly higher in all buffaloes with UT compared with the control group. On the other hand, there was a significant increase in serum blood urea nitrogen and a significant decrease in serum phosphorus levels only in animals classified according to the prognosis. There was a significant increase in serum total antioxidants capacity in all buffaloes suffered from UT in relation to duration and prognosis of uterine torsion compared with the control group, associated with insignificant changes in serum malondialdehyde levels. In conclusion, serum AST, CK and total antioxidants can be used as indicators for the duration and prognosis of uterine torsion in water buffaloes.

Key words: Blood, buffaloes, duration, prognosis, uterine torsion

INTRODUCTION

Uterine torsion has been reported as a serious cause of dystocia in buffaloes (Murty *et al*., 1999; Nanda *et al*., 2003; Amer and Hashem, 2008) and cattle (Pearson, 1971; Frazer *et al*., 1996; Aubry *et al*., 2008), threatening the lives of both fetus and dam. It is observed more commonly in multiparous and advanced pregnant animals (Roberts, 1986). The etiology and pathogenesis of this condition is inadequately understood and remains open for speculation. A diverse list of contributing causes has been proposed including the anatomy, slipping, the manner in which the animal rises and the strong movements of the fetus during the first stage of labour (Pearson, 1971; Sloss and Duay, 1980; Manning *et al*., 1982; Roberts, 1986; Arthur *et al*., 1989). This anatomical arrangement permits an increased uterine mobility in late gestation and predispose to development of a uterine torsion (Roberts, 1986; Sloss and Duay, 1980). Frazer *et al*. (1996) suggested that uterine torsion in cows is ultimately of fetal origin as it tended to be associated with oversized male fetuses. Several researchers have supported the idea that active movements by a large fetus during late gestation and early stage of labor might precipitate rotation of the unstable uterus (Pearson, 1971; Baker, 1988; Arthur *et al*., 1989). Decreased amniotic fluid in relation to the size of fetus and uterus has been suggested as a very plausible explanation for the selective occurrence of uterine torsion in cows (Schonfelder and Sobiraj, 2005).

Oxidative stress results when reactive forms of oxygen are produced faster than they can be safely neutralized by antioxidant mechanisms (Sies, 1991; Abd Ellah, 2010) and/or from a decrease in antioxidant defense which may lead to damage of biological macromolecules and disruption of normal metabolism and physiology (Trevisan *et al*., 2001). This condition can contribute and/or lead to the onset of health disorders in

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animals (Miller *et al*., 1993). Some studies were carried out to investigate the biochemical and haematological changes associating uterine torsion in buffaloes but no one of them discussed those changes in relation to duration and prognosis of uterine torsion. On the other hand, oxidative stress is involved in the etiologies of certain disorders of dairy cattle as mastitis (Erskine *et al*., 1987; Parantainen *et al*., 1987), retained placenta and udder edema (Miller *et al*., 1993). However, studying the status of antioxidants and lipid peroxidation in blood of buffaloes with uterine torsion is lacking. The primary objective of the present study is to investigate the relationship between blood hematological, biochemical and antioxidants status and both duration and prognosis of torsion and to clarify their clinical significance upon handling of this serious condition.

MATERIALS AND METHODS

A total number of 16 buffalo-cows (*Bubalus bubalis*) (aged 5-10 years) were presented to the Veterinary Teaching Hospital at Assiut University in middle Egypt with a history of dystocia. Parity (primi or multiparous), stage of gestation (during pregnancy or at term), the duration of the condition (time from the appearance of clinical signs until submission) and the presenting clinical signs were recorded. Definite diagnosis of uterine torsion was achieved only in the Veterinary Hospitals after careful rectal n and vaginal examinations. The full term was defined as being within 7 days around the expected calving date (Frazer *et al*., 1996). In addition, 10 normal pregnant buffalo cows near term were kept as a control group.

Animals were classified according to the duration of uterine torsion into two groups: group 1 (12-24 h, n = 7) and group 2, 24-48 h, n = 9). Based on the general condition of the animals, clinical examination and duration of torsion, buffaloes were given the following prognostic terms; very good (n = 4), good (n = 7) or fair (n = 5) condition.

Blood samples were collected from all the buffalo cows with uterine torsion (n = 16) and from control group (n = 10). Two types of blood samples were collected from all animals, one in plain vacutainer tube and the other in vacutainer tube with EDTA. Blood samples were prepared directly after collection.

Serum was separated from the plain tube by centrifugation for 10 min at 3000 rpm and was immediately frozen for future analyses (within 2 weeks). Serum concentrations of total proteins, albumin, creatinine, Blood Urea Nitrogen (BUN), calcium, phosphorus, magnesium and serum activities of Aspartate aminotransferase (AST) and Creatine phosphokinase (CK) were measured using commercial test kits supplied by Spectrum Diagnostics (Egyptian Co., for Biotechnology, Obour City Industrial Area, Cairo-Egypt) and by Digital VIS/Ultraviolet Spectrophotometer (Cecil instruments, Cambridge, England, Series No. 52.232).

The whole blood sample was used for measuring Total Red Blood Cells (TRBCs), Hemoglobin concentration (HB), hematocrit (PCV), Mean Corpuscular Volume (MCV), Mean Corpuscular Hemoglobin (MCH), Mean Corpuscular Hemoglobin Concentration (MCHC), Total White Blood Cells (TWBCs) and differential leucocytic counts, hematological analyses was performed directly after collection using automatic blood cells counter (Medonic Vet. CA 620, Sweden).

Serum concentrations of Total Antioxidants Capacity (TAC, mM/L) and Malondialdehyde (MDA, nmol/mL) were measured using commercial test kits supplied by Bio-Diagnostics (Cairo, Egypt).

Statistical analysis: Statistical significance was determined by the analysis of variance using Statistical Package of the Social Sciences for Windows (SPSS, Version 10.0, Chicago, IL, USA). Groups were tested for difference using analysis of variance LSD Post-hoc test. Statistically significant differences were determined at $p \leq 0.05$.

RESULTS AND DISCUSSION

The main clinical signs of torsion included straining or colic for prolonged time, reduction in feed intake and constipation. Mortality rate was high in fetus 13/16 (81.25%), maternal mortality rate was 3/16 (18.75%). Surgical correction of torsion was done in two cases, torsion was corrected through rolling once or twice in the remaining 14 cases.

Changes in serum biochemical and haematological constituents in relation to duration of torsion: There were significant decreases in serum total proteins, albumin and globulins levels in buffaloes that suffered from uterine torsion for duration of 24 and 48 h compared with the control group. Serum AST and CK activities were significantly higher in buffaloes with uterine torsion till 24 and 48 h from the onset of torsion compared with the control group. Haematological constituents showed significant increases in neutrophis, monocytes and band cells count in buffaloes with uterine torsion at 24 h from the onset of torsion compared with the control group (Table 1).

Changes in serum biochemical and haematological constituents in relation to prognosis: There was a significant decrease in total proteins, albumin and globulins in buffaloes with very good prognosis compared with the control group. Serum AST and CK activities were significantly higher in buffaloes with very good, good and fair prognosis compared with the control group. BUN showed a significant increase in buffaloes with very good, good and fair prognosis compared with the control group. Serum phosphorus showed a significant decrease in buffaloes with very good, good and fair prognosis compared with the control group. Haematological constituents showed significant increase in monocytes count in buffaloes with fair prognosis (Table 2).

Table 1: Changes in serum biochemical and haematological constituents in relation to duration of uterine torsion in buffaloes

		Duration of torsion	
Parameters	Control (No. = 10)	12-24 h (No. = 7)	24-48 h (No. = 9)
Total proteins (g L^{-1})	77.9±5.9^{a}	64.5±10.1^{b}	66.5±7.9^{b}
Albumin (g L^{-1})	25.72±3.43^{a}	31.0±3.1^{b}	29.7±3.7^{b}
Globulins (g L^{-1})	52.2±6.5^{a}	32.6±10.2^{b}	36.7±11.6^{b}
A/G ratio	0.5±0.1^{a}	1.0±0.3^{b}	0.9±0.3^{b}
AST (U L^{-1})	72.8±7.2^{a}	177.8±60.3^{b}	180.1±78.2^{b}
CK (U L^{-1})	20.3±11.0^{a}	368.5±384.2^{b}	492.6±321.6^{b}
BUN (mmol L^{-1})	2.9±0.8^{a}	4.2±1.8^{a}	4.7±1.9^{a}
Creatinine (mmol L^{-1})	93.1±27.2^{a}	146.3±81.9^{a}	99.2±21.6^{a}
Phosphorus (mmol L^{-1})	1.6±0.6^{a}	0.9±0.4^{a}	0.9±0.3^{a}
Magnesium (mmol L^{-1})	0.7±0.3^{a}	0.9±0.3^{a}	0.8±0.2^{a}
Calcium (mmol L^{-1})	1.6±0.5^{a}	1.7±0.6^{a}	1.7±0.6^{a}
RBCS (×10^{6}/mm^{3})	5.9±.1.9^{a}	6.6±0.7^{a}	6.5±3.2^{a}
HB (g dL^{-1})	11.8±2.3^{a}	11.7±1.2^{a}	9.9±2.1^{a}
PCV (%)	32.1±9.3^{a}	39.0±4.4^{a}	36.4±4.2^{a}
MCV (fl)	52.8±3.5^{a}	59.4±2.5^{a}	63.9±24.0^{a}
MCH (pg)	20.4±2.6^{a}	17.9±1.8^{a}	17.5±6.7^{a}
MCHC (%)	38.3±4.1^{a}	30.2±2.0^{b}	28.1±8.6^{b}
W^{b}cs (×10^{3})	8.6±3.7^{ab}	11.6±3.0^{a}	6.3±1.7^{b}
Neutrophils (×10^{3}/mm^{3})	4.4±1.7^{ac}	7.2±1.5^{b}	3.5±1.4^{c}
Lymphocytes (×10^{3}/mm^{3})	3.9±1.9^{a}	3.7±1.9^{a}	2.2±0.5^{a}
Eosinophils (×10^{3}/mm^{3})	0.201±0.140^{a}	0.127±0.148^{a}	0.102±0.204^{a}
Monocytes (×10^{3}/mm^{3})	0.080±0.069^{ac}	0.457±0.325^{b}	0.156±0.150^{c}
Band cells (×10^{3}/mm^{3})	0.0±0.0^{a}	0.063±0.074^{b}	0.017±0.034^{ab}

Data expressed as Mean±SD, value followed by different letter is significant at $p<0.05$

Serum Total Antioxidants Capacity (TAC, mM/L): There were significant increases in serum TAC in all buffaloes suffered from uterine torsion in relation to duration and prognosis of torsion compared with the control group (Table 3 and 4).

Serum Malondialdehyde (MDA, nmol/mL): There were insignificant changes in serum MDA levels in all buffaloes suffered from uterine torsion in relation to duration and prognosis of torsion compared with the control group (Table 3 and 4).

The significant hypoproteinaemia after 24 and 48 h from the onset of torsion may be attributed to hypoalbuminaemia and hypoglobulinaemia. The decrease in total proteins or albumin as the animals approach birth may be the cause of physiological oedema occurring at this time or as a result of bloody fluid loss at birth (Kanekeo *et al*., 1997). Moreover, hypoalbuminemia was probably associated with liver malfunction (Rowlands *et al*., 1980). Furthermore, albumin is mildly

Table 2: Changes in serum biochemical and haematological constituents in relation to prognosis of uterine torsion in buffaloes

		Prognosis		
Parameters	Control (No. = 10)	Very good (No. = 4)	Good (No. = 7)	Fair (No. = 5)
Total proteins (g L^{-1})	77.9±5.9^{a}	61.8±5.4^{b}	68.4±9.0^{ab}	73.72±17.8^{ab}
Albumin (g L^{-1})	25.7±3.4^{a}	30.9±1.8^{b}	30.7±4.1^{b}	29.3±3.9^{ab}
Globulins (g L^{-1})	52.2±6.5^{a}	30.9±5.8^{b}	37.6±12.1^{b}	43.2±22.4^{ab}
A/G ratio	0.5±0.1^{a}	1.0±0.2^{b}	0.9±0.4^{b}	0.8±0.3^{ab}
AST (U L^{-1})	72.8±7.2^{a}	253.6±41.7^{b}	176.8±63.1^{b}	179.67±78.8^{b}
CK (U L^{-1})	20.3±11.0^{a}	860.3±82.4^{b}	208.1±105.3^{c}	295.32±79.4^{c}
BUN (mmol L^{-1})	2.95±0.8^{a}	5.9±1.7^{b}	3.9±1.7^{ab}	4.67±1.9^{ab}
Creatinine (mmol L^{-1})	93.14±27.2^{a}	155.8±34.5^{a}	121.6±83.2^{a}	110.76±27.7^{a}
Phosphorus (mmol L^{-1})	1.59±0.6^{a}	0.5±0.2^{b}	1.0±0.2^{b}	0.9±0.2^{b}
Magnesium (mmol L^{-1})	0.7±0.3^{a}	1.18±0.4^{a}	0.9±0.3^{a}	0.9±0.2^{a}
Calcium (mmol L^{-1})	1.6±0.5^{a}	1.7±0.8^{a}	1.5±0.6^{a}	1.6±0.6^{a}
RBCS (×10^{6}/mm^{3})	5.9±1.9^{a}	5.9±1.6^{a}	6.8±2.6^{a}	6.6±3.2^{a}
HB (g dL^{-1})	11.8±2.3^{a}	11.1±0.7^{a}	11.1±2.2^{a}	10.5±1.2^{a}
PCV (%)	32.1±9.3^{a}	38.3±3.1^{a}	38.4±5.5^{a}	33.6±4.4^{a}
MCV (fl)	52.8±3.5^{a}	67.3±13.8^{a}	60.2±16.6^{a}	57.0±21.1^{a}
MCH (pg)	20.4±2.6^{a}	20.0±6.9^{a}	17.3±4.8^{a}	18.1±6.6^{a}
MCHC (%)	38.3±4.1^{a}	29.2±3.9^{ab}	29.7±8.6^{b}	31.9±7.9^{ab}
Wbcs (×10^{3})	8.6±3.7^{a}	10.4±1.5^{a}	9.1±4.5^{a}	9.1±3.5^{a}
Neutrophils (×10^{3}/mm^{3})	4.4±1.7^{a}	6.7±1.1^{a}	5.6±3.4^{a}	5.2±1.7^{a}
Lymphocytes (×10^{3}/mm^{3})	3.9±1.9^{a}	2.8±1.1^{a}	3.08±1.0^{a}	2.8±1.7^{a}
Eosinophils (×10^{3}/mm^{3})	0.201±0.140^{a}	0.339±0.409^{ab}	0.0±0.0^{b}	0.173±0.206^{ab}
Monocytes (x10^{3}/mm^{3})	0.080±0.069^{a}	0.407±0.058^{ab}	0.327±0.523^{ab}	0.490±0.347^{b}
Band cells (x10^{3}/mm^{3})	0.0±0.0^{ac}	0.142±0.074^{b}	0.030±0.061^{c}	0.052±0.067^{c}

Data expressed as Mean±SD, value followed by different letter is significant at $p<0.05$

Table 3: Changes in serum TAC and MDA in relation to duration of torsion

		Duration of torsion	
Serum	Control (No. = 10)	12-24 h (No. = 7)	24-48 h (No. = 9)
TAC (mM L^{-1})	0.1±0.01[a]	0.2±0.1[b]	0.3±0.02[b]
MDA (nmol mL^{-1})	3.5±0.80[a]	5.4±2.6[a]	5.9±3.03[a]

Table 4: Changes in serum TAC and MDA in relation to prognosis

		Prognosis		
Parameters	Control (No. = 10)	Very good (No. = 4)	Good (No. = 7)	Fair (No. = 5)
TAC (mM L^{-1})	0.1±0.01[a]	0.2±0.1[b]	0.3±0.1[b]	0.2±0.1[ab]
MDA (nmol mL^{-1})	3.5±0.80[a]	5.6±0.8[a]	4.8±2.4[a]	6.2±3.3[a]

Data expressed as mean±SD. In each row different letter means significant ($p<0.05$)

decreased in acute tissue injury or inflammation as a negative acute phase reactant (Evans and Duncan, 2003). Similar results were obtained in mare (Perkins *et al*., 1992) and in buffaloes (Patel *et al*., 1999; El-Din *et al*., 2000). It was reported that such reduction in the concentration of maternal plasma proteins coincided with the rapid increase in the uterine weight and its contents mainly the fetus, fetal fluids and fetal membranes (Shehata *et al*., 1991).

The significant increase in serum AST ($p<0.01$) and CK ($p<0.01$) activities in buffaloes with uterine torsion compared with the control group may be attributed to muscle exertion produced by strong abdominal contractions (Kraft and Durr, 2005). This is in coincidence with the results by El-Din Zain *et al*. (1997), Oliveira *et al*. (1998), Hoeben *et al*. (2000) and Ali *et al*. (2011). The increases in enzymatic activities of AST and CK are often a reflection of great muscular effort or damage results in leakage of such necrotic or damaged uterine cells (Coles, 1986; Kraft and Durr, 2005) which is exerted during the process of calving (Farrag *et al*., 1984). The increase of AST was necessary for accelerating the rate of metabolism and protein biosynthesis needed for foetal growth as well as milk production (Arthur *et al*., 1989). In addition, the increase in AST levels may be related to the hormonal changes that occurred during the last stages of gestation. At the same time, the nutritive differences such as imbalance in proteins and carbohydrates or insufficient crude fiber content resulted in upsetting the proper function of the rumen causing more or less harmful energetic deficiency and damage of liver leading to increase of AST activity (Younis, 1990).

The increased levels of urea in this study could be related to stress condition exerted on the affected buffaloes with concomitantly reduced blood flow to kidneys and reproductive tract. Furthermore, results from this study may be attributed to nephropathy resulted from toxic substances liberated from dead fetuses in some cases of uterine torsion (Arthur *et al*., 1989) or may be due to concomitant breakdown of tissues during gluconeogenesis under effect of increased cortisol level (Payne and Payne, 1987). The present results are in coincidence with those reported in previous studies (Perkins *et al*., 1992; Manal *et al*., 1999).

The significant decrease in serum phosphorus level ($p<0.01$) may be attributed to the assimilation of phosphorus in the process of excessive energy production needed for muscle contraction as the body needs phosphorus to convert ADP to ATP (Mayes and Botham, 2003). Moreover, hypophosphatemia may be the result of a combination of factors as transcellular shift of phosphorus (from extracellular volume to either soft tissues or bones), poor dietary intake especially when associated with impaired gastro-intestinal absorption or diarrhea and increased phosphate excretion resulting from renal and non-renal causes (Berkelhammer and Bear, 1984). No significant change was observed in serum calcium level between control and diseased buffaloes, comparing calcium level from the present study with the reference calcium level (2.43-3.1 mmol L^{-1}) reported by Kanekeo *et al*. (1997) indicated that serum calcium from the present study is lower than reference level this may be attributed to the drop of calcium level near parturition.

Haematological constituents showed significant increases in neutrophis, monocytes and band cells count in buffaloes with uterine torsion till 24 h compared with the control group (Table 1). Similar results were obtained in uterine torsion in cows (Rakuljic-Zelov and Zadnik, 2002), the researchers reported that the hematological changes occurred as a consequence of uterine torsion and may attributed to increased ACTH and adrenalin secretion (Durdevic, 1986). Increase number of the monocytes indicating long standing uterine infection that observed in many cases while appearance of the band cells is the consequence of the toxemic status of some affected buffaloes (Feldman *et al*., 2000; Hoffman *et al*., 2004).

Measuring TAC gives a complete picture of the oxidative stress state in the body by looking at the levels of markers for the ongoing oxidative damage in serum (Arguelles *et al*., 2004). In the present study, in spite of the significant increase in TAC there was insignificant change in serum MDA level, the present study demonstrated a state of increased oxidative stress in the blood of buffaloes with uterine torsion with absence of blood lipid peroxidation products.

CONCLUSION

Deterioration in serum biochemical constituents starts at 24 h from the onset of torsion. It is preferable to classify animals depending on prognosis of uterine

torsion. It seemed that uterine torsion adversely affected the liver and kidney functions with a state of increased oxidative stress in the blood of buffaloes with UT.

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