Biochemical and haematological studies on clinical and sub-clinical cases of hypophosphatemia among buffaloes on Menoufia Governorate

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Abstract

This study was designed to investigate the biochemical and haematological alteration on buffaloes suffering from clinical and sub clinical hypophosphatemia which excessively feed on Trifolium alexandrium (Barseem) during green season in Menoufia Governorate. Blood samples were collected from 15 clinically healthy buffaloes used as control group and 12 animals showing signs of pica and preferring dry food instead of green Barseem, these animals were considered as subclinical cases of hypophosphatemia. Other 10 animals showed signs of anaemia, jaundice, recumbency, laboured breathing, constipation, and haemoglobinuria, these animals were considered as clinical cases of hypophosphatemia. The haemogram results showed decreased erythrocytic count, haemoglobin concentration and packed cell volume, with significantly the total leukocytosis and neutrophilia combined by relative lymphopenia and monocytopenia in clinical cases of hypophosphatemia. The biochemical studies revealed that hypophosphatemia accompanied by significant decrease in red cell membrane phospholipids, serum total protein, albumin, globulin, phosphorus, iron and total iron binding capacity. Urea, creatinin, AST and ALT were significantly increased, while calcium, magnesium and A/G ratio were not changed. Urine analysis also discussed in both clinical and sub-clinical cases of hypophosphatemic buffaloes. The osmotic fragility of erythrocytes was significantly higher in clinical cases of hypophosphatemia.

Key words: (Biochemical- haematological- hypophosphatemia- buffaloes)

Introduction

Hypophosphatemia is a documented consistently in affected animals (Chugh et al., 1996 and Hussein et al., 1991). Parturient haemoglobinuria is one of the major and economically important diseases of dairy animals in Pakistan, India and elsewhere in the world (MacWilliums et al., 1982; Chugh et al., 1996; Pirzada and Hussain, 1988). It is an acute disease of high yielding buffaloes and cows characterized by hypophosphatemia, intravascular haemolysis, haemoglobinuria and anemia (Radostitis et al., 2000). In Egypt, Awad and Abdel-Latif (1963) recorded for the first time a syndrome simulating postparturient haemoglobinuria in buffaloes (hypophosphatemia) which occurs during pregnancy and seemed to be associated with prolonged feeding on Trifolium alexandrium (Barseem). On the other hand Nagpal et al., (1968) observed that the incidence of haemoglobinuria syndrome occurs in post-parturient period in Indian buffaloes.

Dietary phosphorus deficiency and/or rations containing cruciferous plants suspected causes of sever hypophosphatemia and have been associated haemolytic anemia in cows, MacWillium et al., (1982).In post - partu haemoglobinuria, red blood cells are lysed most probably due to their expo to altered function and structure, a loss of normal deformability and increa osmotic fragility of red blood cells due to impaired glycolysis and ATP synth Wang et al (1985). Sever stress due to intravascular haemolysis in parturient haemoglobinuria affects the structural and functional activate different body organs, mainly liver, heart and kidneys Akhtar et al. (2008).
Red cell membrane phospholipids level was found to be reduced in buffaloes with postpartieurent hemoglobinuria compared with healthy an (Rana and Bhardwaj, 1990b).
Therefore, the present study was, planned to investigate the cl haematological and biochemical aspect of hypophosphatemia along with analysis in buffaloes on Menoufia Governorate.

Material and methods

1- Animals

The present investigation was carried out in the villages of Mer Governorate on 22 diseased buffaloes aged from 3-8 years old at the late of pregnancy with similar history of excessive prolonged feeding on bar with signs of decreased appetite, preferring of dry feeds and refused bar feeding. Ten of them voided reddish to coffee coloured and foamy urine an group was considered as clinical cases of hypophosphatemia. Othe buffaloes not voided coloured urine and considered as subclinical cas hypophosphatemia other 15 clinically healthy buffaloes were used as c group.

2- Blood Samples:

The blood samples were collected from jugular vein, the first sa was collected on EDTA as anticoagulant for hematological studies, se blood sample was collected on heparin for phospholipid estimation o membrane of erythrocytes, and the third blood sample was collected w anticoagulant for separation of serum. Leishman's stain was used in st blood smears for differential leukocytic count. The collected sera were us determination of serum calcium (Gitelman, 1967), phosphorus and magn (Burite and Ashowood, 2001), total protein (Peters, 1986), Albumin (Roc 1965), serum aspartate aminotransferase (AST) and alanine aminotransf (ALT) (Reitman and frankle, 1957), urea (Patton and Crouch, 1977), crea (Bartels et al., 1972), serum total bilirubin (Jendrassik, et al., 1938), iron (E 1984) and total iron binding capacity (Fairbanks and Klee, 1987).

3 - Estimation of RBC membrane phospholipids according to (Connert 1961)

3.1- Processing of RBC

The blood were collected in purple colure tube was centrifuged for 10 m and the supernatant (plasma) was discarded. Two ml of normal saline added to the tubes, mixed and centrifuged at 5000 rpm for 10 minutes supernatant was decanted and this procedure was repeated twice. Then 0

tris HCL buffer, pH 7.4 (isotonic) was added and centrifuged for 5 minutes. This procedure was repeated three times. The clear cells obtained were suspended in a hypotonic buffer and stored at 4 °C for four hours. Afterwards the cells were washed again with hypotonic buffer and then centrifuged at 5,000 rpm and 10,000 rpm for 30 and 20 minutes, respectively. This procedure was continued until the solution became colourless. Two drops of isotonic buffer were added to the solution and it was homogenized to get the red cell membrane. This was used for the extraction of lipids

3.2- Extraction of lipids

The red cell membrane collected was again centrifuged for10 minutes at 15,000 rpm. The supernatant was decanted and the pellet obtained was suspended in 1 ml of methanol and homogenized. The solution was made up to 5 ml with the same methanol and centrifuged for 15 minutes at the same speed. The supernatant was decanted, added 14 ml of chloroform to the tube and transferred in to a flat bottom flask. The content of this flask was evaporated in a fume hood. To the evaporated content, added 5 ml of chloroform- methanol mixture to dissolve the solid and the resulting solution is poured into a labelled centrifuge tube and covered with paraffin. These tubes are centrifuged at 6000 rpm and two differed layers of fluid were visible. The upper aqueous layer was aspirated and discarded while the lower layer was retained for lipid testing. Aliquots of this layer were used for Colorimetric determination of phospholipids using Bio-diagnostic kits- Egypt.

4- Urine samples

Urine samples were collected from each animal using a sterilised catheter into clean, dry sterilized brown coloured glass bottles, and processed for physical, microscopic and biochemical analysis. The urine samples were examined for colour, odour, ph, protein, sugar, blood and keton bodies using urine strips. Microscopical examination of urine samples were done to identify erythrocytes, pus cells, epithelial cells, casts, proteins crystals and bacteria according to Kelly, (1984)Urine samples were examined for calcium (Gitelman, 1967), phosphorus and magnesium (Burite and Ashowood, 2001), urea (Patton and Crouch, 1977) and creatinine (Bartels et al., 1972).

5- Statistical analysis:

The obtained data were analyzed using student t-test as described by Petrie and Watson., (1999).

Result

The clinical examination of the clinical cases of hypophosphatemic buffaloes were haemoglobinuria decreased appetite, normal body temperature range (38-39C°), accelerated respiration, increased pulse rats and pale mucous membrane., The subclinical cases showed decreased appetite and refused to eat barseem. The present data (table 1) revealed a highly significant decrease ($P<0.05$) in RBCs membrane phospholipids, haemoglobin, erythrocytic count and packed all volume, associated with significant decrease in lymphocyte and monocyte and highly significant increase in osmotic fragility, total leukocytic count and neutrophil percentage in clinical buffaloes of hypophosphatemia.

The present data (table 2) revealed a highly significant decrease of phosphorus in both clinical and sub clinical group, highly significant decrease of

iron and total iron binding capacity in clinical group and significant decreas sub clinical group.The data (Table 3) showed highly significant decrease in t protein, albumin and globulin in clinical group and significant decrease in clinical group of hypophosphatemia.

The current data (Table 4) showed a highly significant increase in GOT, G Urea, creatinin and total bilirubin in clinical cases and significant increase in clinical cases of hypophosphatemia.Biochemical examination of urine sam (Table 5) showed highly significant decrease of urine calcium, phospho magnesium, urea and creatinin in clinical cases but significant decrease in th parameters within sub clinical cases of hpophosphatemia. Urine haemoglobinuric buffaloes was positive of hemoglobin and albumin but nega for sugar, keton bodies, bile salt and bile pigment. Microscopic examinatio urine of diseased buffaloes revealed no intact erythrocytes but few epith cells and amorphous phosphate were found.

Table (1) ***Hemogram of the healthy control, sub clinical and clinical cases of hypophosphatemia in buffaloes***

Items / Groups	Hb gm/dl	RBCs 10^6/ mm^3	PCV %	WBCs 10^3/ mm^3	N %	L %	M %	E %	B %	RBCs Fragility	phospholipids mg/ gm RBCs membrane
Normal Group	12.24 ± 0.32	7.8 ± 0.29	34.2 ± 0.64	7.045 ± 1.625	35.5 ± 0.42	50.16 ± 0.47	5 ± 0.56	9.16 ± 0.7	0.18 ± 0.16	2.4 ± 0.16	1.47 ± 0.036
Sub clinical Group	11.14 ± 0.17	7.33 ± 0.11	32 ± 0.33	7.650 ± 1.81	40.6 ± 0.55	48.1 ± 0.47	3.16 ± 0.47	8.16 ± 0.6	0.16 ± 0.16	3.3 ± 0.17	1.31 ± 0.025
Clinical Group	5.7** ± 0.17	4.428** ± 0.17	19** ± 0.36	9.316** ± 2.78	46.16** ± 0.8	43.6* ± 0.98	2.8* ± 0.31	7.3 ± 0.6	0.33 ± 0.2	6.3** ± 0.15	0.94** ± 0.02

* Significant variation at (P ≤0.01) ** Highly Significant Variation at (P ≤0.001)

Table (2) *Major and minor element in serum of the healthy control, sub clinical clinical cases of hypophosphatemia in buffaloes*

Items / Groups	Ca Mg/dl	Ph Mg/dl	Mg Mg/dl	Iron	TIBC
Normal Group	8.84 ± 0.16	6.5 ± 0.17	2.63 ± 0.07	181.2 ± 3.05	413.7 ± 34.7
Sub clinical Group	8.24 ± 0.1	3.6** ± 0.1	2.53 ± 0.06	167.3* ± 2.37	396.5* ± 2.75
Clinical group	8.03 ± 0.13	1.9** ± 0.15	2.43 ± 0.03	150.2** ± 2.55	36.73** ± 5.2

* Significant variation at (P ≤0.01) * * Highly Significant Variation at (P ≤0.00

Table (3) Serum *proteins of the healthy control, sub clinical and clinical case hypophosphatemia in buffaloes*

Items / Groups	T.Protein Gm/dl.	Albumin Gm/dl	Globulin Gm/dl	A/G ratio
Normal Group	7.08 ± 0.12	3.77 ± 6.06	3.3 ± 0.1	1.13 ± 0.04
Subclinical Group	6.7 ± 0.05	3.5* ± 0.05	3.18 ± 0.04	1.1 ± 0.02
Clinical group	3.03** ± 0.09	3.2** ± 0.06	2.8** ± 0.06	1.14 ± 0.03

* Significant variation at (P ≤0.01) * * Highly Significant Variation at (P ≤0)

Table (4) Liver and kidney function in *healthy control, sub clinical and clinical c of hypophosphatemia in buffaloes*

Items / Groups	AST 1u/L	ALT 1u/L	Urea Mg/dl	Creatinine Mg/dl	T. bilirubin Mg/dl
Normal Group	61.8 ± 2.05	29.9 ± 1.36	18.4 ± 0.38	1.02 ± 0.04	0.62 ± 0.15
Subclinical Group	7.1 ± 2.2*	34.4 ± 1.07	23.6 ± 0.57*	1.2 ± 0.04	1.18 ± 0.19
Clinical group	125.1 ± 2.95**	39.5 ± 0.88**	42 ± 0.65**	1.76 ± 0.07**	3.8 ± 0.36**

* Significant variation at (P ≤0.01) * * Highly Significant Variation at (P ≤0.001)

Table (5): Some biochemical parameter in urine of *healthy control, sub clinical and c*

Items / Groups	Ca Mg/Kg/day	Ph Mg/Kg/day	Mg Mg/Kg/day	Urea Mg/Kg/day	Creatinin Mg/Kg/da
Normal Group	1.95 ± 0.03	24.6 ± 0.84	2.25 ± 0.09	17.2 ± 0.42	17.3 ± 1.24
Subclinical Group	0.79** ± 0.04	20* ± 0.6	2.13 ± 0.06	14.25* ± 0.35	5.86** 0.44
Clinical group	0.42** ± 0.02	15.9** ± 0.56	2.01* ± 0.04	12.9** ± 0.3	3.67** ± 0.13

cases of hypophosphatemia in buffaloes

* Significant variation at (P ≤0.01) * * Highly Significant Variation at (P ≤

Discussion

From history, clinical, heamatological and biochemical analysis of serum and urine affected animals, hypophosphatemia was seems to be the main cause of haemogl in buffaloes. These results were in agreement with there reported by Wrigth and Woo (1958), Awad and Abdel-Latif (1963), Omran et al., (1987) and Abdel - maksoud and - Raoef (1998).The most prominent clinical signs in case of hypophosphatemia in bu were urination of red to coffee coloured urine depending on the disease severi stage, other symptoms were dullness, anemia, dehydration and sever decrease production (stockdale et al., 2005). The red to coffee urine and anemia have attributed to acute intravascular haemolysis (Dhillon et al., 1972). Respiratory and rats were significantly accelerated, while ruminal motility was significantly wea reduced in both tone and frequency in hypophosphatemic buffaloes as reported by (al., 1988). In advanced stage of the disease, affected buffaloes showed marked ar that was accompanied with ruminal stases and sever straining while defecatio excessive formation of haemosidrin and its deposition in the gastrointestinal muc diseased buffaloes could be responsible for gastrointestinal disturbances like r stasis, (Radostitis et al, 2000).

Regarding to haematological picture, There were highly significant decrease in eryth count, haemoglobin concentration and haematocrite in hypophosphatemic bu indicates haemolysis which agreed with results were obtained by Benjamin, Pandey and Misra., (1987), Smith (2000), Digraskar, et al., (1991) and Akhtar (2007).The intravascular haemolysis occurs due to impaired glycolytic pathwa depletion of ATP in erythrocytes which results from phosphorus deficiency. Sub concentration of ATP predisposes red blood cell to alter their function and structure, normal formability and increase in osmotic fragility that lead to haemolysis Wang (1985) and Ogawa et al., (1989).There was an increase in total leukocytic cou neutrophil and a decrease in lymphocyte and monocyte percentage in clinical c hypophosphatemia which could be attributed to the endogenous release corticos increased stress due that results in increased neutrophils and depressed lymp Singari, et al., (1991) and Akhtar et al., (2007).

Regarding to red cell membrane phospholipids, there was a highly sig decrease in red cell membrane phospholipids in clinical cases of hypophosphatemi was agreement with the result obtained by Rana and Bhardwaj, 1990b.

Significantly decreased serum phosphorus in hypophosphatemic buffaloes was in the present study also had been reported by Kurundkar et al., (1981). Heavy di phosphorus through milk particularly in high milk producing animals hypophosphatemia (Bhikane et al., 1995). Fodders grown on soils that were deficient in phosphorus consequently low in phosphorus content and thereby feeding on such fodders can lead to hypophosphatemia (Smith, 2000).
Serum calcium and magnesium usually remain within normal limits as reporte Williums et al., 1982 and Brain and Eric, 1996.The highly significant decrease total protein, albumin and globulin in clinical hypophosphatemic group was nearly results that obtained by Abdl-Aal (1977) and Abdel-Maksoud and Abdel-Raoef (1 low values could be attributed to the loss of protein from the destructed erythro its release in the urine as albuminuria also this may be due to tubular degen kidney during haemoglobinuria or poor nutritional status and/or related to depriva diet as reported by Kurundikar et al., (1981) and Abdel-Salam et al., (1994).

The decreases in both serum iron and total iron binding capacity in both cli subclinical hypophosphatemic buffaloes were similar to that recorded results by maksoud and Abdel - Raoef., 1998). The end result was the development of h anemia and haemoglobinuria followed the hypophosphatemia where the haemog the major iron containing compound (Ogawa et al., 1987). Anemia might be d decreased serum copper level which essential for iron absorption and iron m through activation of ferroxidase enzyme (abdel - Maksoud, 1991).The highly increase in both GOT and GPT may be due to liver affection in-haemoglobinuric mentioned by Akhtar et al., (2008) who reported that liver was pale, congested, f swollen in gross examination of died buffaloes from hypophosphatemia.
Marked increase in the level of serum urea and creatinin in hypophosphatemic might be attributed to the involvement of kidney causing its inability of elim excessive urea and creatinin via urine (Kurundiker et al., 1981). Increased serum creatinin level could be attributed to the endogenous release of corticosteroids, and tubular epithelial necrosis (Digraskar et al., 1991). Additionally, dehydratic occurs with haemoglobinuria which was the source of decreased renal perfusion in decreased glomeruler filtration rate and increased blood urea level (Stogda and Latimer et al., 2003).

A significant increase in the serum total bilirubin in hypophosphatemic buffaloes with healthy buffaloes was recorded in the present study. These findings agreement with the results reported by Digraskar et al. (1991) and Kurundkar et .The increase in the serum total bilirubin concentration in hypophosphatemic could be due to increased destruction of erythrocytes (haemolysis), hepa damage, cholestasis and anorexia/ dehydration (Benjamin, 1978; Kurundkar et a In severe haemolysis, hypoxia was developed which affects the structure and fu liver (Stogdale, 1981). Loss of hepatic functions results in decreased capacity fo uptake, conjugation and/or secretion (Latimer et al., 2003).
Regarding to the osmotic fragility of erythrocytes there was highly significant in the osmotic fragility in hypophosphatemic buffaloes as recorded by Mac Willi (1982).The colour of urine in hypophosphatemic buffaloes ranged from red coloured, depending upon the severity of illness (Abdel-Maksoud and Abdel-Rao and Akhtar et al., 2008). Urine analysis revealed presence of haemoglobin an and absence of sugar, keton bodies, bile salt and bile pigment. On mi

examination, there were no intact erythrocytes but few epithelia cells and crys phosphate. Similar observation was reported by Omran et al., (1987), Digrasker (1991) and Akhtar et al., (2008). Changes observed in urine could be attributed to d to kidneys resulting from anemic hypoxia caused by excessive haemolysis (Digra al., 1991).

Also decreased urea and creatinin in urine may be attributed to this damage of which decrease secretion of urea and creatinin in urine. From our present study conclude that at the end of green excessive feeding of Barseem season rich phos diet must be supplemented as bran to subside the clinical and subclinicl ca hypophosphatemia as phosphorus deficiency play an important role in hypophosphatemia aming buffaloes.

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خص العربى

ات بيوكيميائية و هيماتولوجية على بعض الحالات الإكلينيكية و تحت الإكلينيكية ل الفسفور بين الجاموس فى محافظة المنوفية

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ب البيطري – جامعة المنوفية فرع مدينة السادات معهد بحوث صحة الحيوان (فرع شبين الكوم – قسم الكيمياء)

هذا البحث للتعرف على التغيرات البيوكيميائية و الدموية المصاحبة لمرض نقص الفسفور يكى و تحت الأكلينيكى فى الجاموس بمحافظة المنوفية. اجري هذا البحث على عدد ٣٧ جاموسة و ت الى ثلاث مجموعات:

ة الأولى عددها ١٥جاموسة و هى سليمة إكلينيكيا و استخدمت كمجموعة ضابطة . المجموعة تضم ١٢ جاموسة تعانى من فقدان الشهية للعلائق الخضراء و تفضل العلائق الجافة و قد مثلت ة التحت أكلينيكية. أما المجموعة الثالثة و ضمت ١٠ حيوانات تعانى من الأنيميا مع بول دموي ن فى التنفس و إمساك بالإضافة إلى انخفاض درجة الحرارة و مثلت المجموعة الأكلينكية. أظهرت بعض التغيرات الدموية مثل نقص حاد فى نسبة الهيموجلوبين و عدد كرات الدم الحمراء وحجم المصمتة بينما كانت هناك زيادة معنوية فى عدد كرات الدم البيضاء و نسبة الخلايا المتعادلة فى ة التى تعانى نقصا حادا فى نسبة الفسفور.و كذلك أظهرت التغيرات البيوكيميائية نقصا معنويا فى المرتبطة بالفسفور فى جدار الخلايا الحمراء و كذلك نقص فى البروتين الكلى و الزلال و ولين و الفسفور و الحديد الكلى وترابط الحديد الكلى بالدم و أيضا زيادة معنوية فى الكرياتينين و إنزيم اسبرتات أمينوترانس فيريز (AST) و إنزيم الألانين أمينوترانس فيريز (ALT) بينما لا غيرات معنوية فى نسبة الكالسيوم و الماغنيسوم. اما تحليل البول فقد اظهر نقصا معنويا فى كلا من م و الفسفور و الماغنيسوم و البولينا و الكرياتينين فى البول.