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Clinical and Laboratory Studies on Milk Lameness in Holstein-Friesian Cows in Egypt

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Abstract

Chronic hypophosphatemia (milk lameness) is one of the most important metabolic disorders concerning dairy cattle. This study was conducted for evaluation of clinical situation and hematobiochemical status of lactating Holstein-Friesian cows that suffered from chronic hypophosphatemia in Egypt. A total number of twenty seven lactating Holstein-Friesian cows belonged to Giza and El-Behera governorates were included in the present study. Fifteen of them were apparently healthy and fed on concentrate mixture and roughage while twelve of them were suffered from pronounced lameness, peculiar slow creepy gait, stiffness, gradual decrease in milk yield and pica. Results showed significant ($P \leq 0.05$) increase in respiration and pulse rates in affected cows. Also, there was significant ($P \leq 0.001$) decrease in the levels of hemoglobin, PCV, and RBCs count while MCV, MCH and MCHC showed significant increase ($P \leq 0.05$ and $P \leq 0.01$) respectively. Significant ($P \leq 0.001$) increase was recorded in the levels of serum BUN and potassium while there was significant ($P \leq 0.001$) decrease in the level of serum inorganic phosphorus in cows with milk lameness. This data is one of few records about chronic hypophosphatemia (milk lameness) in Holstein-Friesian dairy cows among Egypt.

Key words: Milk Lameness, Holstein-Friesian cows, Hematology, Biochemistry.

Introduction

Hypophosphatemia (milk lameness) is one of the most important metabolic disorders concerning dairy cattle. The clinical relevance of hypophosphatemia in diseased and periparturent cows is still under research and continuous debate **(Grünberg et al., 2015)**. Such disorder was considered as the second most commonly noticed in dairy cattle next to the simple lack of total feed **(Morrison and Whitehair, 1963)**. Hypophosphatemia was a major disease facing high yielding dairy cattle with detrimental economic consequences **(Al mujalli, 2010)**. Phosphorus poor feeds and feed products beside some occasions when there is calcium excess and/or vitamin A deficiency are considered the principle causes of hypophosphatemia **(Abdou et al., 1986)**. Also, the national research council (NRC) recommended that dairy cow rations shouldn't less than 0.32 % from dry matter intake. Another important cause that Egyptian soil considered one of semi-arid tropical areas that suffered from phosphorus deficiency and need to be fertilized with phosphorus **(Koala et al., 1988)**. Phosphorus had an important metabolic role that shared a variety of essential biological functions such as oxidative phosphorylation, oxygen delivery, and buffer for acid-base balance, glycolysis and generation of adenosine triphosphate (ATP). Without the latter, many physiological processes couldn't be completed **(Grünberg, 2008)**. At an individual farm level, there is a significant imbalance between phosphorus inputs and outputs as cow didn't utilize dietary phosphorus very efficiently and about 70% of its content can end up in manure **(Ferris and Harrison, 2014)**. Chronic hypophosphatemia can be resulted from decreased absorption which may lead to muscle weakness and osteomalacia **(Maxwell and Kleeman, 1990)**. Milk production had strong impact on phosphorus homeostasis in dairy cows as milk contained a part of phosphorus which was independent from other part in plasma **(Morse et al., 1992)**. Affected cows with milk lameness showed allotriophagia, lameness, peculiar slow creepy gait, unthriftness, poor growth, weight loss, decreased milk production and low fertility **(Abdou et al., 1986; Betterridge, 1989; Cheng et al., 1998 and Kaya et al., 2008)**. This study was conducted for evaluation of clinical and

hematobiochemical status of lactating Holstein-Friesian cows that suffered from chronic hypophosphatemia in Egypt.

Materials and Methods

A total number of twenty seven lactating Holstein-Friesian cows were included in the present study. These animals belonged to Giza and El-Behera governorates. Fifteen of them were apparently healthy and fed on concentrate mixture and roughage while twelve of them were suffered from pronounced lameness, stiffness, gradual decrease in milk yield and pica. Clinical examination was applied to each cow and performed according to **Radostitis et al. (2007)**. Two blood samples were collected from jagular vein of all examined cows in the present study and the first sample was collected with anticoagulant (EDTA) for examination of hemoglobin concentration, packed cell volume, red blood cells count, MCV, MCH, and MCHC according to **Feldman et al. (2000)**. The other blood sample was collected without anticoagulant and left to clot then centrifuged at 3000 rpm for five minutes and serum was separated according to **Kaneko et al. (2008)** and used for estimation of total protein **(Kaplan and Szalbo 1983)**, albumin **(Tietz, 1990)**, globulin, glucose **(Zilva and Pannall 1979)**, BUN **(Tietz, 1990)**, triglycerides **(Stein, 1987)**, total cholesterol **(Roeschlau et al. 1974)**, calcium **(Young, 1990)**, inorganic phosphorus **(Daly and Ertingshausen 1972)**, magnesium **(Thomas, 1998)**, sodium **(Henry et al. 1974)** and potassium **(Hoeflmayr, 1979)** according to specific test kits produced by Spectrum diagnostics, MDSS, GmbH, Hannover, Germany while glucose was estimated using kits produced by Stanbio Laboratory, Boerene, Texas, U.S.A. Fecal analysis was applied to each cow for exclusion of parasitic infestation according to **Solusby (1982)**. The obtained data were analyzed statistically by using SPSS program version 16 according to **Levesque (2007)**.

Results

The present study was applied on twenty seven Holstein-Friesian cows included fifteen apparently healthy and twelve cows suffered from milk lameness. Historical farms records revealed that calculated dietary inorganic phosphorus was less than 0.15% of DMI. Also, clinical signs were appeared for more than two weeks and lameness was treated traditionally without exploring the real cause. Results of physical parameters were shown in table (1) (fig. 1). These results included significant increase ($P \leq 0.05$) in both respiration (29±1.06 time/min) and pulse rates (67±2.04 pulse/min) in cows suffered from milk lameness in comparison to control healthy cows (22±2.01 time/min) and (60±1.03 Pulse/min) respectively while there was non-significant increase in rectal temperature in comparison to control healthy group. Toward hematological examination, there was high significant decrease ($P \leq 0.001$) in the levels of hemoglobin concentration (g/dl), PCV (%) and RBCs count ($X^6/\mu l$) in cows with milk lameness and these levels were 4.85±0.24, 13.6±0.66 and 2.79±0.17 respectively in comparison to healthy cows 9.62±0.32, 29±1.04 and 6.2±0.21 respectively (table 2) (fig. 2). Regarding red cell indices, MCH (pg) and MCHC (%) showed significant increase ($P \leq 0.01$) and the results were 17.6±0.76 and 35.7±0.74 respectively in comparison to apparently healthy cows 15.3±0.04 and 33.1±0.18 respectively. MCV (fl) in affected cows was 49.2±1.57 compared with control group 46.1±0.20 with mild significance ($P \leq 0.05$) increase for such parameter (Table 2) (fig. 2).

Table 1. Respiration, pulse rates and rectal temperature of Holstein-Friesian cows suffered from milk lameness versus apparently healthy cows.

Table 1. Respiration, pulse rates and rectal temperature of Holstein-Friesian cows suffered from milk lameness versus apparently healthy cows.

	Apparently healthy cows	Cows with milk lameness
Respiration rate (time/min)	22 ± 2.01	29 ± 1.06^{c}
Pulse rate (Pulse/min)	60 ± 1.03	67 ± 2.04^{c}
Rectal temperature (C°)	38.6 ± 0.08	38.8 ± 0.05

c: P≤0.05

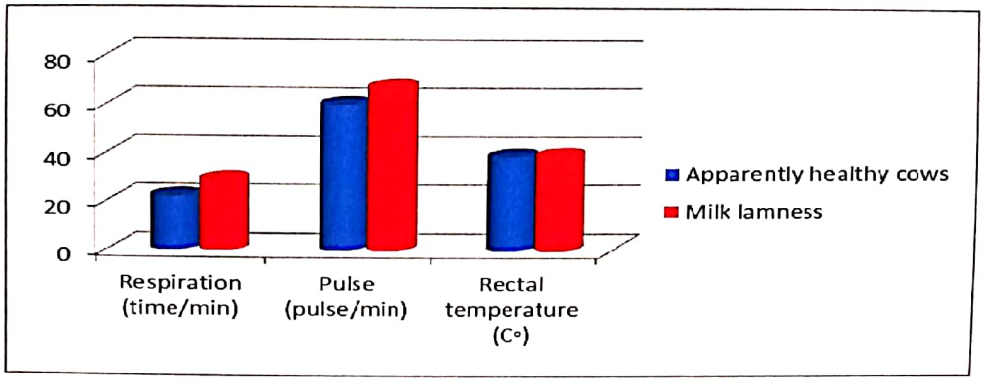

Figure 1. Showed respiration, pulse rates and rectal temperature in cows suffered from milk lameness and apparently healthy cows.

Table 2. Hematological blood parameters of Holstein-Friesian cows suffered from milk lameness compared with apparently healthy group.

	Apparently healthy cows	Cows with milk lameness
Hemoglobin (g/dl)	9.62 ± 0.32	4.85 ± 0.24^{a}
PCV (%)	29 ± 1.04	13.6 ± 0.66^{a}
RBCs (X^{6}/µl)	6.2 ± 0.21	2.79 ± 0.17^{a}
MCV (fl)	46.1 ± 0.20	49.2 ± 1.57^{c}
MCH (pg)	15.3 ± 0.04	17.6 ± 0.76^{b}
MCHC (%)	33.1 ± 0.18	35.7 ± 0.74^{b}

a: P≤0.001 b: P≤0.01 c: P≤0.05

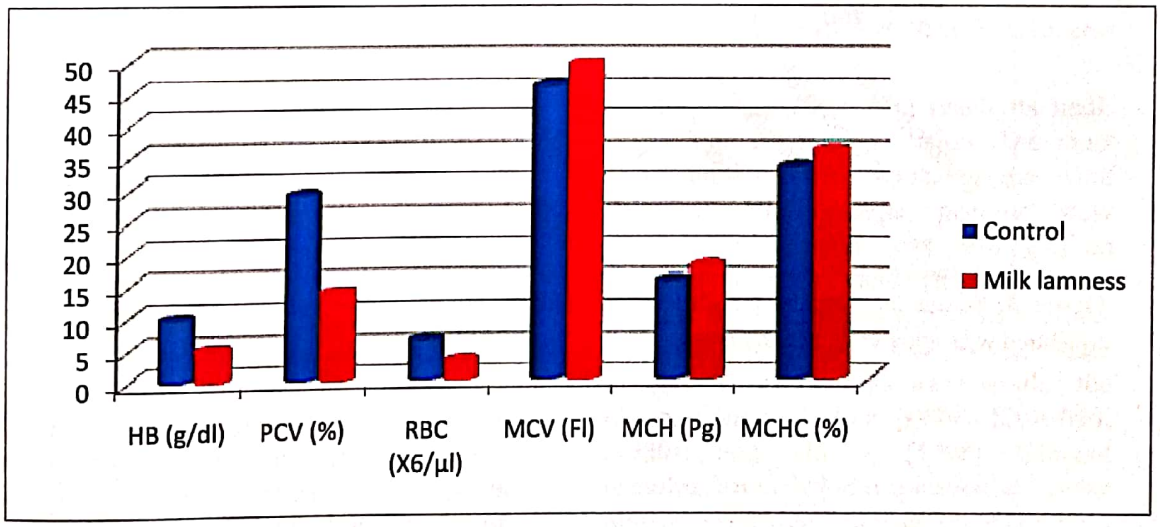

Figure 2. Hematological blood parameters of Holstein-Friesian cows suffered from milk lameness compared with apparently healthy cows.

Concerning serum biochemical constituents (table 3) (fig. 3), the present study showed that there was a mild significant (P≤0.05) increase in serum glucose level (mg/dl) and the recorded level was 58.6±2.11 in affected cows in comparison to control healthy group 52.8±1.57. In term of blood urea nitrogen (mg/dl), there was high significant (P≤0.001) increase in affected cows (table 3) (fig. 3) and the recorded level was 72.7±3.73 while this level in control group

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was 43.5±1.46. Regarded to inorganic phosphorus (mg/dl), results illustrated that there was high significant (P≤0.001) decrease in affected cows (3.33±0.18) compared to apparently healthy cows (6.8±0.26) (table 3) (fig. 4). A high significant increase (P≤0.001) was noticed in the level of serum potassium (mmol/l) as the recorded level in the affected cows was 5.99±0.36 when compared to control group 4.2±0.15 (table 3) (fig. 4). Other parameters showed a non-significant data when compared to control group.

Table 3. Serum biochemical constituents of Holstein-Friesian cows suffered from milk lameness compared with apparently healthy cows.

	Apparently healthy cows	Cows with milk lameness
Total protein (g/dl)	7.1 ± 0.22	7.02 ± 0.17
Albumin (g/dl)	3.1 ± 0.12	3.36 ± 0.11
Globulin (g/dl)	3.89 ± 0.16	3.61 ± 0.19
Glucose (mg/dl)	52.8 ± 1.57	58.6 ± 2.11[c]
BUN (mg/dl)	43.5 ± 1.46	72.7 ± 3.73[a]
Triglycerides (mg/dl)	9 ± 0.63	8.16 ± 0.56
Total cholesterol (mg/dl)	147.6 ± 4.71	156.6 ± 4.53
Calcium (mg/dl)	10.7 ± 0.43	10.29 ± 0.25
Inorganic phosphorus (mg/dl)	6.8 ± 0.26	3.33 ± 0.18[a]
Magnesium (mg/dl)	2.1 ± 0.12	2.43 ± 0.19
Sodium (mmol/l)	154.6 ± 2.76	157 ± 2.19
Potassium (mmol/l)	4.2 ± 0.15	5.99 ± 0.36[a]

a: P≤0.001 c: P≤0.05

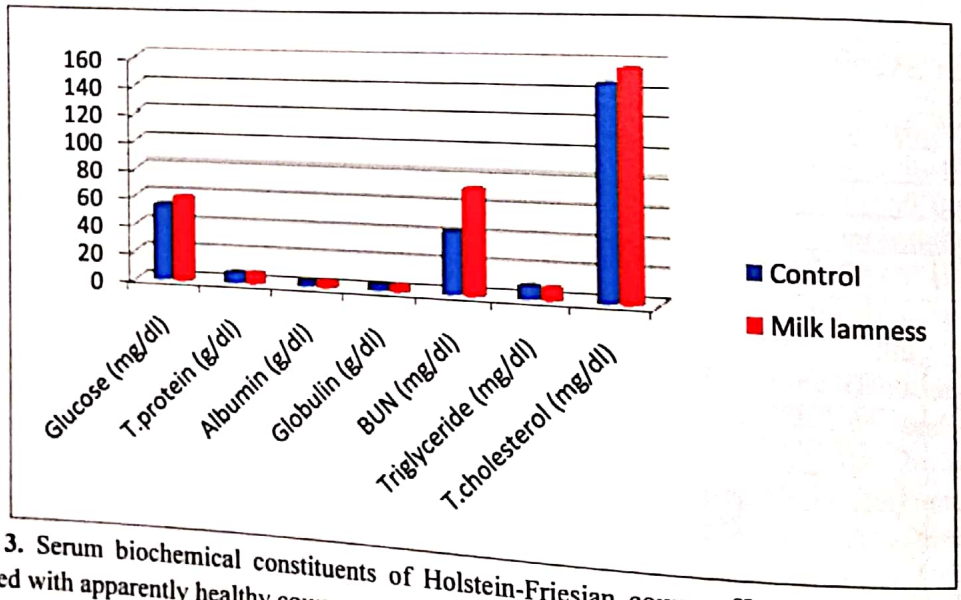

Figure 3. Serum biochemical constituents of Holstein-Friesian cows suffered from milk lameness compared with apparently healthy cows.

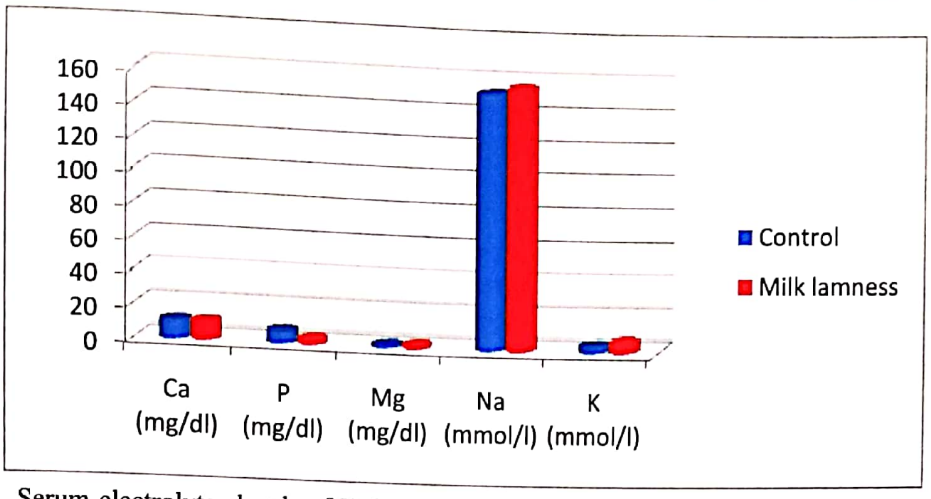

Figure 4. Serum electrolytes levels of Holstein-Friesian cows suffered from milk lameness compared with apparently healthy cows.

Figure 5. Mild arching of back, stiffness and inability to move in adult lactating Holstein-Friesian cow suffered from chronic hypophosphatemia.

Discussion

The main objective of the present work is shedding the light on the clinical and hematobiochemical changes associated milk lameness in Holstein-Friesian cows in Egypt. The present work included twenty seven lactating Holstein Friesian cows belonging to Giza and El-Behera governorates. This number included fifteen apparently healthy cows and twelve cows suffered from milk lameness. Heavy lactation and dietary phosphorus deficiency were the principle causes of chronic hypophosphatemia that were recorded and this was inconsistence with **Benjamin, (1978); Bhikane et al. (1995),** and **Grünberg et al. (2015)** who revealed that feeding lactating cattle about 40% below the daily phosphorus requirements over five weeks resulted in rapid decline of plasma phosphorus to reach its nadir within one week of depletion. The most prominent recorded clinical signs were lameness, allotriophagia, peculiar slow creepy gait; weight loss and gradual decrease in milk yield (fig. 5). Other animals were seen eating soil, dirt, ropes, fecal matters or lick the walls and water troughs. These findings were similar the observations of **Stober, (1978); Kronfeld, (1980);** and **Mousa, (1998).** Clinical examination included respiration and pulse rates showed significant increase ($P \leq 0.05$) in cows suffered from milk lameness (table 1) and these findings were in agreement with **Benjamin, (1978),** and **Abdou et al. (1986)** as there was severe anemia linked to inability for carrying and exchange of oxygen by tissues that was needed for various metabolic processes.

comparison to apparently healthy cows (table 3) (fig. 4). The clinical signs were more pronounced in cases with serum inorganic phosphorus level below 3 mg/dl. This finding matched the findings of **Gartner et al. (1982); Read et al. (1986); Jubb and Crough, (1988),** and **Cheng et al. (1998)**. It is of value noticing that slight decline in serum inorganic phosphorus in some cases was the hidden enemy for very late clinical symptoms when phosphorus level decreased markedly. Serum BUN revealed high significant ($P \leq 0.001$) increase in cows with milk lameness compared to healthy cows (table 3). These data were in accordance with **Latimer et al. (2003)** and **Stockdale et al. (2005)** as both starvation and dehydration were considered the main causes of increased BUN. Regarding serum glucose level, study revealed that there was mild significant ($P \leq 0.05$) increase in cows suffered from milk lameness when compared with apparently healthy cows (table 3). Elevation of serum glucose level was attributed to the endogenous release of corticosteroids when stress increased as a resulted from chronic hypophosphatemia. This explanation comes in agreement with **Singari et al. (1991)** and **Stockdale et al. (2005)**. Another reason was dehydration which considered a source of decreased renal perfusion resulting in reduction of glomerular filtration rate and increase in serum glucose level. This finding disagreed with **Selim et al. (1998)** and **Kurek et al. (2010)** who recorded decreased serum glucose in such condition. The present study included a non-significant decrease in the serum triglycerides level and this was similar the findings of **Kurek et al. (2010)** (table 3). This decrease was due to loss of energy and mobilization of the cow own fat reserves. Toward serum potassium level, cows with milk lameness showed a high significant ($P \leq 0.001$) increase in comparison to control healthy cows. Destruction of cells due to oxidative stress beside increase fragility of RBCs, leads to leakage of potassium content out of cells into plasma resulted in this increase.

Conclusion

From the present study, it can be concluded that lactating Holstein-Friesian cows are prone easily to milk lameness in Egypt as on an individual farm levels, there were many disruptions in feeding programs and calcium to phosphorus ratio of rations. Also, soil in Egypt was considered deficient in phosphorus and need to be fertilized periodically with phosphorus. Cases with milk lameness showed peculiar slow creepy gait, lameness, allotriophagia, weight loss and gradual decrease in milk yield. Also, marked anemia, severe decline in serum level of inorganic phosphorus and increase the levels of glucose, BUN and potassium were noticed. The final breaking point is how to obtain greater milk yield from animals in progressive farms without causing phosphorus deficiency, imbalance and chronic hypophosphatemia.

Acknowledgement

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"دراسات اكلينيكية ومعملية على عرج الحليب في الأبقار الهولشتاين فريزيان في مصر"

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يعتبر نقص عنصر الفوسفات المزمن (عرج الحليب) واحدا من الاضطرابات الأيضية الأكثر أهمية في الأبقار الحلوب. أجريت هذه الدراسة لتقييم الحالة السريرية ومكونات الدم والمصل في الأبقار الهولشتاين فريزيان المرضعة والتي تعاني من نقص فوسفات الدم المزمن في مصر. اشتملت الدراسة على سبعة وعشرين بقرة من سلالة الهولشتاين فريزيان والتي تنتمي الى محافظتي الجيزة والبحيرة. اشتمل هذا العدد على خمسة عشرة بقرة سليمة والتي كانت تتغذى على مخلوط المركزات بالاضافة الى العلف الجاف بينما كان هناك اثنتا عشرة منهم تعاني من عرج واضح ومشية غير طبيعية وانخفاض تدريجي في انتاج الحليب وبيكا. أظهرت النتائج زيادة معنوية ($P\leq0.05$) في معدلات التنفس والنبض في الأبقار المصابة. أيضا كان هناك انخفاض معنوي ($P\leq0.001$) في تركيز الهيموجلوبين، حجم الخلايا المتراكمة وتعداد كرات الدم الحمراء. أظهرت الدراسة أن متوسط حجم كرات الدم الحمراء ومحتواها من الهيموجلوبين له زيادة معنوية ($P\leq0.05$) بينما كان هناك أيضا زيادة معنوية ($P\leq0.01$) في متوسط تركيز الهيموجلوبين داخل كرات الدم الحمراء في الأبقار المصابة. تم أيضا تسجيل زيادة معنوية ($P\leq0.001$) في مستوى كل من اليوريا والبوتاسيم في مصل الدم بينما تم رصد نقص معنوي ($P\leq0.001$) في مستوى الفسفورالغيرعضوي في مصل الدم في الأبقار المصابة بعرج الحليب. تعتبر هذه البيانات واحدة من الدراسات القليلة عن النقص المزمن لعنصر الفسفور (عرج الحليب) في الأبقار الهولشتاين فريزيان في مصر.

الكلمات الدالة: عرج الحليب ، الأبقار الهولشتاين فريزيان ، مكونات الدم ، كيمياء الدم.