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FAT COW SYNDROME IN A HOLSTEIN DAIRY HERD: CLINICAL, BIOCHEMICAL AND HEMATOLOGICAL STUDIES

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SUMMARY

A total number of 21 multiparous Holstein cows (4-8 years) were used in this study. These cows were assigned into 2 groups: the control group was 7 clinically healthy and the diseased group was 14 which were appear too fat with marked drop in milk yield. The herd history of feeding excessive concentrates in the late lactation and the prolonged dry period had led the cows to excessive fattening. The fat cow syndrome (FCS) occur in the immediate postpartum period. The body condition score of the diseased cows was 4 or more. The main clinical signs were depression, anorexia, weakness, ketonuria, marked drop in milk production and recumbency. Significant increase in plasma beta-hydroxy butyrate (BHB) ($P < 0.01$), serum nonesterified fatty acids (NEFA) ($P < 0.001$) and significant decrease in glucose ($P < 0.01$), cholesterol ($P < 0.001$), triglyceride ($P < 0.001$), were recorded in cows with FCS in comparison to the control group. Significant increase in serum bilirubin ($P < 0.01$), AST ($P < 0.01$), GGT ($P < 0.01$) and GLDH ($P < 0.001$), however, non-significant increase in CK and SDH were reported in cows with FCS in comparison to the control group. Significant increase in serum urea ($P < 0.01$), and significant decrease in total protein, albumin ($P < 0.05$), and insignificant decrease in globulin were found in cows with FCS. Non-significant hypocalcemia, hypophosphatemia, hypomagnesemia, hypochloremia, hyponatremia and hypokalemia and non-significant alterations were also reported in the hemogram of the cows with FCS. It cauld be concluded that BHB, NEFA, T. lipids, cholesterol, triglyceride, bilirubin and liver enzymes were reliable markers for diagnosis of FCS in dairy herds.

INTRODUCTION

Fat cow syndrome or lipid mobilization syndrome is a multifactorial condition that occurs in dairy cows after parturition. The syndrome is characterized by progressive depression and failure to respond to treatment of other predisposing diseases. It is associated with excessive mobilization of fat to the liver in well-conditioned obese cows. The mobilization of fat is induced by the negative energy balance and hormonal changes that occur during the periparturient period. This negative energy balance in most cases is aggravated by concurrent periparturient diseases that reduce feed intake and increase energy needs (Pearson and Maas, 2002 and Sevinc et al., 2002).

Fat cow syndrome was firstey described and labeled by Morrow (1976) and in Egypt by El-Sebaie (1987), El-Sebaie et al. (1988) and Sadiek (1992).

Over feeding of cows during the dry period, lasts longer than normal (Radostits et al., 2000), and when the ration rich in protein poor in fiber (Stober and Dirksen, 1983) are the common causes of fat cow syndrome. Visual inspection of fat cow from the rear with determination of flat lumbosacral area and base of the tail, can help in the evaluation of body condition score (Stober and Dirksen, 1983 and Herdt 1988).

One of the consequences of mobilization of body fats is the development of a fatty liver. Approximately one-half of multiparous dairy cows experience moderate to severe fatty liver at calving. The fat cow syndrome is associated with an increased incidence of metabolic, infectious and reproductive disorders such as parturient paresis, ketosis, displacement of the abomasums, indigestion, retained placenta, mastitis and metritis (Roberts et al., 1981, Saleh, 1990, Andrews et al., 1991, Laven and Andrews, 1998 and Bremmer et al., 2000).

The aim of present investigation is to study the possibility of incidence of fat cow syndrome in a Holstein dairy herd with the clinical examination and some biochemical and hematological parameters.

MATERIALS AND METHODS

Animals:

A total number of 21 multiparous Holstein cows (4-8 years) were used in this study. These cows were assigned into 2 groups the first group (control group) was 7 clinically healthy and the second group (tested group) was 14 which were appear too fat (obese) with marked drop in milk yield. All these cows belonged to a private farm (El-Tobgy) located at Fayoum governorate, Egypt. Informations about the herd history were collected and keen clinical examination was also carried out.

Body condition Score (BCS):

Body condition of cows was scored by visual inspection and palpation of the loin region and tail head of the animal. The degree of fatness over these areas was evaluated and scored from 0-5 as described by Herdt (1988).

Blood samples:

Blood samples were taken from cows in both the control and tested groups. The blood plasma was used for determination of BHB with reagent kit obtained from Sigma (St. Louis, MO, USA), while the serum samples were used for estimation of NEFA with kit obtained from WAKO (Dallas, TX, USA). Also, the serum levels of other biochemical constituents were determined by a reagent kits as the follow: glucose (Trinder, 1969), total lipids (Zollner and Kirsch, 1962), cholesterol (Allain, 1974), triglyceride (Royer, 1969), bilirubin (Walters and Gerarde, 1970), AST (GOT) (Anon, 1970), creatine kinase (CK) and gamma glutamyl transferase (GGT) (Anon, 1977), glutamate dehydrogenase (GLDH) (Anon, 1970), sorbitol dehydrogenase (SDH) (Lopez, 1977), total protein (Weichselbaum, 1946), albumin (Drupt, 1974), globulin by subtraction of albumin concentration from total protein, urea (Fawcett and Scott, 1960), calcium (Corns and Ludman, 1987), inorganic phosphorus (Erthingshausen and Daly, 1972), magnesium (Gindler, 1971) and chloride (Feldkamp, 1974). Serum levels of sodium and potassium were estimated by using flame photometer. Whole blood samples with EDTA were analyzed for determination of hemoglobin, hematocrit, RBCs, total and differential leukocytic count (Coles, 1986).

Statistical analysis:

Statistical analysis of the data was done using statistical analysis systems (SAS, 1992).

RESULTS AND DISCUSSION

Clinical findings:

The herd history of feeding excessive amounts of concentrates during the late lactation and the prolonged dry period (3-4 months) had led the cows to excessive fattening. The cows were loosly housed and grouply fed. Also, the history of unresponsive postpartum diseases e.g milk fever, ketosis, retained placenta, metritis, mastitis) and failure of cows to become pregnant was a common complaint. History of high milk production (20 kg/cow/day) in the previous season. The clinical condition occur in the immediate postpartum period. Most affected cows were either obese or well conditioned with a large amount of omental and subcutaneous fat. The body condition score of the diseased cows was 4 or more. Excessive fat was deposited in the subcutaneous tissue at the area of lumbosacral and the tail head. The diseased cows showed anorexia, weight loss, depression, weakness and finaly recumbency ended with culling or death in many cases. Milk production was markedly dropped. Urine analyses for ketone bodies were positive in different degrees.

The feces was in many cases dry and scanty and in some other cases soft. The livers of the diseased cows were enlarged by 5 or more fingers on percussion. The clinical findings were summarized in Table (1).

Table (1): Heart rate, respiratory rate, temperature and clinical parameters in healthy and fat syndrome Holstein cows.

Parameters	Healthy cows		Fat syndrome	
	Mean	S.E.	Mean	S.E.
Heart rate/min.	70.2	1.3	72.0	1.8
Respiratory rate/min	38.5	1.4	41.0	2.0
Temperature (°C)	37.8	0.1	38.9	0.2
Rumen motility/2min.	3.5	0.2	2.3	3.0
Urine ketone bodies	Nil		++	
Appetite	Normal		Inappetance (5) Anorexia (9)	
Milk production	Normal		Severe drop	
Liver percussion	Normal		Enlarged	

* S.E. = Standard error

Similar history and clinical signs were reported by Morrow (1976), El-Sebaie (1987) and Pearson and Maas (2002). In this study, the excessive feeding in the late lactation and dry period play a role in the development of fatty liver, since in this situation feed levels are not actually related to the actual milk production resulting in over conditioned cows at calving (Higgins and Anderson, 1983).

Blood biochemical constituents (Table 2):

The plasma beta-hydroxy-butyrate (BHB) level was significantly increased ($P < 0.01$) in the diseased group which indicate the condition of negative energy balance after calving. This result was in harmony with Sadiek (1992) Andrews (1998), Bertics and Grummer (1999), Hippen et al. (1999) and Radostits et al. (2000).

The serum nonesterified fatty acids (NEFA) showed significant increase ($P < 0.001$) in the diseased group versus the control group. Concentration of blood NEFA is an important and diagnostic indicator of fat mobilization after calving (Gerloff et al., 1986). The increase in serum levels of NEFA was the constant finding in all cas-

es of fatty liver which reflect the drain on body fat reserve as a result of energy deficit after calving (Morrow, 1976, Morrow, 1979 and Bertics and Grummer, 1999). A significant positive correlation (r = 0.97) between liver fat content and serum NEFA was previously recorded by Reichel and Kovac (1990).

The statistical analysis showed significant decrease ($P < 0.01$) in serum glucose level in the diseased group in relation to the control group indicating negative energy balance in these cows after calving. This result was in agreement with Reid and Roberts (1982), Herdt (1988), West (1990), Smith et al. (1997) and Rukkwamsuk et al. (1999). The lowered values of glucose may be attributed to the anorexia in addition to the disturbed liver function in the diseased cows (Sadiek, 1992).

The data revealed significant decrease ($P < 0.01$) in serum total lipids of the diseased group in comparison to the control group. This result was agreed with Herdt et al. (1983) and Sadiek (1992) and disagreed with El-Sebaie (1987). The decrease in serum total lipids may be attributed to low levels of cholesterol and triglyceride in these cows, where cholesterol esters and phospholipids constitute approximately 90 % of the serum total lipids, or to the deposition of lipids in the liver and other organs of the body in association with fatty liver disease (Sadiek, 1992).

The serum cholesterol level was significantly decrease ($P < 0.001$) in the diseased group in correlation with the control group, which coincide with those reported by Reid and Roberts (1982), Herdt et al., (1983), Herdt (1988) and Andrews (1998). Serum cholesterol concentrations appear to vary inversely with liver fat concentration and the correlation between fatty liver and reductions in serum cholesterol is indication of low serum lipoprotein concentrations and may reflect reduced hepatic lipoprotein secretion (Herdt, 1988). The results indicated that the serum triglyceride was significantly decreased ($P < 0.001$) in the diseased group in comparison to the control group, which agreed with Morrow (1976), Herdt et al. (1983), and Andrews (1998). The reduced level of serum triglyceride in the diseased cows was attributed to the accumulation of triglyceride (TG) in the fatty liver and decrease of its secretion into circulation due to insufficient lipoprotein formation by the fatty liver (Reid, 1973). Significant negative correlation between liver fat content and serum TG levels in cows have fatty liver after calving was recorded by Reichel and Kovac (1990).

Table (2): Some blood biochemical constituents in healthy and fat syndrome Holstein cows.

Parameters	Healthy cows		Fat syndrome	
	Mean	S.E.	Mean	S.E.
BHB (mg/dl)	9.7	1.2	29.1**	1.5
NEFA (meq/L)	785	20	1660***	30
Glucose (mmol/L)	2.5	0.3	1.6**	0.2
Total lipids(g/L)	7.0	1.8	5.2**	1.6
Cholesterol (mmol/L)	3.6	0.9	2.0**	0.9
Triglyceride (mmol/L)	2.1	0.9	1.5**	0.9
Bilirubin (µmol/L)	5.5	2.0	16.2**	2.0
AST (GOT) UL)	50.0	5.0	110.0**	8.0
Creatine kinase (CK, UL)	30.0	8.0	55.0	10.0
Gamma glutamyl transferase (GGT, UL)	13.0	2.0	28.0**	3.0
Glutamate dehydrogenase (GLDH, UL)	4.0	2.0	20.0**	3.0
Sorbitol dehydrogenase (SDH/UL)	5.5	0.2	7.7	0.3
Urea (mmol/L)	4.0	2.2	9.0**	3.0
Total protein (g/L)	75.0	7.0	66.5*	10.0
Albumin (g/L)	38.5	3.0	32.0*	8.5
Globulin (g/L)	36.5	9.0	34.5	10.0
Calcium (mmol/L)	2.5	2.0	2.4	5.0
Inorganic phosphorus (mmol/L)	1.75	3.0	1.5	8.0
Magnesium (mmol/L)	0.9	1.0	0.7	9.0
Chloride (mmol/L)	112	1.0	109	1.0
Sodium (mmol/L)	140	1.0	137.7	1.0
Potassium (mmol/L)	4.5	1.0	3.8	1.0

S.E. = Standard error
** = Significant at $P < 0.05$
** = Significant at $P < 0.01$
** = Significant at $P < 0.001$

Significant increase ($P < 0.01$) was reported in the serum bilirubin of diseased group in relation to the control group, which could be attributed to the fatty infiltration of the liver (West, 1990). This result was in accordance to that of Reid and Roberts (1982), Sadiek (1992), and Andrew (1998). The elevated level of serum bilirubi was found to be correlated positively with hepat ic fat content during late pregnancy and early lac tation (Reid et al., 1983).

The serum asparatate amino transferase activity (AST) was significantly increased ($P < 0.01$) in diseased group in correlation to the control group. Such increase in AST in combination of similar increase in bilirubin, GGT, GLDH and SDH are indicators for disturbances in liver function of dairy cows after calving (Rosenberger, 1979). An increased activity of AST was proved to be positively correlated with fatty liver (Reid and Collins, 1980, Herdt and Gerloff, 1982 and Sadiek, 1992). This result was in accordance to those of Reid and Roberts (1982), West (1990) and Andrews (1998).

Non-significant increase was recorded in creatine kinase activity (CK) in diseased cows versus the control group.

The serum gamma glutamyl transferase (GGT) and glutamate dehydrogenase (GLDH) activities were significantly increased ($P < 0.01$) and ($P < 0.001$), respectively, in diseased group in comparison to the control group, indicating hepatocellular injury in fat cow syndrome (Stober and Dirksen, 1983 and Sadiek, 1992). West (1990), West (1997) and Andrews (1998) reported similar findings.

The serum Sorbitol dehydrogenase (SDH) activity showed non-significant increase in diseased cows in comparison to the control ones. Morrow (1976) and Herdt (1988) reported similar results. Serum SDH activity is considered to be a more specific indicator of liver damage in cattle than serum AST, but AST appears to be somewhat more sensitive to the effects of fatty infiltration (Herdt, 1988).

Significant increase ($P < 0.01$) was recorded in serum urea level in the diseased group in comparison to the control group which in agreement with Morrow (1976), West (1990), Sadiek (1992) and Andrews (1998). The fact that lipomobilization is a generalized phenomenon in high lactating cows after calving and not specific to the liver (Robert et al., 1981) may give an explanation to the significant increase in the serum urea in these cows indicating involvement of the kidneys in the process of fatty infiltration and by turn its functional disturbances. These observations were in agreement with that described by Stober and Dirksen (1983).

The serum proteinogram profile in the diseased group showed significant decrease ($P < 0.05$) in total protein and albumin and in-significant decrease in globulin in correlation to the control group. These results were went parallel to that of Reid and Roberts (1982), West (1990), Sadiek (1992) and Andrews (1998). On the other hand significant increase in total protein, albumin ($P < 0.01$) and significant decrease ($P < 0.05$) in globulin were reported by El-Sebaie (1987) in Fat cow syndrome (FCS) and El-Ghoul (1996) in FCS with claw problems. The reduction of total protein and albumin in FCS cows may be

attributed to the reduced protein synthetic capacity of the diseased fatty liver (Reid and Collins, 1980).

Non-significant decrease was reported in the serum levels of calcium, inorganic phosphorus, magnesium, chloride, sodium and potassium in the diseased group in comparison to the control group. Blood electrolytes were reported to be usually low normal to slight below normal in association with FCS except when a condition such as parturient paresis is present to account for severe degrees of hypocalcemia (Morrow, 1976). Hypocalcemia, hypophosphatemia, hypomagnesemia, hypochloremia, hyponatremia and hypokalemia were previously reported in cows with fat cow syndrome by Reid and Roberts (1982), West (1990), Sadiek (1992) and Andrews (1998).

Hematotological findings (Table 3):

Non-significant alterations of the erythrogram and leukogram in diseased group in correlation to the control one, leukopenia, lymphopenia, increased band cells and increased neutrophil:lymphocyte ratio were previously recorded in cowswith lipid mobilization syndrome by Morrow (1976), Reid and Roberts (1982), El-Sebaie (1987), Sadiek (1992) and Andrews (1998).

In conclusion, BHB, NEFA, T. lipids, cholesterol, triglyceride, bilirubin and liver enzymes were reliable markers for diagnosis of FCS in dairy herds.

Table (3): Hematological findings in healthy and fat syndrome Holstein cows.

Parameters	Healthy cows		Fat syndrome	
	Mean	S.E.	Mean	S.E.
Hemoglobin (g/L)	100.0	0.5	96.5	0.8
Hematocrit (L/L)	0.35	0.8	0.29	1.6
RBCs ($x10^{12}$/L)	6.5	1.0	5.9	4.0
WBCs ($x\ 10^{9}$/L)	7.5	2.0	7.0	3.0
Segmented neutrophils (%)	31.0	6.0	48.0	8.0
Band neutrophils (%)	2.5	3.0	5.5	4.0
Lymphocytes (%)	60.0	8.0	42.0	9.0
Eosinophils (%)	1.5	0.2	1.5	0.3
Monocytes (%)	2.5	0.5	2.0	0.6
Basophils (%)	2.5	2.0	1.0	2.0

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