Dept. of Animal Medicine,
Fac. Vet. Med., Assiut University,
Head of Dept. Prof. Dr. M.F. Raghib.

FURTHER STUDIES ON BOVINE LIPOMATOSIS AS ENZOOTIC HERD PROBLEM IN EGYPT

(With 4 Table & 5 Figures)

By
A. EL-SEBAIE and W. HOFMANN*

(Received at 30/8/1992)

دراسة متقدمة عن تنكرز الدهن في الماشية

كمشكلة قطيع في مصر

علي السباعي ، ف. هوفمان

أجريت دراسات عن تنكرز الدهن في الماشية كمشكلة قطيع. سجلت العلاقات المرضية وكانت في صورة زيادة ملحوظة في وزن الحيوان – مع زيادة في ترسيب الدهن تحت الجلد ومنبت الذيل ونقص ملحوظ في ادرار اللبن، وفي الحالات المزمنة كانت النحافة هي العلامة المميزة بالاضافة الى نوبات متقطعة من الاسهال والنفاخ. الجس المستقيمي أفاد وجود تجمعات دهنية صلبة متباينة الحجم والشكل – موجودة في مسار المساريقا وحول الأمعاء والكلية. كما أن التحليل الكيمائي لمصل الدم اشتمل على تحليل بعض الأملاح مثل: البوتاسيوم، الصوديوم، الكلور، الماغنسيوم، الكالسيوم والفسفور. اضافة الى الجلوكوز وأنزيم الأميليز، والألبيومين، ثلاثي الجلسريدات، الزلال الكلي والدهون الكلية ثم الفسفوليبدات والسيلينيوم. وكان هناك زيادة ملحوظة في الدهون الكلية في جميع الحالات الحادة لتنكرز الدهن أما ثلاثي الجلسريدات فقد زادت في الحالات المزمنة، أما السيلينيوم فقد نقص في الحالات المتقدمة والمزمنة. أما تحليل الأحماض الدهنية فقد أفاد وجود زيادة ملحوظة في حامض الاستيريك مع نقص ملحوظ في حمض الأوليك. واستخدم العلاج التجريبي باستعمال ملح سيلينات الصوديوم وكانت نتائجه ايجابية فقط في الحالات المبكرة لتنكرز الدهن في حين لم تكن هناك استجابة بين الأبقار المصابة في مراحل المرض المزمنة.

*: Klinik fur Klauentierkrankheiten und fortpflanzungskunde der Freien Universitat Berlin Germany.

SUMMARY

Bovine lipomatosis as enzootic problem was observed in cows of a native breed herd. The clinical signs were in the form of obesity, body condition score (4-6); drop in milk yield and marked deposition of subcutaneous fat at the base of the tail and at the entrance of the chest. Chronic cases were characterized by severe emaciation; loss of appetite; long standing diarrhea and recurrent tympany. Rectal examination revealed the presence of hard irrigular masses of variable size in the prerenal fat; in the intestine and in the mesentry. Biochemical essay of blood serum including K; Na; CL; Mag; Ca; P; Glucose; Amylase; Albumin; Triglycerides; Total Proteins; Total Lipids; Phosphlipids and Selenium was carried out. A marked hyperlipemia was observed in all groups of diseased animals. Phosphlipids levels were elevated in cows at the advanced stage of the disease and also in chronic cases. Triglycerides levels were only increased in chronic cases. Amylase enzyme-level was droped in chronic cases of bovine lipomatosis. Both groups of advanced and chronic lipomatosis showed a marked decrease in selenium level. Fatty acids determination in necrosed fatty tissues indicated a marked increase in the concentration of stearic acid (C: 18.0) 34%; while olic acid (C: 18:1) showed decrease in its level 28.6%. Diagnostic therapy using sodium selenite-in a dose of 5 mg/cow/day-was supplied to the animals with the ration over a period of 6 months. Seventy percent of treated cows, in the early stage of the disease , were improved and no hard masses were palpated rectally, while the rest of cows-in this stage-showed variable degree of necrosis. On the other hand no detectable changes were observed in cows treated in the chronic phase (fat necrosis) of bovine lipomatosis.

INTRODUCTION

Bovine lipomatosis (abdominal fat necrosis) in cattle is generally characterised by the presence of hard irrigual masses of necrotic fat tissue in abdominal cavity, specially in mesenteric, perirenal and intestine. The disease has been reported from several countries (MOON, 1945; ARBUCKLE, 1962 and

BRIDGE & SPRATLING, 1962; ITO et al., 1968; WILLIAMS et al., 1969; RUMSEY et al., 1979; EL-SEBAIE et al., 1985; EL-SEBAIE & HOFMANN, 1988; OAK et al., 1988 and HOFMANN & EL-SEBAIE, 1990). In Egypt the disease has been described as a herd problem in local-breed cows (EL-SEBAIE et al., 1985; EL-SEBAIE & HOFMANN, 1988 and HOFMANN & EL-SEBAIE, 1990).

A review of available literature on the aetiology and pathogenesis of fat necrosis is incompletly understood. EDGSON (1952) suggested genetic influences, while HOFLUND et al. (1952) attributed such condition to disturbance of circulation. ADAM et al. (1954) reported that hyperthermia could be a cause of fat necrosis. On the other hand HERTINGS and CRAIN (1958) explained that pressure on the tissue may be a cause of such condition. DIRKSEN (1965) stated that hormonal disturbance may consider as cause of fat necrosis. In general several causitive factors of fat necrosis are thought to initiate a change in the composition of fat, within normal fat cells. Fat composition can change as a result of number of factors, pancreatitis (RIBELIN and DeEDS, 1960) and feeding long chain saturated fatty acids (RUMSEY et al., 1979). Furthermore EL-SEBAIE et al. (1985) mentioned a marked depression of thyroid function associated with fat necrosis. RIBELIN and DeEDS (1960) described the process of fat necrosis as a result of liberated glycerol-which is soluble-and carried away the short chain saturated fatty acids and unsaturated fatty acids which are liquid at body temperature, while longer chain saturated fatty acids are solid. The liquid fatty acids tend to abstract basic ions and resulting acid condition cause necrosis of adjacent cells. The polymerization of the peroxidase of unsaturated fatty acids may account for yellowish color associated with lesion. Pathogenesis of fat necrosis is closely related to Vit. E. selenium deficiency (FOREMAN et al., 1986). Treatment of bovine fat necrosis has been reported. OAK et al. (1988) used successively isoprotheialne in diseased Japanese cows. After treatment there was a marked softening of necrotic masses with marked reduce in its size.

The purpose of this study was to carry out further investigations on the biochemical aspects of bovine fat necrosis and determination the composition of fatty acids in necrosed fat as well. Experimental diagnostic therapy using sodium selenite, for treatment of clinical cases of fat necrosis, is also aimed.

MATERIAL and METHODS

Dairy herd:

A herd of 250 local and cross-breed cows were kept for milk production in Al-Awamer dairy farm, Assiut Governorate, Egypt. This farm is located 10 Km east of Assiut City in a new cultivated land. Individuals of the herd showed signs of fat necrosis in various degrees and forms.

Study design:

A) Clinical and biochemical study:

Sixty four cow were selected from the herd for this purpose, and classified according to their clinical status into the following groups:

Group I: Healthy cows: Ten healthy cows were included in this group. All cows were clinically healthy without any signs of bovine lipomatosis.

Grou II: Early stage of fat necrosis: A group of 39 cows showed signs of fat necrosis in early stage, which were characterised by marked obesity, increased body condition score and accumulation of large quantities of soft tissue in abdominal cavity.

Group III: Advanced stage of fat necrosis: A total number of 18 cows were included in this group. Cows were emaciated, showed marked drop in milk yield. Rectal examination revealed the presence of small palpable, hard nodules in the area of intestine.

Group IV: Chronic form of fat necrosis: Seven aged cows were involved in this group. All cows were emaciated and had a history of fat necrosis. Milk production was severly dropped or even with no-milk production. Constant diarrhea or constipation were present. Hard irrigular masses of necrotic fat in the intestine, perirenal and mesentry were palpated rectally.

B) Experimental diagnostic therapy using sodium selenite:

This part of study included 5 cows selected from Group II, in the early stage of the fat necrosis, in addition to other 5 cows selected from Group VI, those resembling the chronic form of fat necrosis. Sodium selenite was used as diagnostic therapy and was given orally in a dose 5 mg/cow/day over a period of 6 months mixed with ration to diseases cows. Close observation and clinical examination were carried out.

C) Blood samples and biochemical analysis:

Blood samples were collected, and blood serum was separated according to the ordinary methods of hematology. Biochemical essay included the estimation of potassium (K), sodium (Na), chloride (Cl), magenessiu (mg), calcium (Ca), phosphorus (P), glucose, amylase, albumin, triglycerides, total proteins, total lipids and selenium. All abovementioned parameters were determined using diagnostic kits supplied by Boehringer Mannheim, Germany. Biochemical analysis were carried out in the diagnostic laboratories of Klinik fur Klauentierekrankheiten und fortpflanzungskunde der Freie Universtat, Berlin, Germany.

D) Tissue samples:

Samples of necrotic fat were collected from slaughter animals. Tissue samples were taken from intestinal, mesentric and perirenal fat, and used for fatty acids estimation. HPLC – chromatography was used for this purpose, and the analysis was carried out in Institute fur Biochemie der Freie Universtat Berlin, Germany.

E) Clinical examination:

All cows were subjected to close clinical examination which included appetite, defecation urination, milk yield, body condition score and rectal examination.

RESULTS

Herd history and clinical findings:

Bovine fat necrosis as a herd problem was investigated over a period of 4 years. The ratio of diseased cows to total number of the herd was about 30%. Aged cows were more susceptible than young ones, sepecially in chronic form of fat necrosis. The most affected cows were over 7 years old. Neither heifers nor bulls were involved in the record of such herd as cases of fat necrosis Individuals of such herd were related to local and cross breed cattle. Ration provided to cows was concentrate and rice straw in summer and alfa-alfa as green fodder in winter. Water supply was of ground source. Diseased cows were examined clinically, selected according to their clinical status, and classified into three groups according to associated clinical signs of fat necrosis.

In table 1 the common clinical findings were summerised. In early stage of the disease (Group II) all cows were obese with body condition score of (5–7), due to massive deposition of subcutanous fat (Fig. 1). Common sites of subcutaneous fat depots were around the base of the tail, in the back and

both sides of the chest, in addition to the presence of small palpable fat nodules. Rectal examination indicated the presence of small scattered hard masses in the area of peritonium and intestinal fat. Appetite and milk yield were reduced in addition to intermittent diarrhea or constipation.

Chronic form of fat necrosis (Group IV) was characterised clinically by marked emaciation, body condition score (1–3), general weakness, partial to complete loss of appetite, ceasation of milk production and long standing diarrhea. Rectal examination revealed the presence of hard masses of various form and size in mesentry, intestine and perirenal fat.

Post–slaughter findings refvealed characteristic lesions in each stage of fat necrosis. Early stage of the disease was characterised by the presence of well circumscribed irregular areas of necrosis. Sometimes much more denser than normal opaque grayish–red colour, surrounded by a well demarcated white zone containing chalk–like calcification was observed (Fig. 2).

Advanced stage of fat necrosis revealed the presence of small nodules of necrotic fat in the area of mesentry and perirenal fat. Lesions in cases of chronic fat necrosis were characterised by the presence of multiple lumpy form nodules in the mesentry along the course of the intestine. Cut section revealed a dry, hard, cheesy opaque appearance and the presence of chalk–like calcification area surrounding the intestine (Fig. 3).

In one of the slaughtered cows a complete adhesion ebtween the large intestine and caecum was observed due to the deposition of large amounts of necrotic fat between them (Fig. 4). The lesion in perirenal fat was characterised by presence of large amount of hard and dry necrotic fat containing white chalky like calcification (Fig. 5).

Results of experimental diagnostic therapy with sodium selenite:

In Table (4) the findings of diagnostic therapy with sodium selenite are illusterated. Cows in the early stage of fat necrosis showed marked improve in their conditon after 6 weeks from start with therapy. Body condition score and milk production showed no alteration. In the last two months of the experiment, there was a marked softening of subcutaneous fat tissue especially at areas of back and base of the tail. Two cows of a treated groups showed complete disappearance of subcutanous nodules, the rest of cows showed no recognisable changes.

Rectal examination revealed a marked reduction in the size of masses present in the abdominal cavity with detectable softening on palpation. Three cows showed these findings but the rest indicated no visible alteration in the size and consistency of masses.

Results of biochemical essay:

Results of biochemical analysis were illusterated in tble 2. The data indicated no significant changes in the mean values of potassium, sodium, chloride, magnessium, calcium and phosphorus in healthy and diseased groups. Glucose level was slightly decreased in Group III and Group IV. Amylase level in serum was significantly droped in Group VI in comparison to other groups. Mean values of serum total proteins showed no makred change between healthy and diseased groups. Triglycerides mean values were elevated only in the cows of Group VI. Mean values for total lipids and phospholipids were markedly increased in all examined groups. Mean values for selenium in serum showed less significant decrease in Group II and Group III. A significant drop was observed in Group VI.

Results of fatty acids essay:

Results of fatty acids composition in healthy and necrotic fat were illusterated in table 3. The values in healthy and necrotic fat for each fatty acid were as follows: myristic acid (C 14:0) was 3.2% and 2.2%, palmitic acid 24.8% and 22.5%, palmitoleic acid 2.3% and 0.6%, Margeric acid 0 and 1.4%, stearic acid 29.4% and 34.1%, olic acid 35.5% and 28.6%, linolic acid 2.0% and 2.3% and linolenic acid 0 and 0.5% respectively.

DISCUSSION

Bovine fat necrosis was investigated as an enzootic herd problem in local and cross-breed cows at Assiut-Egypt (EL-SEBAIE et al., 1985; EL-SEBAIE & HOFMANN, 1988 and HOFFAN & EL-SEBAIE, 1990). Throughout long standing clinical observations of affected herd, bovine fat necrosis was classified according to the related clinical signs into early, advanced and chronic stages. No absolute clinical lines of demarcation between these stages, but clinical diagnosis and rectal examination could help in identification of each stage specially between early and chronic form of the disease. General signs of fat necrosis recorded in this study were similar to the published findings (HOFLUND et al., 1952; MOON, 1954; COX & DeEDS, 1985; BRIDGE & SPRATING, 1962; ITO et al., 1968; FORNEY et al., 1969; OAK et al., 1988 and EL-SEBAIE & HOFMANN, 1988).

The analysis of fat samples in the present study indicate that fatty acids composition in necrotic fat (Table 3) were characterised by a marked increase in stearic acid (C:18:0) concentration, a significant ($P<0.05$) decrease in olic acid (C:18:1) level in addition to a slight increase in the level of

palmitic acid (C:16:1). These findings go parallel with the results reported by ADAMS et al. (1954); RIBELIN & DeEDS (1960) and RUMSEY et al. (1979). Normally mesenteric and perirenal fat tissue contain a higher proportion of saturated fatty acids particularly stearic acid. This degree of saturation in mesentric and perirenal fat depots could explain why a greater portion of necrotic fat lesion was found in mesenteric and perirenal fat depots. However, the compositional changes causes the affected tissue to be recognized as a foreign body, with the appearance of fibroplacia and collagen associated with necrotic fat. Such conclusion was supported by explanation by RUMSEY et al. (1979). Biochemical analysis of blood serum in healthy and diseased groups revealed a significant ($P<0.05$) increase in mean values of triglycerides, total lipids and phospholipids specially in cows which showed the signs of chronic fat necrosis. These findings incidated the presence of disturbance of fat metabolism in cows affected with fat necrosis. Thus an abnormality of lipid metabolism may be responsible for the occurrence of fat necrosis. Results of fatty acids composition agreed with results reported by RIBELIN & DeEDS (1960); RUMSEY et al. (1979) and OAK et al. (1988). Amylse enzyme level was decreased in serum of cows showed chronic fat necrosis (Group VI) while mean values of other diseased groups showed no significant ($P>0.05$) changes. A drop in amylase level in Group VI could be attributed to digestive disturbance which accompanied chornic fat necrosis. Such findings are of less clinical or diagnostic importance.

Mean values of other measured parameters in serum e.g. potassium, sodium, chloride, magnesium, calcium, phosphorus and glucose showed no significant changes between healthy and diseased groups. Results of selenium analysis indicated a slight drop in selenium level in cows of Group III (advanced fat necrosis), while a significant drop of selenium value appeared in cows at chronic stage of the disease. No changes were observed in the early stage of the disease, if compared with the healthy group. Marked drop in selenium level in cows showing chronic fat necrosis could be interpreted as selenium deficiency. Selenium is considered a component of enzyme–glutathione peroxidase and playsa role in the protection of cells by destroying oxidizing agent–such as hydrogen peroxide and lipid peroxidase–thus inadequate level of selenium or selenium deficiency result in degeneration and necrosis of fat cells. Such role of selenium, in the occurrance of fat necrosis, is supported by findings of FOREMAN et al. (1986) on the pathogenesis of general steatitis in foal due to selenium deficiency. Other strong evidence on the role of selenium in ruminant's nutrition was

stated by AMMERMAN and MILLER (1975).

Experimental diagnostic therapy using sodium-selenite:

pi5

Oral administration of sodium-selenite, mixed with ration, for cows in early and chronic stage of fat necrosis showed variable degree of response. A marked improvement was observed in 60% of cows in early stage of the disease, improvement was in form of decrease in size of fat masses in the abdominal cavity and subcutaneous depots as well. Palpable softening of hard masses was detected. Subcutaneous fat nodules were disappeared partially or completly.

On the other hand, cows in chronic fat necrosis showed no response to sodium selenite therapy allover the period of treatment. Variations in response to sodium selenite do not exactly understood, but results could be decleared by the role of selenium in the early stage of the disease through protection of the fat cells from further oxidative processes that finally is followed by degeneration and necrosis of fat cells. However, the already degenerated and necrotic fat cells (chronic stage) did not response totally to sodium therapy.

Variations in response to sodium selenite therapy provided in one hand, an evidence on the role of selenium in incidence of fat necrosis, but on the other hand throw some light on the complicated aspect of pathogenesis of fat necrosis and introduce much more difficulties in the interpretation of such results.

REFERENCES

Adam, J.E.; Foster, J.H.; Faulk, W.H. and Scott, H.W. (1954): Experimental production of subcutaneous fat necrosis by general hypothermia, relation to the chemical composition of fat. Surg. Forum 5: 556–561.

Ammerman, C.B. and Miller, S.H. (1975): Selenium in ruminants nurition: a review. J. Dairy Sci. 58: 1561–1577.

Arbuckle, B. (1962): Diffuse lipomatosis in heifers. Vet. Record, 768–772.

Bridge, P.S. and Spartling, E.R. (1962): Bovine lipomatosis. Vet. Record 74: 1357–1362.

Cox, A.J. and DeEds, F. (1958): Dietary production of lipogranuloma in rats. Am. J. Path. 34: 263–273.

Dirksen, G. (1952): Klinische boebachtungen uber die fattgewebsnekrosen beim rind. Wien. Tierarztl. Monatschr. 55: 517–525.

Edgson, F.A. (1952): Bovine lipomatosis. Vet. Rec. 64: 449–454.

El-Sebaie, A.; Hassan, N.K.; Bayoumi, A.H. and Ibrahim, K. (1985): Lipomatosis associated with fat necrosis in native breed cows in Egypt. 1- Clinical and some biochemical observations. Ass. Vet. Med. J. 15: 149-153.

El-Sebaie, A. and Hofmann, W. (1988): Gehauftes auftreten von lipomatose in einer rinderherade in Assiut (Agypten). Tierarztl. Prax. 16: 359-369.

Foremen, J.H.; Potter, K.A. and Bayly, H.D. (1986): Generlized steatitis associated with selenium deficiency and normal Vit. E. status in foal. J. Am. Vet. Med. Ass. 189, 1: 83-86.

Forney, M.D.J.; Williams, S.M.; Papp, D.E. and Tyler, D. (1969): Limited survey of Georgia cattle with fat necrosis. J. Am. Vet. Med. Ass. 155: 1603-1604.

Herting, D.C. and Crain, R.C. (1985): Foreigen-body type reaction in fat cells. Proc. Soc. Exp. Biol. Med. 98: 347-454.

Hofmann, W. and El-Sebaie, A. (1990): Auftreten von enzootischer fettgewebennekrose bie rinder in Agypten. Symposium Energi und Fettstoffwechsel der Milckuh 476-487. Humboldt-Universtat zu Berlin, Berlin Germany.

Hoflund, S.J.: Helmber, L.K.; Appleviken, B. and Nittlen, H. (1952): On the aetiology, pathogenesis and clinical picture of fat necrosis in cattle. Proc. 15th Intern. Congr. of Cattle Disease, 642-652, Stockholm.

Ito, T.; Miura, S.; Oshima, K. and Vymakunis, S. (1968): Pathoglogical studies on fat necrosis (lipomatosis) in cattle. Jap. J. Vet. Sci. 30: 141-150.

Moon, G.J.H. (1954): Bovine lipomatosis. Newzeland Vet. J. 2: 21-23.

Oak, A.; Yamaskai, T.; Shibatant, M.; Suzuki, T. and Saito, T. (1988): Efficacy of isoprothiolane for the treatment of fat necrosis in cattle. Br. Vet. J. 144: 507-514.

Ribelin, E. and DeEds, F. (1960): Fat necrosis in man and animals. J. Am. Vet. Med. Ass., 136: 135-139.

Rumsey, T.S.; Stuedemann, J.A.; Wilkinson, S.R. and Williams, D.J. (1979): Chemical compositon of necrotic fat lesion in beef cows grazing fertilized 'Kentucky-31' tall fescue. J. Anim. Sci. 48: 3, 673-681.

Williams, D.J.; Tyler, D.E. and Papp, E. (1969): Abdominal necrosis as a herd problem in Georgia cattle. J. Am. Vet. Med. Ass. 154: 1017-1021.

LEGNDS

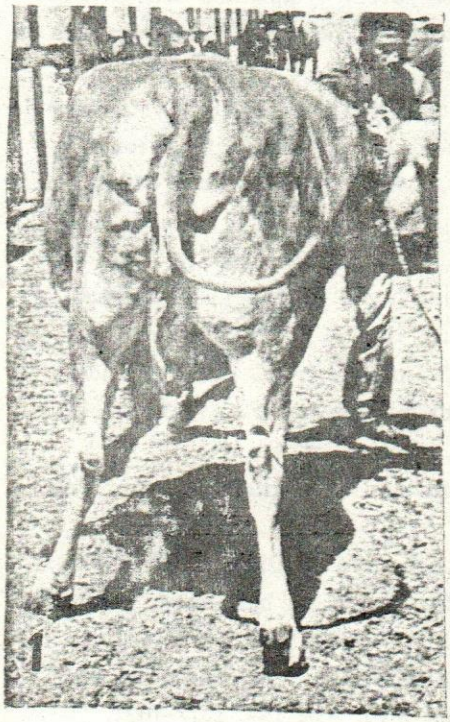
Fig. 1: Obese cow due to massive deposition of subcutaneous fat with the appearance of small fat nodules.

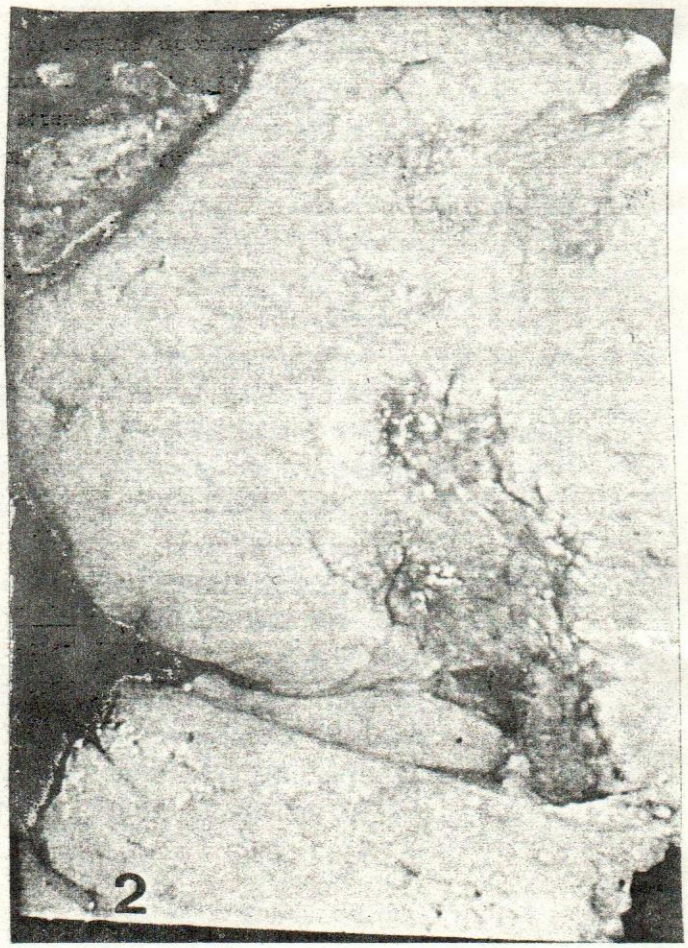
Fig. 2: Early stage of necrosis characterised by the presence of irregular area of necrosis, opaque-grayish red colour surrounded by well demarcated white zone.

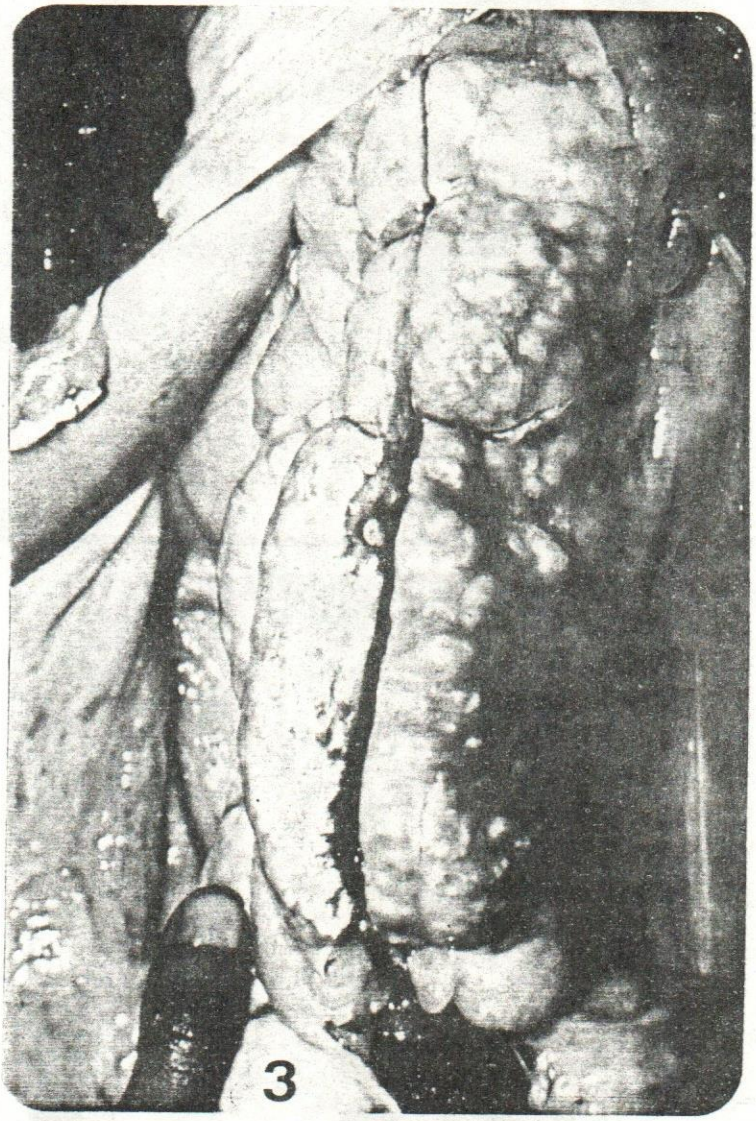
Fig. 3: Cut section in necrotic fat around the intestine in chronic fat necrosis with hard, dry, cheesy opaque appearance and the presence of chalk-like calcification.

Fig. 4: Complete adhesion between large intestine and caecum.

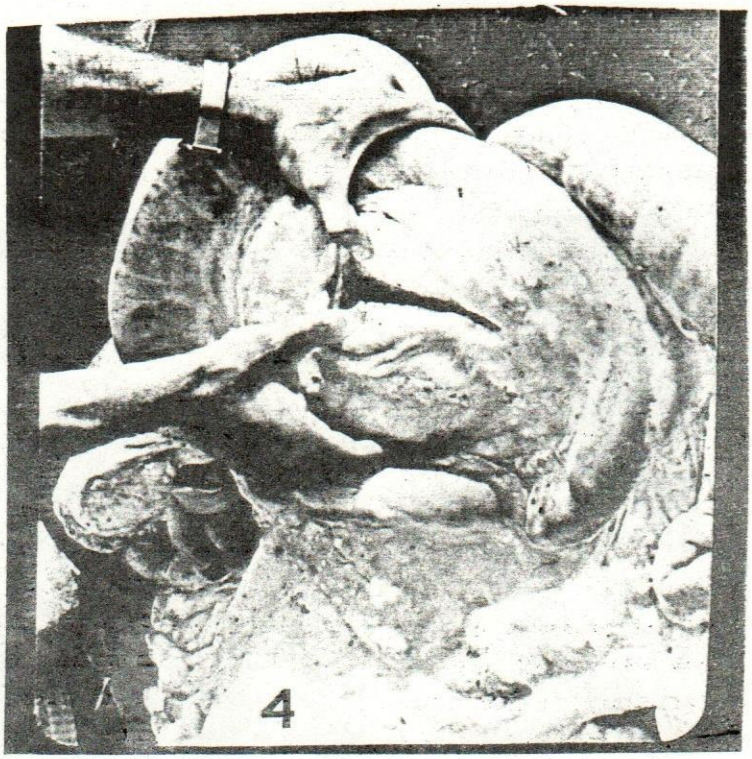
Fig. 5: Perirenal fat: Kidney was embeded in large quantity of necrotic fat containing white chalky like calcification.







3


4

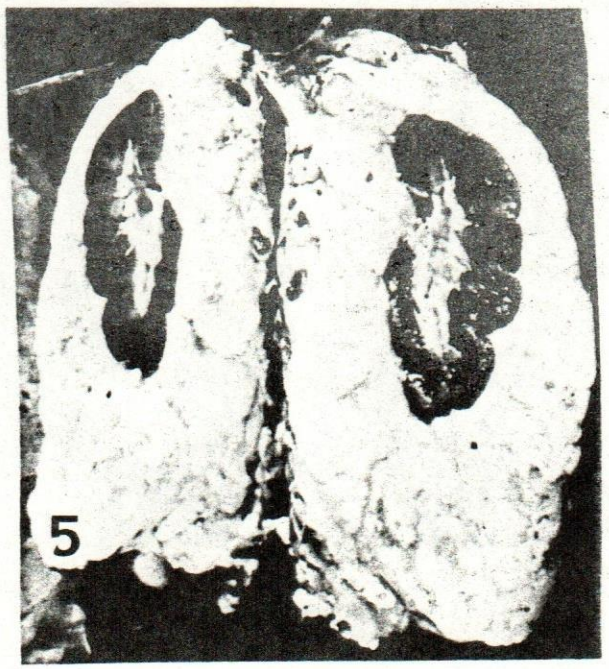

5

Table 1: Clinical status and associated clinical findings of examined groups of bovine fat necrosis

Animal's group	N	Clinical-status	Body condition score	Appetite	Milk production	Other associated clinical signs and rectal findings
Group I	10	Healthy cows	2.5-4	Normal	Normal	All cows were clinically healthy without apparent signs indicating fat necrosis.
Group II	39	Cows in early stage of the disease	5-7	Normal	Decrease	Marked deposition of subcutanous fat tissue in back, chest and around the tail Rectal examination revealed large amount of soft fat.
Group III	18	Cows in advanced stage of disease	3-5	Depressed	Marked drop	Slight reduction subcutanous fat. Rectal examination revealed the presence of moderate size hard masses.
Group IV	7	Chronic form of fat necrosis	1.5-3	Inappetance	No milk production	General weakness, marked emaciation Cows unwill to move, constant diarrhea, tymapany. Rectal examination revealed large, hard and irregular masses in abdominal cavity.

N = number of case

Table 2: Mean values of measured parameters for biochemical essay in healthy and diseased groups

Animals groups	N	K mmol/L	Na mmol/L	Cl mmol/L	Mg. mmol/L	Ca. mmol/L	P mmol/L	Glucose mmol/L	Amylase U/L	Albumin g/L	Trigl. mmol/L	T. proteins g/L	T. lipids mmol/L	Phosp. lipids mmol/L	Selenium ng/ml
Group I Healthy	10														
X		4.6.25	148.7	103.42	0.371	2.185	1.371	2.228	54.63	33.83	3.2	73.14	3.361	2.461	54.7
S.E		±0.248	± 3.26	±14.58	±0.373	±0.373	±0.146	±0.399	±13.03	±3.42	±0.720	±3.52	±1.061	±0.355	±2.30
Group II Early stage	39														
X		5.022	148.13	79.72	0.811	1.861	2.075	1.315	40.15	34.78	1.225	81.28	7.044	2.902	45.8
S.E		0.5322	±5.91	±5.22	±0.143	±0.556	±0.449	±1.780	±12.62	±3.58	±0.179	±6.17	±1.99	±1.535	±1.63
Group III Advanced	18														
X		5.233	148.13	94.41	0.959	2.08	2.233	0.638	65.04	36.18	2.562	81.44	8.578	4.558	41.7
S.E		±0.720	±8.19	±2.12	±0.174	±0.619	±0.501	±0.107	±13.61	±3.44	±1.62	±5.59	±2.213	±3.642	±2.83
Group IV Chronic	7														
X		5.213	145.17	95.78	0.604	1.67	2.62	0.842	19.60	32.88	12.82	70.93	5.66	12.80	38.1
S.E		±0.462	±7.28	±4.19	±0.167	±0.61	±0.71	±0.210	±8.33	±4.10	±8.47	±2.75	±1.43	±2.63	±2.04

N = number of animals
X = Mean
S.E = Standard errors

Table 3 Composition of fatty acids in healthy and necrotic bovine fat

Fatt acids composition		Nature of fat sample	
		Healthy fat (bovine	Necrotic fat (bovine)
Myristic acid	C:14:0	3.2	2.2%
Palmitic acid	C:16:0	24.8	22.5%
Palmitoleic acid	C:16:1	2.3	0.6%
Margeric acid	C:17:0	-	1.4%
Stearic acid	C:18:0	29.4	34.1%
Olic acid	C:18:1	35.5	28.6%
Inolic acid	C:18:2	2.0	2.3%
Linolenic acid	C:18:3	-	0.5%
		97.2	92.2%
Not identified fatty acids		2.8%	7.8%

Table 4: Results of experimental diagnostic therapy of bovine fat necrosis using natrium selenite

Animals group	No.of animals	Clinical status	Period of experiment	Body condition score Before treatment	Body condition score After treatment	Clinical observation during the period of experiment
Group I	5	Early stage of bovine fat necrosis	6 months	5-7	4-5	Visible decrease in body condition score with palpable softening of subcutanous fat especially in areas of back & around the base of Tail. Subcutanous fat nodules partially or completely disappeared. Rectal examination indicated that the hard masses decresed in size and became soft.
Group II	5	Chronic bovine fat necrosis	6 months	1.5-3	no change	No visible changes in general health of animals and their body condition score. Rectal examination revealed no detectable changes in size and consistency of necrotic fat.