Ulceration, Dilatation and Incarceration of the Abomasum in Calves: Clinical Investigations and Experiences+

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Introduction

The three abomasal diseases as listed in the title are different disorders and therefore have to be treated individually. Among these ailments ulcerations of the abomasum are of major importance. First reports on the occurrence of abomasal erosions and ulcerations in calves have been published already in the previous century. The majority of these communications, however, was based on observations in slaughtered animals. Tantz (1912) and Bongert (1912), for example, stated that 78 to 98% of four to fourteen week old calves were affected by abomasal erosions and/or ulcers. This should be kept in mind to understand, that ulcerative abomasitis by far is no product of modern times.

It can be suggested from former as well as from recent observations, that in the majority of the cases abomasal lesions take a clinical inapparent course. They cannot (or seldom) be diagnosed by means of routine methods of clinical examination. However, clinical signs become evident, when ulcers cause markable haemorrhages, when the tissue defect approaches the visceral peritoneum or when it perforates the entire gastric wall. Such severe forms obviously have increased among the inpatients of the Munich cattle clinic during the past years.

Papers recently published on the ulcus abomasi in the calf mainly deal with its possible etiology and pathogenesis. There is assumed, that the same or very similar pathogenic mechanisms are taking place in the mucosa of the abomasum as in the development of gastric ulcers of men, pigs or dogs. With the exception of some few striking relationships, the primary cause or causes are not yet sufficiently clarified. This question, however, recently has attracted interest with regard to the recommendations of the Council of the European Community concerning minimum standards for the protection of calves (91/629/EWG). With the following there will be presented some results of investigations carried out in this field during the past years. For details see the papers listed in the references.[4,6,7,8,9,13] Presently, the following types of abomasal lesions can be distinguished:

Types of abomasal lesions in calves

- Clinically inapparent erosions and ulcers
- Bleeding ulcers
- Deep ulcers with local peritonitis
- Perforating ulcers
  - with abscessation in the greater omentum along the greater curvature
  - with bursitis omentalis
  - with generalized peritonitis
  - with perforation of the abdominal musculature up to the subcutis
  - with peracute shock

Clinically inapparent abomasal lesions

Investigations on early diagnosis

In view of the forementioned diagnostic difficulties the possibility of early diagnosis of mucosal defects in the abomasum was investigated in an experiment with 12 veal calves. At the beginning, the calves were four to six weeks old and their average body weight was 68 kg. They were fed a commercial milk replacer and additionally 500 g hay/animal/d. During the 11½ weeks of the experiment, blood samples were taken two times a week and analyzed for 24 parameters, including red and white blood picture, enzymes, and electrolytes. Three times a week fecal samples were tested for occult blood by means of the Haemoccult-Test (Röhm Pharma).

At slaughter, active or healing ulcers and erosions could be observed in the abomasum of all calves. Remarkable changes of the relevant blood components, however, could not be noticed during the experiment. The Haemoccult-Test, performed 90 to 96 times per animal during the experimental period, reacted positively 2 to maximal 10 times per animal. Whether the positive reactions actually indicated bleeding defects of the abomasal mucosa could not be definitely clarified. In

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the opinion of the author, gastrointestinal haemorrhages can be supposed in such cases, in which the test reacts positively in the course of several succeeding days.

Clinically apparent abomasal lesions

Incidence

An evaluation of 118 cases of clinically apparent ulcerations of the abomasum observed during three years (1986/87/88) revealed the following distribution:

Clinically apparent abomasal ulcerations in calves in 1986/87/88

Ulcers		
bleeding	**deep**	**perforating**
17	20	81
	118	

Anamnestic data

The owners of the forementioned 118 calves with clinically apparent abomasal ulcers were thoroughly interviewed for anamnestic data possibly implied in the pathogenesis of abomasal lesions. As the respective data could not be obtained in all cases, the total number of animals varies in the following evaluation.

- *Breed and sex (n = 118)*

With the exception of two calves (1 Holstein-Friesian, 1 Braunvieh) all patients were Simmentals (Deutsches Fleckvieh). This breed is predominating in the Munich area, which can explain their high representation. The distribution by sex showed a ratio of 105 male to 13 female animals. This exceptionally high proportion of male patients probably can be attributed to regional factors, that is, most of them came from the customary bull fattening units in that area.

Table 1. History data of 118 calves with clinically apparent abomasal lesions.

- Distribution by age (n = 118)
  - < 5 weeks 3.3%
  - 5 - 8 weeks ..... 54.2% } 87.2%
  - 9 - 12 weeks ..... 33.0% }
  - > 12 weeks 9.3%

- Duration of sickness prior to hospitalization (n = 118)
  - Calves with bleeding ulcer (n = 17)
    - 1 - 3 days 59.0%
    - > 3 days 41.0%
  - Calves with deep ulcer (n = 20)
    - 1 - 3 days 65.0%
    - > 3 days 35.0%
  - Calves with perforating ulcer (n = 81)
    - 1 - 3 days 36.0%
    - 4 - 6 days 25.0%
    - 7 - 9 days 18.0%
    - > 9 days 21.0%

- Preceding change of the farm (n = 113)
  - yes 70.0%
  - no 30.0%

- Period of time since change of farm (n = 79)
  - 1 - 2 weeks 51.0%
  - 3 - 4 weeks 33.0%
  - >4 weeks 16.0%

- Housing systems (n = 118)
  - Loose housing with ample straw litter 42.3%
  - Individual box with straw litter 8.5%
  - Tethered housing with straw litter 17.0%
  - Loose box with slatted floor 31.4%
  - Individual box with wooden slatted floor 0.8%

- Feeding regimen (n = 118)
  - milk 3.4%
  - milk/MR+ + solid feed 89.0%
  - solid feed 7.6%
  - (MR = milk replacer)

- Weaning regimen (n = 118)
  - milk/MR + solid feed (no corn silage) + water 52.0%
  - milk/MR + solid feed (with corn silage) + water 20.0%
  - milk/MR + solid feed, no water 28.0%

- Quantity of milk/MR (n = 107)
  - up to 6 1/day 59.0%
  - 7/8 1/day 33.0%
  - > 8 1/day 8.0%

- Concentration of milk replacer (n = 87)
  - < 100 g/L 8.0%
  - 100 g/L 53.0%
  - > 100 g/L 39.0%

- Type of milk replacer

  Ten types of milk replacers of different producers have been used. No preference for certain milk replacers could be demonstrated.

  +MR = milk replacer

Signs and diagnosis of bleeding abomasal ulcers

This type is relatively easy and safe to diagnose: Characteristic signs are inappetence, increasing weakness and depression, sometimes slight colic, increased heart and respiration rates and in particular the black, tarry and foul smelling faeces (Fig. 1). Depending on

the blood loss, the mucous membranes and the episcleral blood vessels appear more or less pale (Fig. 2,3). Sometimes a quick diagnosis can be made by means of aspirated rumen fluid, in which blood can be seen macroscopically (dark red color) or can be proved by clinico-chemical methods (Fig. 4). The suspicion on anaemia can be confirmed in the field by determination of the PCV by means of the minicentrifuge Compur M 1100.

Localizations and pathological consequences of deep and perforating abomasal ulcers

If the defect extends to the deeper layers of the abomasal wall or is even perforating it, this process causes grave abdominal complications with various clinical courses.

For better understanding, the different forms or ways of perforations are demonstrated schematically and at necropsy in the Figures 5 to 14. In this context, many recent publications on abomasal ulcers refer to those located on the torus pylori, however, according to our experience, such ulcers seldom show a tendency to perforate. Perforations predominantly occur anterior to the pylorus in the pyloric part or - less frequently - in the fundic part, with the greater curvature as the preferred site.

If a deep ulcer slowly progresses towards the abdominal wall, it is possible that the perforation into the abdominal cavity can be blocked by an adhesion between abomasal and parietal peritoneum by fibrinous exudation, that is, by a local peritonitis (Fig. 5,6). In such a case, healing can take place. Similar adhesions may also occur with other abdominal organs.

Occasionally, the perforation opens into the attachment of the greater omentum and the abomasal contents, which flow out, become encapsulated, forming an abscess as shown in Figures 7 and 8. Relatively often, however, the perforation is proceeding into the omental bursa, causing a putrid bursitis omentalis. Such a situation is demonstrated in Figures 9 and 10.

In the majority of the cases, the ulcus perforation goes into the abdominal cavity, for example into the intestinal recess, as shown in Figures 11 and 12. The consequence is a generalized peritonitis or shock. In contrast to the observations in adults, shock occurs relatively seldom in calves.

After a solid adhesion between abomasal and parietal peritoneum has been formed, as shown in Figure 5, a deep ulcer can penetrate the abdominal musculature as far as the subcutis. The outflowing abomasal liquid forms a fluid containing subcutaneous pouch, extending over a large area of the abdominal wall (Fig. 13, 14, 17).

Signs and diagnosis of deep ulcers

In spite of the deep tissue damage, such ulcers can be clinically inapparent for a long time. In controlled investigations in veal calves it could be observed that calves with a deep ulcer showed normal development until slaughter or until the day before the perforation took place. Some of them reduced or refused the milk intake for one or two meals. Permanent clinical symptoms occur when the local peritonitis sets in (Fig. 5,6). In that case, a more or less marked indigestion, increased tension of the abdominal wall and signs of pain at deep palpation of the abdomen can be observed. At thorough examination of the organ systems the findings may vary as with perforating ulcers.

Signs and diagnosis of perforating ulcers

From the very variety of the ways and the consequences of abomasal perforations, as demonstrated in Figures 7 to 14, it can be concluded that the clinical manifestations as well as the courses must be very different. That can also be deduced from the anamnestic data of the owners on the duration of the sickness prior to hospitalization. Therefore, the clinical diagnosis usually is difficult. Nevertheless, by considering the anamnesis and systematic clinical examination the diagnosis can be made at least so far that there exists a severe abdominal disease involving the abomaso-intestinal tract.

General status

Usually, the animals show depression, normal or elevated temperature, more or less involvement of the circulatory and respiratory systems. As already mentioned, signs of peracute shock are rarely seen in the young bovine in contrast to the observations in adults. The appetite is reduced, but sometimes, even animals with generalized peritonitis are still eating and drinking fairly well.

Abdomen and digestive organs

The abdominal wall is bulging on the left, on the right or on both sides (Fig. 15, 16). Its tension is increased in 75% of the cases. Rumen contractions are mostly reduced or absent, and ruminal tympany can occur with all types of perforating abomasal ulcers.

The auscultatory examination of the abdomen with ballottement and percussion reveals splashing and/or metallic sounds either on one side or on both sides of the abdomen as indicated in Table 2. In that context it should be mentioned, that perforating abomasal ulcers are frequently associated with LDA or, less frequent, RDA: 22% of these cases were connected with LDA or - differently expressed - 39% of LDA cases were connected

with a perforating ulcer. In 20% of the calves with perforated abomasum the faeces contained blood.

Table 2. Perforating abomasal ulcer in the calf: Important findings

- General status depressed
- Abdomen:
  - bulging on the *left* or right or both sides
  - tension increased (75%)
  - pain on deep palpation
  - splashing sounds on auscultation with ballottement: left and right (48%), only left (27%), only right (16%)
  - metallic sounds on auscultation with percussion: left and right (10%), only left (40%), only right (16%)
- Left abomasal displacement: 22% to 39%
- Rumen fluid:
  $-Cl^-$ concentration > 60 mmol/L: ≈ 30%

Rumen fluid: In 5 of 61 aspirated samples blood could be proved macroscopically or by clinico-chemical method.
The pH ranges as follows:

pH	<5.0	5.1 - 6.0	>6.0
No.	3	11	47

The chloride content exceeded 60 mmol/L in 19/61 samples, probably due to abomaso-ruminal reflux.

Abdominal puncture

When considering the different pathological alterations, which develop in the abdomen after an ulcus perforation, one should understand that puncture of the abdominal cavity can only support the diagnosis under certain favorable prerequisites and that it includes some risks. Abdominal puncture has been carried out on different locations in 50 calves suffering from a perforated abomasal ulcer. The results are listed in Table 3.

Table 3. Perforating abomasal ulcer in calves (n = 81)
Puncture of the abdominal cavity (n = 50): Results

Puncture on	one location	28
	two locations	21
	three locations	1
Punctates	defining the diagnosis	15 (30%)
Punctates	identified as	
	rumen fluid	4 (8%)
	abomasal juice	2 (4%)
Punctates not clearly identifiable		29 (58%)

In 15 cases (30%), the punctate could be clearly identified as pathologic and a secure diagnosis of perforated abomasal ulcer could be made. Main criteria are: pungently acid or putrid smell, low pH, high chloride content. These animals were immediately euthanatized without any further treatment.

In the remaining 35 animals, the puncture did not support or define the diagnosis. Whereas the (second) puncture revealed rumen fluid in four and abomasal juice in two cases, the punctate could not be reliably diagnosed in the rest. This casuistic does not include patients, where the abomasal content had flown out into the subcutis (Fig. 17). In this case the diagnosis can be easily made just by puncturing the subcutaneous pouch.

The differential diagnosis requires consideration of:

- parasitic abomasitis with anaemia
- hemorrhagic enteritis (e.g. BVD)
- uncomplicate LDA or RDA
- dilatation and dislocation/torsion of the caecum
- intestinal ileus
- peritonitis of other origin

Treatment and prophylaxis

In general, the treatment is confined to patients suffering from bleeding ulcers or deep ulcers not yet perforated. The therapy preferably used in case of bleeding lesions consists in repeated blood transfusions together with oral application of antacid drugs and dietary measures.

A surgical intervention and resection of the ulcus has to be taken into consideration only in special cases. That is, the animal must still be in an operable status, the intervention should be early enough, so that massive outflow of abomasal content can be avoided and the abomasal mucosa should not be alterated over a larger area. Own experiences refer mainly to such cases in which deep ulcers could be detected during a diagnostic laparotomy. Depending on the findings in the individual case, either the ulcer has been resected or the ulcerated area of the abomasal wall was enfolded by a mattress suture. Additional measures as with conservative treatment.

As to the therapeutic and prophylactic efficacy of Clenbuterol on abomasal ulcers in calves, the results published up to now do not show a satisfying agreement.[3,12] Good results in the treatment of gastric ulcers in men[16] as well as in ponies[11] could be obtained by administration of histamine H_2 receptor antagonists. It has been repeatedly discussed that such drugs also could be effective in therapy and prophylaxis of abomasal ulcers in the calf. Therefore the efficacy of the H_2 receptor antagonist Cimetidine on the ulcus abomasi has been tested in an experiment with 12 veal calves,[9] as indicated in Table 4.

Table 4. Abomasal erosions and ulcerations in calves:
Results of Cimetidine treatment

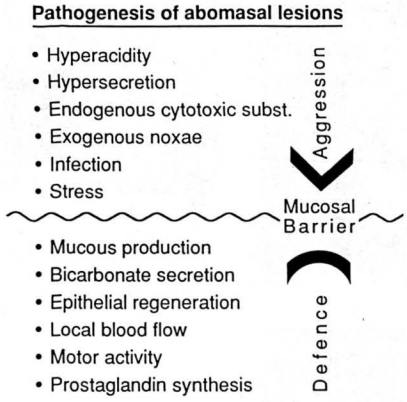
Group 1: n = 6, 30mg/kg/d, 30 days
Group 2: n = 6, controls

Findings 5 days after end of treatment		
Cimetidine	Controls	
	Number of calves	
Ulcers	3	5
Erosions	1	1
Scars	2	0

At slaughter 5 days after the end of medication, active or healing abomasal ulcers and erosions could be observed in calves of both groups. The findings led to the conclusion that Cimetidine administrated as mentioned before, had no distinct therapeutic or prophylactic effect on abomasal ulcerations in calves, but may have positively influenced the healing of the lesions.

Conclusions

Etiology and pathogenesis of peptic abomasal ulcers and erosions in the calf are not yet sufficiently clarified. However, agreement exists in so far that principally the same or very similar processes are going on in the abomasal mucosa of the calf as in the pathogenesis of ulcus ventriculi in men. That is, it can be assumed, that peptic lesions in the abomasum of calves are likewise caused by a disturbance of the equilibrium between the protective mechanisms and the aggressive factors acting in or on the abomasal mucosa, as shown in the following scheme.



As major protective mechanisms are considered the mucous barrier of the mucosa with its high capacity for the neutralization of acid (bicarbonate secretion), its absorptive ability for pepsin and its high viscosity, the superficial epithelium and its regeneration ability, undisturbed local blood flow, prostaglandin synthesis as well as undisturbed motor activity of the stomach. Aggressive factors are those which negatively influence the forementioned defence mechanisms, among those, relative or absolute hypersecretion of pepsin and/or hydrochloric acid and/or gastrin, endogenous cytotoxic substances (e.g. bile acids) and exogenous harmful agents (e.g. infection by Campylobacter pylori) and stress.

As has been demonstrated in several investigations, the incidence of abomasal lesions increases by 20 to 30% when veal calves had access to roughage.[1,5,10,14,15,18] Also the anamnestic data presented in this paper allow us to conclude that the highest risk for the development of abomasal lesions is in the transitional period, when the milk-fed calf is consuming increasing quantities of solid feed. Nutritional and environmental factors must gain even more importance, when both a change of farm and a change of feed are taking place at the same time as could be shown for 70% of the patients of this casuistic. It must be mentioned, that those calves have been bought in by the farmers via so called calf markets without knowing the preceding feeding. Usually those calves are fed with the surplus milk exceeding the quota of the farm.

If roughage intake, as already mentioned, markedly increases the incidence of abomasal lesions in veal calves, it seems to be logical that the risk for a clinical manifestation likewise increases. Therefore, it is not understandable that the rules of the Council of the EC, which now have become national laws, provide, that each calf after the age of two weeks shall have daily access to at least 100 g dry food containing digestive fibre.

Since presently no specifically effective prophylaxis of abomasal lesions is available, the following recommendations for the feeding and handling of newly purchased calves are given to the farmers:

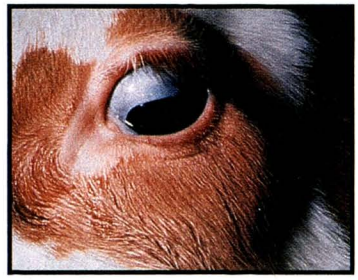
- Milk replacer: should not exceed 2 x 3 L/d
- MR concentration: 50 to 60 g/L for the first 4 to 5 days, and then gradually increasing to 100 g/L
- Water ad libitum
- Hay of high quality + small quantities of calf pellets (concentrates)
- Silages not before end of the third week, not before hay and concentrates are well accepted
- Stress should be avoided as far as possible: long transports, dehorning, high housing density and further
- No corticosteroids containing drugs, careful use of nonsteroidal anti-inflammatory drugs, if not avoidable; careful use of antibiotics in dry feed
- Continuous control of the faeces for dark coloration

The preceding comments may have thrown some

Legends of figures: Abomasal ulceration, incarceration, delatation

Fig. 1. Calf with bleeding abomasal ulcer: depression, black faeces (melaena).
Fig. 2. Pale episcleral blood vessels of a calf with bleeding abomasal ulcer.
Fig. 3. Pale vaginal mucosa and black, tarry faeces due to a bleeding abomasal ulcer.
Fig. 4. Aspirated rumen fluid from a calf with a bleeding abomasal ulcer.
Fig. 5. Local adhesion of the abomasum due to a deep ulcer: schematic representation.
Fig. 6. Local fibrinous peritonitis on the abomasum due to a deep ulcer: intra-operative finding.
Fig. 7. Ulcus perforation into the attachment of the greater omentum: schematic representation.
Fig. 8. Abscess formation after ulcus perforation into the attachment of the greater omentum along the greater curvature: finding after laparotomy and euthanasia.
Fig. 9. Ulcus perforation into the omental bursa: schematic representation.
Fig. 10. Putrid omental bursitis after ulcus perforation into the omental bursa: demonstrated after laparotomy and euthanasia.
Fig. 11. Ulcus perforation into the intestinal recess: schematic representation.
Fig. 12. Ulcus perforation into the abdominal cavity: demonstration after laparotomy and euthanasia.
Fig. 13. Abomasal ulcer penetrating the abodominal musculature as far as to the subcutis: schematic representation.
Fig. 14. Ulcus perforation into the subcutis: necropsy finding after euthanasia.
Fig. 15. Calf with ulcus perforation into the omental bursa: the abdomen bulging on the right side.
Fig. 16. Calf with perforated ulcer and generalized peritonitis: the abdomen bulging on both sides and slight ruminal tympany.
Fig. 17. Calf with an abomasal ulcer penetrating the abdominal musculature: fluctuating enlargement of the subcutis caudally to the costal arch.
Fig. 18. Incarceration of the pyloric part of the abomasum in an umbilical hernia: schematic representation.
Fig. 19. Calf with incarcerated abomasum in umbilical hernia in dorsal recumbency, prepared for operation.
Fig. 20. Fusiform circumcision of the umbilical pole and continuing incision in the midline.
Fig. 21. The inner umbilical sac is uncovered.
Fig. 22. Exteriorization of the abomasum.
Fig. 23. Demonstration of fibrous adhesions between the abomasum and the inner hernial sac.
Fig. 24. The fibrous adhesions ligated and cut.
Fig. 25. Puncture in the centre of a purse-string suture to remove an abomasal gas accumulation.
Fig. 26 Normalization of haemoconcentration, uraemia and hypochloraemic alkalosis in the course of the treatment. HC = haematocrit, UR = urea, BE = base excess.

Figure 1.



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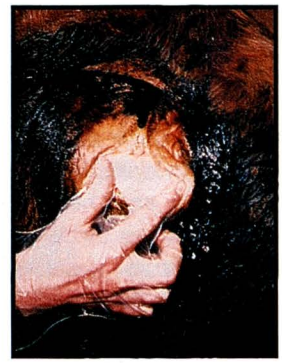
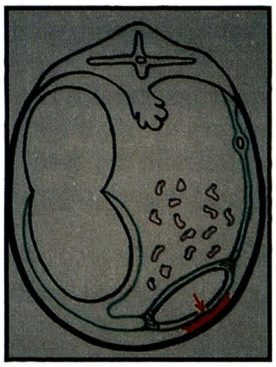


Figure 3.

Figure 4.



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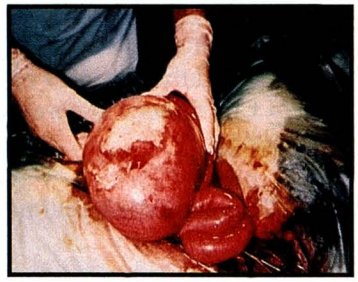

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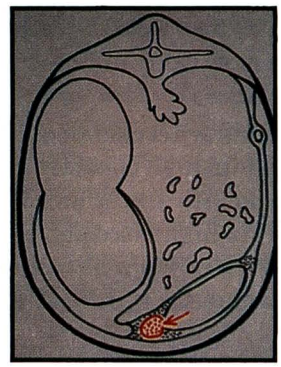

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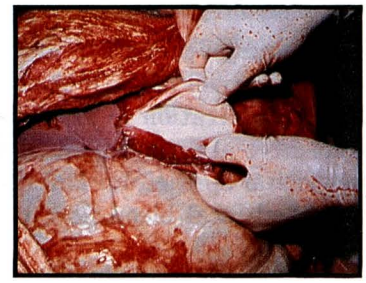

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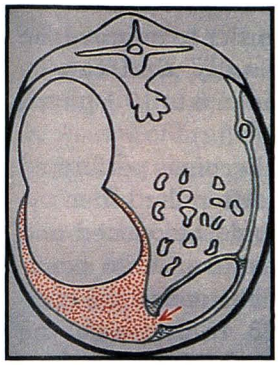
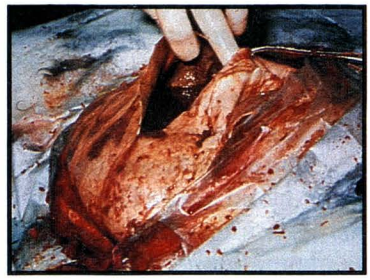


Figure 9.



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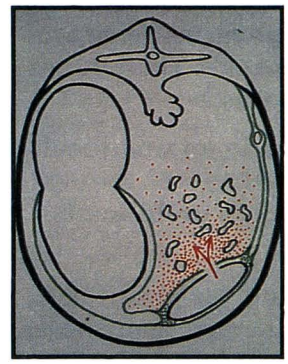

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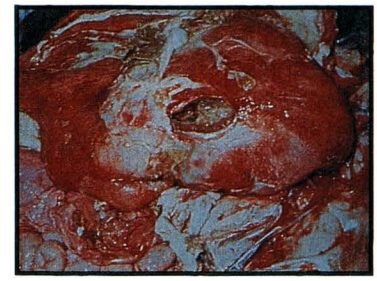

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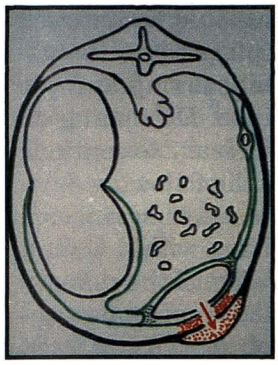

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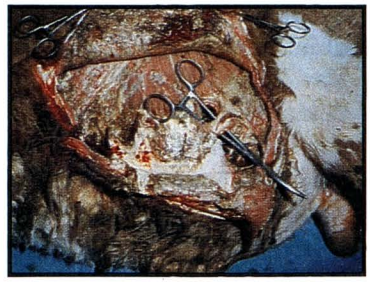

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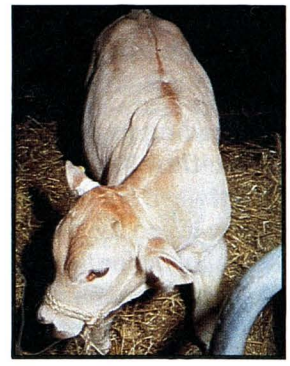

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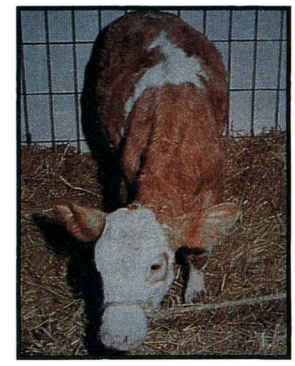

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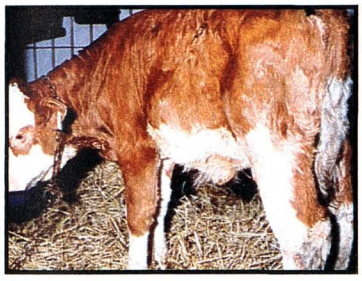

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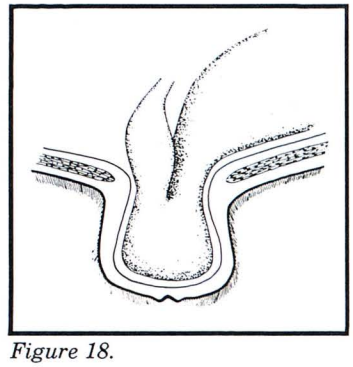


Figure 18.

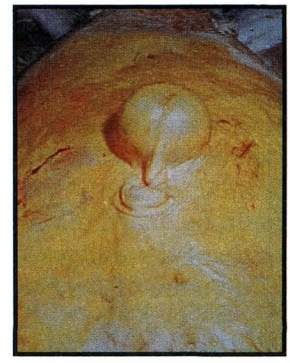


Figure 19.

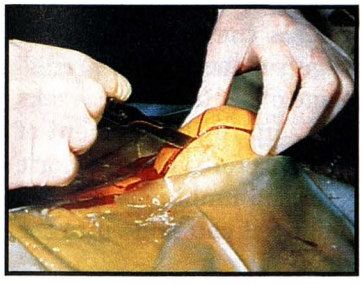
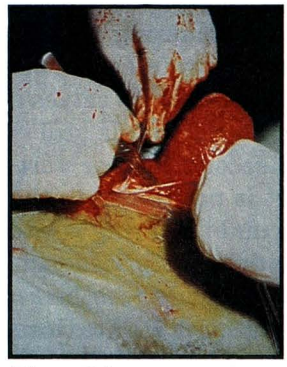


Figure 20.



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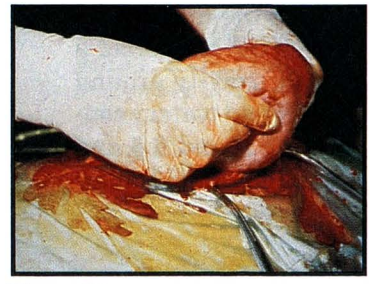

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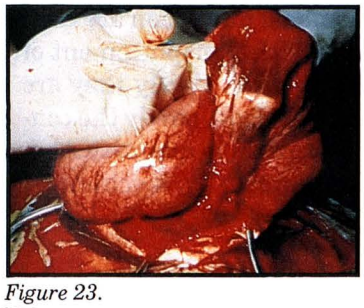
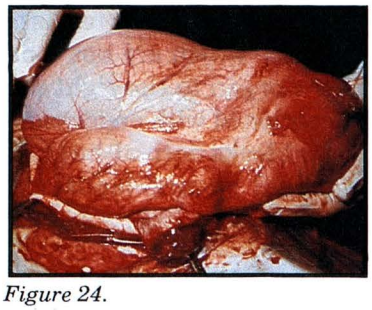


Figure 23.



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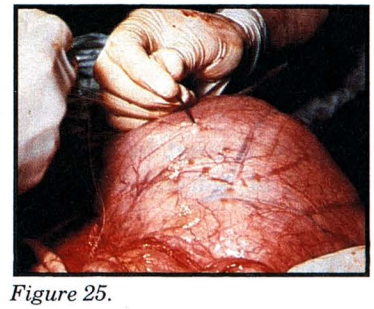

Figure 25.

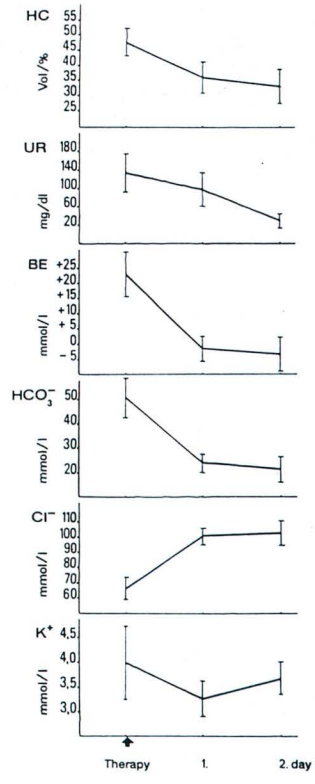

Figure 26.

flash lights on the very variety of problems being connected with abomasal lesions, in the calf. There seems to be an urgent need for further research.

Incarceration of the abomasum in an umbilical hernia (Incarceratio abomasi in herniam umbilicalis)

Definition

A part of the abomasum, mainly the pyloric part, is entrapped in a large umbilical hernia. Sometimes, also loops of the small intestine are included. The ileus situation is schematically demonstrated in figure 18. Usually, the abomasal incarceration is accompanied by a marked abomaso-ruminal reflux syndrome.

Incidence, symptoms, diagnosis

As shown in table 2 of the preceding paper 11 cases have been admitted to the clinic within 3 years (1990/91/92). The patients are usually between six and twelve weeks old. According to the history given by the owner the animals had shown reduced appetite for two to four days prior to hospitalization and continuous enlargement of the navel. In former cases also a longer lasting development has been reported.

On the day of admission the following symptoms could be observed: markedly reduced or absent appetite, depression, distinct exiccosis, increased heart and respiration rates, pale mucous membranes, in the majority normal temperature. There were only weak or no rumen movements and the forestomachs were dilated by liquid content and gas. At auscultation with ballottement, splashing sounds could be heard mainly on the left but also on the right side of the abdomen. Mostly the faeces had dark color and were of dry consistency.

The main finding was the presence of a slightly fluctuating but otherwise doughy/firm enlargement approximately 8 to 15 cm in diameter, in the area of the umbilicus (Fig. 19). In advanced cases the *urine* had a pH between 5.1 and 6.5 and a specific gravity around 1020 or less. The aspirated *rumen fluid* had watery consistency, pungent odor, a pH between 6.0 and 6.8 and on average a chloride concentration of 90 mmol/L indicating abomaso-ruminal reflux.

Haematological findings

The blood changes are characterized by a severe disorder of, electrolyte and acid-base metabolism with dehydration, haemoconcentration, uraemia and a severe hypochloraemic (hypokaliaemic) metabolic alkalosis (Fig. 26).

Treatment

Before starting the operation it must be decided, whether a preceding treatment of dehydration and metabolic alkalosis is necessary, in order to improve the general status of the animal. Usually, that can be achieved by continuous drip infusion of 6 to 10 1 physiological saline over 6 to 8 hours.

The operation of the umbilical hernia is performed in the usual way (Figures 20, 21). After the inner sac has been uncovered (Fig. 21) it should be opened and the abomasum should be exteriorized in order to examine the organ for any damage or adhesions to the peritoneum, as demonstrated in Fig. 23. In that case, the adhesions have to be cut after ligation (Fig. 24).

If gas accumulation has developed in the abomasum during the operation, the gas should be removed by puncture before the organ is replaced into the abdominal cavity (Fig. 25). The umbilical ring and the wound are closed by usual technique. Depending on postoperative progress it can be necessary to continue the fluid-electrolyte therapy during the following days. However, as soon as the ingesta flow sets in again, usually the blood changes are normalizing within a short time. Figure 26 shows the normalization of the changed blood parameters in the course of the treatment; average of six patients.

Conclusions

The incidence of incarceration of the abomasum in an umbilical hernia seems to be linked to the increase of umbilical hernias in cattle of several breeds. Therefore, the owners of such calves should be informed that the umbilical hernia can be hereditary and that these animals should not be used for breeding. However, the patients admitted to the clinic mostly were intended for fattening. In that case the successful treatment of the disease means to avoid a considerable economical loss for the farmer. With early diagnosis and early treatment there is a good chance for complete recovery.

Discussing the pathogenesis the position of the abomasum in calves of that age seems to be a predisposing factor. In calves at the age of six to twelve weeks the abomasum is still extending up to the segment of the second lumbar vertebrae. Therefore, if the pyloric part is positioned in the area of the umbilicus and - if the umbilical ring is wide enough - it can glide into the hernial sac.

Dilatation of the abomasum (Dilatatio abomasi simplex)

By this term forms of abomasal dilatation are classified, which are not connected with a displacement of the organ to the right side or left side. Partly they are combined with engorgement of firm food, partly the content looks normal but can be increased in quantity. Abomasal engorgements can be caused by mechanical

obstruction of the gastro-intestinal passage in the region of the pylorus (e.g. ulcer on the pyloric torus, blunt foreign body) or by disturbance of the motor activity due to lesions of branches of the ventral trunk of the nervus vagus. For some years, disturbances of the motor functions of the stomach caused by paralysis of the vagus nerve also have been observed in calves. Mostly, they could be diagnosed in the wake of severe purulent bronchopneumonias.

It is still in discussion whether there exists also an independent form of abomasal dilatation. In the opinion of the author that seems to be possible. In calves showing signs of indigestion in the weaning period, abomasal sounds could be auscultated on the left side beneath the rumen (filled with firm content) as well as in the area of the lower third of the right abdominal wall. Beside solid food these calves were fed relatively large quantities of milk. After reduction of the milk intake the abomasal sounds disappeared and the rumen extended to the entire left abdomen. If abomasal dilatations go together with severe clinical signs, a diagnostic laparotomy mostly is the best way to make a clear diagnosis and a clear decision for the further treatment.

Summary

1. Abomasal ulcer: After a short report on clinical investigations on the diagnosis of clinically inapparent abomasal lesions, results of an evaluation of 118 calves with clinically apparent abomasal ulcerations are reported: anamnestic data, diagnosis of bleeding ulcers, types of perforating ulcers and consequences, diagnosis, treatment and prophylaxis, and conclusions.
2. Incarceration of the abomasium in an umbilical hernia: Incidence, symptoms, haemuatological changes, diagnosis and treatment of the disease are described on the basis of the experiences obtained in several cases.
3. Dilatation of the abomasum: the forms of abomasal dilatation without displacement are discussed.

Key words

Calf - abomasum - clinically inapparent/apparent abomasal lesions - perforating abomasal ulcer - abomasal incarceration in umbilical hernia - dilatation of the abomasum.

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