SZIE Faculty of Veterinary Medicine Department and Clinic of Internal Medicine

Case Study

Gwenn, 2546, Cow Tierklinik im Fürtli, Urnäsch (CH)

Sabrina Weber, D/2685

Table of contents

| 1. Introduction | 3 |
|--|----|
| 2. Nationale | 3 |
| 3. Clinical examination | 3 |
| Anamnesis | 3 |
| Status praesens of 18 th of September | 3 |
| Basic clinical values. | 4 |
| Skin | 4 |
| Lymph nodes | 4 |
| Respiratory system | 4 |
| Cardiovascular system | 5 |
| Gastrointestinal tract | 5 |
| Urinary tract system | 6 |
| Endorcrine system | 6 |
| Locomotor system | 6 |
| Nervous system | 6 |
| Additional clinical examinations | 7 |
| 18 th of Septemeber | 7 |
| Rectal examination | |
| Ultrasound examination | |
| Explorative Laparatomy | |
| 20 th of September | 7 |
| Rectal examination | |
| Ultrasound examination | |
| Clinical laboratory examination | 8 |
| 18 th of September | 8 |
| Blood gas | |
| Blood chemistry | |
| 19 th of September | 8 |
| Blood gas | |
| Blood chemistry | |
| 20 th of Sepember | 9 |
| Blood gas | |
| Blood chemistry | |
| Decursus | 9 |
| 18 th of September | |
| 19 th of September | |
| 20 th of September | |
| 4. Opinion | 11 |
| Differential diagnosis | 11 |
| Abomasal ulcer | 11 |
| Abomasal displacement | 12 |
| Hemorrhagic bowel syndrome | 14 |
| Intussusception | 16 |
| Diagnosis | 16 |
| Treatment | 17 |
| 5. Appendix | 18 |
| | |

1. Introduction

This case was performed at the Tierklinik im Fürtli, located in Urnäsch (Switzerland). The name of the cow was Gwenn who was a 5 year old of the breed Holstein Friesian. The owner was Martin Signer and the responsible veterinarian Dr. Peter Weisser. The animal was brought into the clinic at the evening on the 18th of September 2013 and was hospitalized the same day. The animals identification number is 3489 and the official number is 3216.

2. Nationale

Owners name and address: Signer Martin, 9230 Flawil (CH) Species: Cattle Breed: Holstein Friesian Colour: Black and white spotted markings Sex: Female Age: 5 years Animals name: Gwenn

3. Clinical examination

Anamnesis

The cow was in pain for 12 hours. She had didn't eat at all for the last 12 hours, got bloody faeces and showed signs of depression and the milk production decreased. The cow showed slight colic symptoms. The family vet already injected Buscopan at 15.00. The cow arrived at 19.00 at the clinic. When the cow arrived she was obviously in pain.

Status praesens of the 18th of September

The body shape and stage of development is according to the breed and age. The body parts are intact and fully developed. The cow got a body condition score of 2 (moderate). The cow is well groomed. She is depressed and is more or less ignorant towards its surrounding. However it stands and walks, bearing weight on all four limbs.



Basic clinical values: Heart rate: 110 beats/minute and a systolic murmur *normal (60-80)* Respiratory rate: 35 breaths per minute Rectal temperature: 37.6 °C

Skin

The skin is black and white in colour. The temperature over the trunk is cool. The skin turgor is decreased and the eye balls are slightly sunken in. The skin has a smell typical for cows and there are no signs of ectoparasites.

Lymph nodes

The mandibular lnn, cervicales superficiales lnn, subiliaci lnn. were palpable. The size and shape was according to the mentioned lymphnode, they were firm, not painful, not attached to the surrounding tissues, smooth and the temperature was the same as the surrounding skin.

Mucous membranes

I examined the conjunctivae, oral, nasal and anal mucosa. The anal mucosa was pigmented, all of the others were light pink, smooth, shiny, moist, intact and the blood vessels can be slightly recognized and the CRT was below 2 seconds.

Respiratory system

The respiratory rate was 20 breath per minute. Both nostrils were intact and partly pigmented otherwise slight pink. The nasal openings are symmetrical and have the characteristic shape seen in cattle. The expired air is of medium strength, symmetric and warm. The smell of the expired airs is ammonia like. The areas of the frontal and paranasal sinuses are symmetrical and shows no visible alterations. Palpation of this area reveals no increased temperature or palpable lesions. Percussing the sinuses gives a strong, resonant and short sound.

The trachea is intact and no pain is revealed during palpation. The cartilage rings can be palpated as elastic rings, and they are compressible with a medium effort. The skin surrounding the pharynx is intact and no abnormal deformities or swellings could be palpated. The temperature is the same as the surroundings. On the auscultation of the pharynx I can hear a weak stridor under inspiration and expiration.

The thorax has a symmetrical shape and the size is characteristic for the given breed. The skin is intact and there are no local deformities. The respiratory rate is 35 per minute and the type is costoabdominal, the depth is medium deep. The rhythm of respiration is regular. The skin of the thorax is intact with a normal temperature. Palpation of the thorax reveals no pain or local deformities in the intercostal spaces. When auscultating over the thorax a strong, rugged blow like can be heard during inspiration and weak blow-like sounds during exspiration.

When performing percussion the resulting sound is sharp, low, non-resonant and short on both sides of the thorax. The caudal lung border are normal for a cow, the 16th intercostal space at level of the tuber coxae, at the 9th intercostal space at the level of the shoulder line and the 5th intercostal space at the level of the elbow line. There is a sharp transition between the percussion sound over the lung area and the dull sound of the surrounding areal.

Cardiovascular system

Heartbeats are heard between the 3rd and 5th intercostal space on the left side and between the 3rd and 4th intercostal space on the right side. Heart rate is found to be 110 beats per minute (tachycardia). A systolic murmur can be detected, however apart from this the rhythm is even. The pulse waves are of medium strength and size, with a normal duration. The jugular veins on both sides are intact. When the jugular vein on the left side is compressed if fills up fast and empties at the next heart beat when the pressure is released.

Gastrointestinal tract

Upon ocular inspection of the mouth, lips and cheeks it can be noted that they have a shape and size which is according to the species and breed. When I put my nose very close to Gwenn's mouth I smell a ammonia odour (uraemic). The mucous membrane is pink, smooth and intact. Pulling out the tongue I can see that it is pale pink and has a surface and papillary structure characteristic to the species. The facial muscles are well developed, intact and symmetrical. The number of teeth are physiological, they are intact and seem to be equally worn. When palpating the oesophageal no abnormalities, such as compression, swellings or obstructions can be felt.

Abdominal distention can be seen and it is quite obvious that the animal feels abdominal discomfort. The abdominal wall is cool and has an even thickness and no palpable swellings or lesions can be felt. A right-sided ping audible sound can be heard during simultaneous

auscultation and percussion of the abdomen and an audible fluid splashing sound detected via ballottement on the right side.

The rectum is closed and there are no visible lesions in that area. The mucosa is pink, dry and intact. The amount of faeces is reduced and a decreased water content with some blood. It looked melena like but it contained fresh blood.



Urinary tract system

There was no sign of pain when the area of the kidneys were percussed on either side. Gwenn did not urinate at the time of physical examination, and no notes of neither the urination or the quality and quantity of the urine had been made.

Endocrine system

To investigate the endocrine system with a normal physical examination is rather limited, and there was no indication of any endocrine disorder so no further investigation was performed.

Locomotor system

Gwenn's posture characteristic for the species, age and breed. The head can be moved manually in all four directions without difficulty. All four limbs are weight bearing and intact. None of the bones show any sign of damage and the percussion sound is bone-like. All joints are intact, can be both extended, flexed and rotated and there is no sign of pain when they are palpated. The hooves are cool and there is no increased digital pulse. The BCS is 2 (moderate). All muscles are symmetrical with a firm muscle like consistency and normal tonus. No pain can be detected upon deep palpation of the muscles.

Nervous system

The spinal cord is inspected and palpated yielding the result that the posture, shape and mobility is normal for the species, breed and age. No pain is detected upon palpation. As there were no signs of a neurological disorder a detailed neurological exam was not performed.

Additional clinical examinations

18th September

Rectal examination

Distended loop of intestine were palpable per rectum and the rectum contained bloody feaces.

Ultrasound examination

Ultrasound revealed very distended intestines on the right side. Some of the loops were contracting regularly. The distended intestines looked liked bicycle tires. (Appendix)

Explorative Laparatomy

When we opened the abdomen we found severe distention of the small intestine. One loop was filled with a blood clot and was therefore harder than the rest. We did a manually clot dissolution without enterotomy.



20th of September

Rectal examination

There was no blood left in the rectum and the intestines didn't feel distended anymore.

Ultrasound examination

All of the loops weren't distended anymore and they peristalsis worked regularly.

Clinical laboratory examinations

18th September

Blood gas analysis

| | | - |
|-------|-------------|----------------|
| | Value | Reference |
| | | Value |
| Na+ | 136 mmol/l | 135-150 mmol/l |
| K+ | 2.7 mmol/l | 3.5-4.5 mmol/l |
| Cl- | 85 mmol/l | 90-110 mmol/l |
| BUN | 26 mmol/l | 20-30 mmol/l |
| Glu | 62 mg/ dl | 45-60 mg/ dl |
| Hct | 20 % | 24-38 % |
| pН | 7.44 | 7.36-7.44 |
| pCO2 | 41.8 | 35-45 mmHg |
| НСО3- | 28.9 mmol/l | 22-26 mmol/l |
| BE | 5 | -2.5-+2.5 |

Blood Chemistry

| Ca2+ | 1.83 mmol/l | 2.2-2.9 |
|------|-------------|---------|
| | | mmol/l |

19th September

Blood gas analysis

| | Value | Reference |
|-------|------------|----------------|
| | | Value |
| Na+ | 138 mmol/l | 135-150 mmol/l |
| K+ | 3.0 mmol/l | 3.5-4.5 mmol/l |
| Cl- | 103 mmol/l | 90-110 mmol/l |
| BUN | 24 mmol/l | 20-30 mmol/l |
| Glu | 67 mg/ dl | 45-60 mg/ dl |
| Hct | 20 % | 24-38 % |
| pН | 7.42 | 7.36-7.44 |
| pCO2 | 39.7 | 35-45 mmHg |
| НСО3- | 27 mmol/l | 22-26 mmol/l |
| BE | 4 | -2.5-+2.5 |

Blood Chemistry

| Ca2+ | 2.1 mmol/l | 2.2-2.9 |
|------|------------|---------|
| | | mmol/l |

20th September

Blood gas analysis

| | Value | Reference Value |
|-------|------------|--------------------|
| Na+ | 136 mmol/l | 135-150 mmol/l |
| K+ | 3.7 mmol/l | 3.5-4.5 mmol/l |
| Cl- | 104 mmol/l | 90-110 mmol/l |
| BUN | 26 mmol/l | 20-30 mmol/l |
| Glu | 59 mg/ dl | 45-60 mg/ dl |
| Hct | 25 % | 24-38 % |
| pН | 7.44 | 7.36-7.44 |
| pCO2 | 41.8 | 35-45 mmHg |
| НСО3- | 25 mmol/l | 22-26 mmol/l |
| BE | 1 | -2.5-+2.5 |

Blood Chemistry

| Ca2+ | 2.57 mmol/l | 2.2-2.9 |
|------|-------------|---------|
| | | mmol/l |

3. Decursus

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18th of September

- 19.00: State depression, mm purple. HR 110, RR 35, T 37.6°C
 Infusion: NaCl + 700 mmol Kaliumchlorid + Calciumborogluconat 500ml (10 litres in total)
- 20.00: Before surgery: 50 ml Pen/Genta, 40ml Ilcocillin P300, 15ml Metacam For anaesthesia: 400ml Lidocaine (inverted L-Block)
- 21.30: During anaesthesia: 1 x 3ml and 1 x 5ml Konstigmin (Neostigmin)
- 22.30: After surgery: 5x every 30min 5ml Konstigmin (sc)

19th of September

- 7.00: State alert but decreased feed intake. HR 135 (arrhythmias), RR 50, T 38.2°C, rumen contractions 2.5/minute, reduced skin turgor, still smells a little like ammonia. Faeces: semi-soft and contained blood clots
- 8.00 Infusion: NaCl + 300 mmol Kaliumchlorid + Calciumborogluconat 250 ml (10 liters in total)
- 12.00 State alert but decreased feed intake. HR 130 (arrhythmias), RR 40, T 38.1°C, rumen contractions 2.5/minute. Faeces: Watery, bright brown
- 20.00 State alert but decreased feed intake. HR 100, RR 40, T 38.5°C, rumen contractions 2.5/minute
 Medication: 50 ml Pen/Genta, 40 ml Ilcocilin P300

20th of September

- 7.00: State alert but decreased feed intake. HR 85 RR 35, T 38.5°C, rumen contractions 3/minute
- 12.00: State calm. HR 80 RR 35, T 38.5°C, rumen contractions 3/minute. Faeces: Soft, dark brown
- 20.00: State calm. HR 80 RR 35, T 38.4°C, rumen contractions 3/minute Medication: 50 ml Pen/Genta, 40 ml Ilcocilin P300

21th of September

- 7.00: State calm. HR 80 RR 35, T 38.5°C, rumen contractions 3/minute. Faeces: Soft, dark brown
- 9.00: Gwenn went home

27th of September

• 17.00 The owner called. Gwenn is fine. Faeces are normal and she eats very well. The milk yield is still a bit decreased but already better than 1 week ago.

4. Opinion

Differential Diagnosis

The cow was presented at the clinic with following symptoms:

- Signs of depression
- Decrease in milk production
- Decrease in feed intake
- Decrease in rumen motility
- Reduced amount of faeces
- Melena or bloody faeces
- Abdominal discomfort
- Abdominal distension
- Right-sided ping audible during simultaneous auscultation and percussion of the abdomen
- Audible fluid splashing sound detected via ballottement of the right side of the abdomen
- Tachycardia
- Dehydration
- Distended loops of intestines palpable per rectum

The main symptoms are abdominal discomfort and bloody stool. Therefore the differential diagnosis will circulate around those symptoms:

- Abomasal ulcer
- Abomasal displacement
- Ileus
 - o Hemorrhagic bowel syndrome
 - Intussusception

Abomasal ulcer

A slowly but steady bleeding ulcus shows typical signs like increasing anaemia, increasing heart rate and respiratory rate, sticky black faeces (melena) and sometimes colic as well. When the ulcus perforates the abomasum the cow will show signs of peritonitis like muscle shivering, painful tensed abdominal skin, pneumoperitonitis, splashing sound during percussion, diarrhoea and later dark, stinky faeces, subnormal or increased body temperature.

In cases with only slight bleeding and mild clinical signs, diagnosis is difficult and may require repeated faecal evaluations for occult blood. Other conditions that can cause partial anorexia and decreased milk production should be excluded by physical examination and laboratory tests, including abdominocentesis. The PCV can help to determine the degree of haemorrhage, although it takes at least 4 h after an acute haemorrhage before the PCV decreases. An occult blood test of the feces can confirm melena. Ultrasound will help for a final diagnosis.

Abomasal displacement

In LDA, as a result of abomasal hypomotility and gas production, the partially gas-distended abomasum becomes displaced upward along the left abdominal wall lateral to the rumen. A mild metabolic alkalosis with hypochloremia and hypokalemia are common. The hypochloremic metabolic alkalosis is due to abomasal hypomotility, continued secretion of hydrochloric acid into the abomasum, and the partial abomasal outflow obstruction, with sequestration of chloride in the abomasum and reflux into the rumen. Hypokalemia is due to decreased intake of feeds high in potassium, sequestration of potassium in the abomasum, and dehydration. Secondary ketosis is common and may be complicated by development of hepatic lipidosis.

In RDA, hypomotility, gas production, and displacement of the partially gas-filled abomasum occur as in LDA. Mild hypokalemic, hypochloremic, metabolic alkalosis develops as well. After this dilatation phase, rotation of the abomasum on its mesenteric axis leads to volvulus and local circulatory impairment and ischemia (hemorrhagic strangulating obstruction). The volvulus is usually in a counterclockwise direction when viewed from the rear and the right side of the animal.

A large quantity of chloride-rich fluid (up to 50l) accumulates in the abomasum, and hypochloremic, hypokalemic metabolic alkalosis develops. The blood supply to the abomasum, and often the omasum and proximal duodenum, is compromised, eventually resulting in ischemic necrosis of the abomasum as well as dehydration and circulatory failure. As circulatory failure progresses, a metabolic acidosis due to hyperlactacidemia and azotemia can become superimposed on the preexisting metabolic alkalosis.

The typical history of displacement includes anorexia (most commonly a lack of appetite for grain with a decreased or normal appetite for roughage) and decreased milk production. In AV, anorexia is complete, milk production is more markedly and progressively reduced, and clinical deterioration is rapid. In abomasal displacement temperature, heart rate, and respiratory rate are usually normal. Hydration appears subjectively normal with displacements except in some chronic cases. Rumen motility may be normal but often is reduced in frequency and strength of contraction. Faeces are usually reduced in quantity and more fluid than normal.

The most important diagnostic physical finding is a ping on simultaneous auscultation and percussion of the abdomen, which should be performed in the area marked by a line from the tuber coxae to the point of the elbow, and from the elbow toward the stifle. The ping characteristic of an LDA is most commonly located in an area between ribs 9 and 13 in the middle to upper third of the left abdomen; however, the ping can be more ventral or more caudal, or both. Pings associated with a rumen gas cap are usually more dorsal, less resonant, and extend more caudally through the left paralumbar fossa. Rectal examination can confirm a gas-filled rumen or an extremely empty rumen that correlates with the rumen ping in these cases. Pings associated with pneumoperitoneum typically are less resonant, present on both sides of the abdomen, and inconsistent in location on repeated evaluation. Frequently, secondary ketosis develops, and ketones are present in the urine or milk. Ketosis that develops in association with abomasal displacement responds only transiently to treatment and recurs. Spontaneous fluid splashing or gas tinkling sounds may be heard on auscultation of the area of the ping or on simultaneous ballottement and auscultation of the abdomen (succussion) The characteristic rectal examination findings with LDA include a medially displaced rumen and left kidney. The abomasum is rarely palpable in LDA and only occasionally in RDA.

The ping associated with RDA also is most commonly located in the area between ribs 10 and 13 on the right abdomen. Differentiation between various causes of a right-sided ping can be difficult in some cases, although a ping cranial to rib 10 usually indicates the presence of AV because the liver is displaced medially by the distended viscus.

For displacement or volvulus, diagnosis is based on the presence of the characteristic ping on simultaneous auscultation and percussion and exclusion of other causes of left- or right-sided pings. Ultrasonography may be helpful in confirming a diagnosis of LDA, RDA, or AV, but it cannot reliably differentiate RDA from AV. Recent parturition, partial anorexia, and decreased milk production suggest displacement. A ketosis that is only temporarily responsive to treatment is consistent with abomasal displacement which may be intermittent. The typical signs on physical examination (in addition to the ping), rectal examination, and laboratory evaluation also support the diagnosis.

Hemorrhagic bowel syndrome (HBS)

The etiology is uncertain, but *Clostridium perfringens* type A plus β2-toxin gene has been isolated from the intestines of naturally occurring cases at higher frequency than from cattle with other intestinal diseases. The primary lesion is similar to that caused by *C. perfringens* in young rapidly growing animals and consists of an acute, localized, necrotizing, hemorrhagic enteritis oft he small intestine that leads tot he development of an intraluminal blood clot. The clot causes a physical obstruction, with porximal accumulation of intestinal fluid and gas and development of hypochloremia, hypokalemia, dehydration and varying degrees of anaemia. The hemorrhagic enteritis is progressive. Ischemia and necrosis extends through the intestinal wall and within 24-48h, there is a fibrinous peritonitis, continued electrolyte imbalance, prfound toxaemia and death.

The clinical signs in cows with HBS are rapidly progressive and affected animals may be found either dead or dying. Because of the combined effects of pathological changes in the intestines, massive haemorrhage into the small intestine, and severe toxaemia, affected cows usually have sudden onset of signs of profound depression, anorexia and decreased milk production. The extremities of affected animals are often cool and rectal temperature is frequently abnormally low.

With the onset of intestinal vasculature rhexis and the resultant blood suffusion within the affected segments of the intestinal tract, the intestines often become obstructed causing some cows to develop abdominal distension, abdominal discomfort characterized by teeth grinding, dehydration, tachycardia, reduced faecal output and melena or dark clotted blood in the faeces. Decreased frequency and amplitude of rumen motility and audible fluid splashing sounds on ballottement of the right aspect of the abdomen and pinging during the

simultaneous auscultation and percussion over right middle portion of the abdomen are often detected. Clostridium perfringens type A is a gas-producing organism and the aggressive multiplication of the organism produces large amount of gases in the proximal portion of the small intestine. The retrograde flow of these gases through the duodenum to the abomasum could ultimately result in abomasal dilation. This might explain the right sided ping detected in 75% of cases and clarify why cows that eventually receive a diagnosis of HBS are often initially referred for management of a possible RDA. Transrectal palpation may reveal distended loops of the small intestine, and a distended firm rumen. However in many instances a rectal examination does not reveal distended loops of intestine because the blood-filled intestines segments sink into the ventral portion of the abdomen and remain beyond the reach of the examiner, in advanced cases, intestinal necrosis takes place. Death of affected cattle occurs within several hours or may take up to 2 days following the onset of clinical signs.

The ultrasonographic findings that are common in cows with HBS are dilation of of the small intestine, usually the proximal portion of the jejunum, with thickening of the intestinal wall. The intestinal contents is mostly echoic to hypoechoic with or without localized hyperechoic masses that are consistent with blood clots. The blood clots can only be found in every 5th case. In animals with HBS, intestinal motility is absent or remarkably.

The hematologic and serum biochemical abnormalities detected in cows with HBS are largely reflected of the acute nature of the disease and the resultant gastrointestinal tract stasis. Hyperglycaemia is a common finding in cows with HBS, it is most likely a due to a stress-dependent response and epinephrine release. Hypocalcaemia, hypermagnesaemia and hyperphosphataemia are also common findings in cows with HBS. This could be attributable to the heavy integration of calcium in the proteases produced by progressively multiplying Clostridium perfringens type A organisms, which reduces calcium availability in the gastrointestinal tract.

In cattle with HBS, a common finding during exploratory surgery examination is severe distention of the small intestine. Also common is devitalisation of the proximal portion of the small intestine, the serosal surface of which develops a dar red to purple discoloration, In addition, frank blood is usually found in the intestinal lumen with or without blood clots and sloughed intestinal mucosa. In advanced cases, the affected segments of intestine are friable,

turgid, and impacted with gelatine-like clotted blood casts and fibrin strands on the serosal surface of the jejunum.

Intussusception

Intussusception is a inversion of the gut into itself, creating a gut blockage. Usually it causes a severe general disturbance and without surgery it leads to death. The place of the intussusception is usually at the end of the jejunum or border to the ileum. It causes local congestion, edema, swellings which leads to a bowel obstruction (ileus). Ileus is prone to cause transudation, bleedings, gangrenes and an increased permeability for bacterias which can lead to peritonitis, sepsis and death.

In the first phase (1-6 (12h)) of the intussusception the following clinical signs appear. First sudden colic signs arise with the classical symptoms like hitting by tail, getting up and down frequently, nervousness, wallow, moan. The pulse and respiratory rate increase, the temperature stays normal and the amount of faeces is normal in the beginning but decreases by the time. By rectal examination you might be able to feel a hard intestines that is an indication for a intussusception.

In the second phase (12h-4d): There is no defecation and sticky mass sometimes with blood (melena) can be felt in the rectum. The animal is apathetic, no rumination can be observed, the ruminal contractions are weak and the general condition worsens hour by hour.

In the third phase (3-4d): Temperature, respiratory rate and pulse will drastically increase. The heart sounds first pounding later it will be very weak. The pulse can't be felt anymore, the body surface is cold, the eyes are sunken in and the cow will lay down. The animal will die from intoxication and sepsis.

In the blood we can find a increased glucose level and decreased kalium and chlorid levels.

Diagnosis

The vet had a strong suspiscion of HBS when the owner called already. The cow showed all of the main symptoms described for HBS like: Depression, melena, bloody faeces, abdominal discomfort, abdominal distension, right-sided ping audible during simultaneous auscultation and percussion of the abdomen, tachycardia, dehydration, distended loops of intestine

palpable per rectum. The laboratory examination was very characteristic as well. She got a hyperglycaemia, hypokalaemia and hypocalcaemia. After those laboratory examinations and clinical findings the abomasal ulcer can be excluded. We did an ultrasonography which underlined the presumed diagnosis. Intussusception and abomasal displacement still can't be 100% excluded. After the ultrasound examination there was a strong suspicion to HBS. A final diagnosis only can be made by opening of the abdomen. During surgery we found the intestine in question. The serosal surface was dark red to purple discolorated. In addition, we found frank blood in the intestinal lumen with blood clots and sloughed intestinal mucosa. We perfomed a manual clot dissolution without an enterotomy. After surgery the diagnosis was obvious: Haemorrhagic bowel syndrome.

Treatment

The treatment of individual cattle with HBS is challenging. The vet experience is that the best chance of survival and recovery to expected production levels occurs with early diagnosis followed by aggressive medical and surgical therapy. Even with intensive and aggressive treatment, the mortality rate for this condition still often exceeds 75%. Occasionally, very early recognition and aggressive medical treatment alleviates the need for surgery. Medical treatment usually includes fluids, pain-killers or anti-inflammatory drugs, antibiotics and Clostridium perfringens C and D antitoxin. Unfortunately, there is no specific antitoxin for Clostridium perfringens A, but there may be limited cross protection from the antitoxin of Clostridium perfringens type C and D. Surgery for this condition is no small undertaking, especially in the field because, at the very least, the intestinal manipulation is extensive. Generally, cows that have clots that can be massaged through the intestine do better than those that require either opening or removal of the intestines. Surgical intervention early in the course of the disease is superior. For all affected cows, treatment success is heralded by a return of normal intestinal movement and production of manure.

Prognosis

The prognosis in this case was quite good because the cow was presented early in the course of the disease and the surgery went good. We found the intestine of question and were able to massage the blood clot through the intestinal wall and the cow reacted well to the medication. The vet already did 5 HBS surgeries before and 4 of them went well and the cows are still alive. A week post surgery the cow was in a very good condition.

5. Appendix

