Digestive Disorders Following Obstruction of Flow of Ingesta through the Abomasum and Small Intestine.

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Introduction

It has been known for quite a while that several forms of indigestion in the ruminant are associated with dehydration, depression, an overvilled rumen fluctuating upon punching and a decreased or absent faeces production. In the blood of these cows there is a hypochloremic, hypokalemic, metabolic alkalosis, an increased hematocrit and a more or less severe uremia. In the well advanced cases, the diagnosis posterior functional gastric stenosis or functional pyloric stenosis is made, but when a thorough clinical examination is performed the same syndrome or part of it can be recognized in cows with other lesions of the gastro-intestinal tract. It is important to recognize the syndrome because the disturbances of water and electrolyte balance as a consequence of this stenosis may be seriously complicating the condition and treatment, although the primary cause can be treated without problems.

The patient.

What does a cow with such a "passage problem" look like? A good example is given by the 3 years old Friesian cow 5 months pregnant, presented to us because she was off feed, had a severe drop in milk production and passed little faeces containing inadequately ruminated (undigested) plant fragments. Her abdomen was distended and an occasional grunt was heard. She had been given a laxative without results. The cow was dull and in poor condition. Her rumen was distended on the left side and in the lower part of the right flank resulting in a typical asymmetric silhouette when the cow was viewed from behind (fig. 1).

The pulse rate was 96, respiration rate was 20 and body temperature was 39°C. Mucous membranes, eyes and skin turgor showed a severe dehydration. The cow had a distinct ammoniacal mouth odor. The rumen contents fluctuated upon punching, there was no free gas bloat and the doughy roughage containing part had disappeared.

Rumen movements were present, however very weak and frequent. Grunt tests were negative. Upon rectal exploration an overloading of the dorsal and ventral ruminal sac was found. The uterus was enlarged, contained a fetus but was otherwise normal. The rectum contained no faeces. With a rubber stomach tube about 80 liters rumen fluid was siphoned from the rumen (fig. 2).

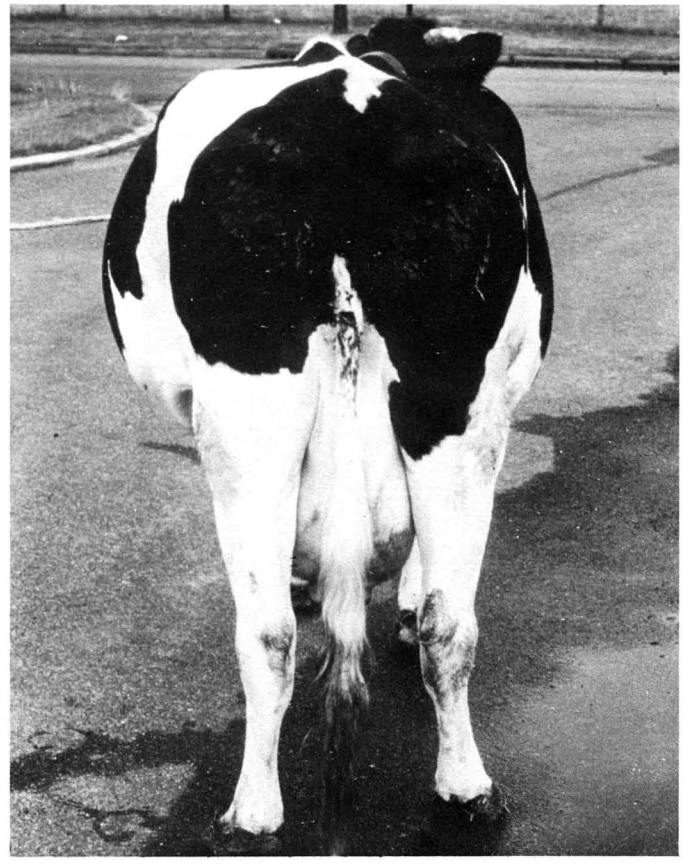

Fig. 1. The patient described showing the typical asymmetric silhouette.

Fig. 2. The same cow immediately after ± 80 liters rumen contents were siphoned off.

Examination of the blood revealed a severe hypochloremic metabolic alkalosis with dehydration and uremia (fig. 3). Despite a high carbon dioxide pressure, compensation of the alkalosis was not achieved. The pH of the rumen fluid was normal, but its buffering capacity was very low and the chloride concentration was strongly increased. An x-ray picture of the reticulum and surroundings showed a piece of wire apparently penetrating the wall of the reticulum and signs of peritonitis in the area of the reticulum and the abomasum.

The cow was treated with large amounts of saline solution intravenously (6-9 likes BID), put on antibiotics (penicillin-streptomycin) and a magnet was introduced with a balling gun. The cow responded fairly well. The next day the water and electrolyte balance had improved (fig 3). The treatment was continued and the condition of the cow improved. The animal started to take some hay and faeces production resumed. This improvement was reflected in the results of the blood and rumen fluid examinations (fig. 3). Therapy was maintained for 5 days and at 11 days after treatment had started the parameters examined in blood and rumen fluid were returned within normal limits. By that time the cow's appetite was normal and on clinical examination no abnormalities could be found. The cow was sent home.

The owner reported 3 months later that she was doing well.

Pathology.

As mentioned before, a number of abomasal and intestinal lesions may lead to a clinical syndrome more or less similar to that of our patient. This syndrome is called posterior functional gastric stenosis, functional pyloric stenosis, vagus indigestion or Hoflund Syndrome. Although it is often questionable whether the stenosis is functional or mechanical, the physio-pathologic consequences are the same. Besides, it still remains to be proven that injury of the vagus nervus is involved at all. THe syndrome bears much resemblance to that of pyloric stenosis occuring in monogastric animals and men. It may threrfore be suitable to use the term pyloric stenosis.

The dehydration, uremia and the hypochloremic, hypokalemic metabolic alkalosis are the consequences of a stasis (and sequestration) of abomasal contents and a reflux of abomasal contents into the rumen. This reflux causes a decrease in the buffering capacity and an increased chloride content of the rumen fluid (fig. 4). The chloride content of the rumen fluid is a good indicator for abomasal reflux qualitatively and quantitatively. In most cases there is a good relationship between the rumen chloride concentration, plasma bicarbonate concentration and the plasma chloride concentration. The production of hydrochloric acid in the abomasum is accompanied by a bicarbonate production at the blood side. The absorption of

Fig. 3. Fluid and electrolyte balance in a cow with the syndrome of pyloric stenosis due to local peritonitis surrounding reticulum and abomasum.

	blood										Rumen fluid			
	Hb	Ht	pH	pCO_2	BE	bicarb.	urea	Na +	K +	Cl −	pH	buff. cap.	osmol.	Cl
	g/100ml	%		mmHg	meq/l	meq/l	mg/100ml	meq/l	meq/l	meq/l		meq/l	mosm./kg	meq/l
normal values	10-12	30-36	7.35 7.45	35 45	−3 +3	21-26	< 40	135-150	3.9-5.6	100-110	5.7-7.3	80-110	240-280	10-25
D1	14.8	46	7.493	80	20	50	124	144	3.5	45	5.9	9	220	99
D2	12.7	38	7.460	60.5	18	39.4	110	133	2.5	71	6.0	40	225	76
D3	10.2	31	7.401	44.9	2.5	26	40	142	2.4	100	5.9	72	280	49
D11	11.1	33	7.432	40.0	2.2	35.9	21	143	3.8	107	6.2	86	255	16

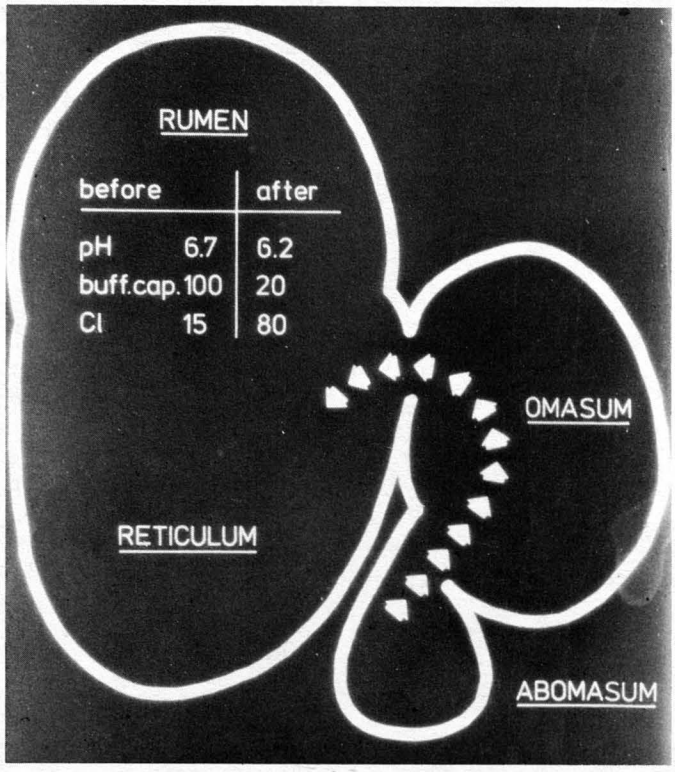

Fig. 4. The changes of pH, buffering capacity and chloride concentrations in the rumen caused by abomasal reflux.

chloride ion in the small intestine in exchange for bicarbonate counteracts any disturbance of the acid-base balance. A failure of ingesta transport from the abomasum to the small intestine causes sequestration of chloride ions in the abomasum and mostly also reflux of abomasal contents into the rumen. A surplus of bicarbonate ions in the blood is the first result.

The reaction of the kidney upon the increased bicarbonate concentration is an excretion of bicarbonate together with sodium, potassium and of course water. There is in this first phase a bicarbonate diuresis and alkaline urine. Due to this diuresis, the chloride sequestration and to the obstruction a severe loss of electrolytes and water occurs. The kidney in the second phase tries to preserve sodium and potassium ionsby exchanging them for hydrogen ions resulting in a parodoxic aciduria, a bicarbonate retention and further increase of the alkalosis. The hypovolemia reduces strongly the effective renal blood flow resulting in uremia.

Metabolic alkalosis also results in hypokalemia because of an increased retention of sodium by the kidney in exchange for potassium and hydrogen ions or because of a shift of potassium ions into the intracellular fluid in exchange for hydrogen ions. Besides the low potassium levels in the blood may also be influenced by the cessation of potassium flow into the small intestine due to the pyloric stenosis and by the urinary loss of potassium in the diuretic phase of the disease.

Differential diagnosis.

The syndrome of pyloric stenosis can be seen in cows with left displaced abomasum (LDA). We examined 100 cows with LDA and found that abomasal reflux had occurred in 80%. Three examples of the results of blood and rumen fluid are given (fig 5).

Cows with abomasal displacement to the right (RDA) frequently show symptoms of pyloric stenosis. In most cases abomasal reflux had occurred (fig. 5).

Cows with abomasal torsion often also suffer from the syndrome of pyloric stenosis. However only in some of the patients reflux of abomasal contents does occur. Others do have a hypochloremia but have no reflux, possibly because of a closure of the omasal-abomasal orifice due to the torsion. The hypochloremia is a consequence of the sequestration of chloride in the overfilled abomasum.

In severe cases of abomasal torsion, the hypochloremic alkalosis is overtaken by a metabolic acidosis due to hypovolemic, toxemic shock (fig. 5).

Clinical diagnosis may not be too difficult in abomasal displacement. This is not true in all cases of obstruction of the small intestine caused by intussusception, obstruction from corpora aliena, strangulation and volvulus. The syndrome of pyloric stenosis is present in all of these cases as in most patients abomasal reflux occurs (fig. 5). Although in most cases rectal exploration produces the exact diagnosis, this may be difficult in cows in late pregnancy, so that an explorative laparotomy is necessary. Apart from the cases where the stenosis is caused by a local peritonitis surrounding reticulum and abomasum as seen in our patient, the syndrome is often present in cows with acute diffuse peritonitis. In these cows acute toxemia, fever or hypothermia, general weakness and an increased tension of the abdominal wall are present. Disgnosis is made by palpating fibrinous adhesions rectally. In most cases abomasal reflux occurs (fig. 5). Examination of the blood further adds to the diagnosis of acute peritonitis (fig. 6). The cause of the obstruction may be partly the involvement of the pyloric region in the process and partly the inhibition of abomasal and intentinal motility due to the toxemia.

An explorative laparotomy will also substantiate a tentative diagnosis of abomasal impaction, another cause of a pyloric stenosis syndrome. Rectally the abomasum may be palpable containing a firm mass of rumen contents like material. The cause of this impaction is mostly not clear. It seems that a disturbance of abomasal motility is involved. Prognosis is mostly poor even after removal of the impaction surgically, relapse often occurs. Abomasal reflux occurs in most cases (fig. 5).

We experienced also a few cases where cows in their last months of pregnancy showed a complete syndrome of pyloric stenosis. These cows have a symmetrically distended abdomen, are anorectic and apathetic. They show severe dehydration and the rumen is overfilled with a thin fluid content. Sometimes splashy sounds can be heard. Intensive

Fig. 5. Fluid and electrolyte balance in cows with the syndrome of pyloric stenosis in blood and rumen fluid.

		Blood										Rumen fluid			
		Hb	Ht	pH	pCO_2	BE	bicarb.	urea	Na^+	K^+	Cl^-	pH	buff. cap.	osmol.	Cl –
		g%	%		mmHg	meq/l	meq/l	mg/100ml	meq/l	meq/l	meq/l		meq/l	mosm./kg	meq/l
normal values		10-12	30-36	7.35 7.45	35-45	–3+3	21-26	40	135-150	3.9-5.6	100-110	5.7-7.3	80-110	240-280	10-25
Diagnosis	cow no.														
LDA	13	12.6	38	7.489	51.8	+13.7	38.7	42	144	3.8	91	5.7	57	230	36
	30	10.6	34	7.415	79.0	+25.7	53.2	91	153	3.3	81	6.6	36	225	72
	23	11.1	30	7.483	41.5	+ 5.4	29.4	22	146	4.5	104	6.8	92	240	21
RDA	8	12.2	36	7.550	51.5	+18.0	40.9	80	147	3.4	81	6.1	42	220	62
	23	12.3	37	7.402	67.9	+14.0	41.9	87	134	2.9	69	6.6	66	250	80
Abomasal	11	14.1	41	7.410	53.1	+ 7.2	33.0	105	141	3.2	90	7.0	75	240	16
torsion	20	16.6	48	7.268	44.6	- 6.9	19.9	114	144	3.8	105	6.8	75	270	46
Intussusception	1	12.2	36	7.476	46.3	+ 9.3	33.0	54	141	3.0	93	6.6	38	--	74
strongulation 13	13	10.9	33	7.563	53.2	+22.4	47.6	64	135	2.2	69	6.7	67	255	51
compressio intestitine? in advanced pregnancy	6	13.4	42	7.480	55.4	+15.4	41.7	114	142	3.2	72	6.0	9	200	55
secundary functional stenosis endometritis mastitis	1	14.5	45	7.512	67.4	+17.4	41.7	98	137	2.9	84	6.1	28	--	77
	2	12.0	38	7.470	45.0	+ 7.8	30.7	186	128	3.0	62	6.1	26	--	78
Diffuse peritonitis	6	10.3	33	7.502	46.8	+11.1	32.9	59	147	2.8	78	6.11	18	--	90
	22	10.6	34	7.550	51.4	+19.7	44.3	135	132	2.7	78	6.53	31	160	48
abomasal impaction	3	11.0	-	7.452	633	+ 6.4	43.4	31	144	3.9	82	5.9	22	160	56
	7	13.6	42	7.546	72.9	+ 34.5	62.7	117	122	2.2	44	6.7	73	180	59
Failure of omasal transport	7	9.6	27	7.410	50.4	+ 5.7	31.5	17	42	3.9	96	7.2	92	260	15
	9	12.6	37	7.340	46.7	- 1.2	24.9	25	142	4.4	99	6.4	84	240	26

reflux has occurred (fig. 5). Blood examination mostly does not give more information. When the cows do calf or abort or when a cesarean section is performed, recovery may be complete in a short while. This indicates that in these cases a compressio intestini by the uterus may be the cause of the failure of abomasal or intestinal transport.

There is a last group of patients that have to be considered in this respect. This is a group of cows with a so called secondary digestive disturbance due to a primary lesion outside the digestive system. It involves cows with endometritis, mastitis, nephritis, bronchopneumonia and liver abcesses (fig. 6). The adynamic ileus is caused by the toxemia. The complicating pyloric stenosis with abomasal reflux (fig. 5) can extremely aggravate the disease and has to be considered and treated along with the primary lesion.

It is important finally that the syndrome of pyloric stenosis is distinguished from that of anterior functional gastric stenosis or failure of omasal transport.

In these cases local inflammatory processes are found with abscess formation in the area of the reticulo-omasal orifice

Fig. 6. Differential diagnostic value of blood examination in cows with obstructive lesions in the gastro-intestinal tract.

Diagnosis	Hb g/100ml	Ht %	WBC x100/ mm³	neutrophils bands %	neutrophils polyhucl.%	lymphoc. %	Cl^- meq/l	urea mg/100	tot. protein g/100ml	albumen %	α glob. %	β glob. %	φ glob. %
normal values	10-12	30-36	50-100	5	25-35	50-60	100-110	40	6-9	40-60	15-17	15-17	35
Local peritonitis surrounding reticulum and abomasum	12.2	37	113	2	56	41	72	137	9.5	30.2	15.6	11.6	42.1
acute diffuse peritonitis	10.8	38	499	36	47	17	61	195	9.4	25.0	22.7	13.8	38.6
late pregnancy "compressio intestini"	13.4	42	87	-	63	34	72	114	9.3	39.2	10.2	12.3	38.4
mastitis, septicaemia and nefritis	9.2	27	206	10	63	27	68	450	5.7	31.9	24.1	11.2	32.8
Failure of omasal transport abcess at omasal orifice.	11.0	33	151	2	50	43	105	25	8.4	41.7	10.6	60	41.7

or the omasal canal. They do not occur frequently but do show anorexia, some dehydration and an overloaded rumen with fluid contents. These cows pass small amounts of dry faeces and often there is some free gas bloat.

In the blood sometimes one finds hemoconcentration, the blood urea concentration is slightly elevated and in most cases indications of a chronic inflammatory proces are present (fig. 6).

There is no alkalosis, the plasma chloride concentrations are normal. There is no abomasal reflux since rumen chloride concentrations remain normal (fig. 5).

Therapy.

Gastrointestinal obstruction is consequence of various lesions of the abomasum and intesttines, primary and secondary. The symptoms of the pyloric stenosis syndrome occurring in these cases may be so dominating that the cause of the stenosis remains unclear.

In any case the metabolic consequences of the obstruction have to be considered when treatment is started. These metabolic consequences can be treated symptomatically by restoring water and electrolyte balance.

Large amounts of saline solution are given intravenously. Postassium is not a necessary substance in these solutions. It may be dangerous in cases where a shift of potassium into the intracellular fluid has occurred. After restoration of the fluid balance with saline, in most cases the hypokalemia has disappeared.

Whenever possible the ruminal contents are siphoned off with a thick rubber stomach tube. This lowers the pressure in the abdomen and makes breathing easier. Moreover it enables a good exploration of the abdomen in cases where surgical treatment is performed.

In cases of local or diffuse peritonitis, antibiotics and heparin are included in the therapy.

When faeces production resumes as the first sign of recovery a transformation is performed with 3-6 liters normal rumen fluid to restore microbial fermentation. In surgical cases close control of water and electrolyte balance is necessary. When the cause of the stenosis is corrected at surgery, but ingesta passage does not resume, fluid and electrolyte treatment has to be continued.

It is possible that after surgical correction of the stenosis, ingesta passage is such that a metabolic acidosis follows because of the large amounts of sequestrated chloride entering the small intestine. In most cases this acidosis is corrected spontaneously.

Conclusions.

In cows with a syndrome of pyloric stenosis, restoration of the disturbances of fluid and electrolyte balance is conditional for a successful treatment surgically or non-surgically. In cases where surgery is not indicated, a prolonged treatment, symptomatically, is quite often rewarded.

References

Kuiper, R. Abomasal reflux in cattle, thesis, Utrecht, 1980.
Further references are available on request.