HOFLUND'S SYNDROME - THE CONSEQUENCE OF FAILURE IN THE SELECTIVE RETENTION OF PARTICLES IN THE RETICULO-RUMEN?

J. Rehage*, N. Stockhofe-Zurwieden**, M. Kaske*** and E. Yalzin*

* Clinic for Diseases of Cattle, School of Veterinary Medicine
** Institute for Pathology, School of Veterinary Medicine
*** Department of Physiology, School of Veterinary Medicine
Bischofsholer Damm 15, D-3000 Hannover 1, Federal Republic of Germany

I) INTRODUCTION

Ruminants achieve a superior digestibility of cell-wall-constituents by retaining feed particles substantially longer in the reticulo-rumen than fluid ("selective retention of particles"). Particles are retained according to their density and size in the forestomach[1]: the probability of small particles (< 3mm) with a high density (1,3 - 1,4 g/ml; mostly well-digested) leaving the reticulo-rumen is about three times greater than for long particles (> 3mm) with a low density[1,2]. The process of particle separation depends mainly on the motility of the reticulum: with the first contraction of the reticulum long particles with a low density are pushed caudodorsally into the rumen (removal from the reticulo-omasal orifice [ROO]); only particles with a high density which are mostly small remain in the reticulum. These particles can leave the reticulo-rumen preferentially during the peak of the second reticular contraction when outflow into the omasum occurs[1,2].

Inflammatory adhesions are found on the floor and wall of the reticulum in cows suffering from traumatic reticuloperitonitis (RPT). Regularly in the feaces of these patients a considerable higher amount of large particles compared to healthy animals is found[3], indicating a disturbance of the normal selective retention of particles in the forestomach[4]. Several patients with traumatic reticuloperitonitis develop the so-called "HOFLUND-syndrome". Characteristic symptoms of such patients are (a) bad general condition with negligible feed intake, (b) no stratification of rumen contents and (c) distention of the rumen[5,6,7]. Prognosis is poor in these patients, although not hopeless. The clinical picture is refered to an until now not defined failure of digesta flow at the level of the ROO (anterior disturbance of digesta passage [AD]) or a failure at the pyloric level (posterior disturbance of digestion passage [PD])[8]. An abomasal reflux with subsequent increase of the chloride concentration in the ruminal liquid (>30 mmol/l) is regarded as pathognomic for a PD[8,9].

Based on his experiments (resection of branches of the N. vagus) HOFLUND postulated in 1940[10], that AD and PD are caused by a damage of the main abdominal vagal nerves (of at least 50%). This hypothesis has been discussed controversially.

The objective of this study was to investigate whether a failure in the ability to retain selectively particles in the reticulo-rumen plays a role in the pathogenesis of HOFLUND's syndrome.

II) PATIENTS

Forty-two cows (German-Black-Pied) suffering from traumatic reticuloperitonits and 10 healthy control animals (mean age 8.7 years) were studied. Patients were grouped as follows:
(a) 17 cows with AD (mean age 4.9 years; selection criteria: enlarged L-shaped rumen, no stratification of ruminal contents, no abomasal

reflux); (b) 10 cows with PD (mean age 6.0 years; enlarged L-shaped rumen, no stratification of rumen contents, abomasal reflux); (c) 15 cows with an uncomplicated RPT (mean age 3.7 years; rumen volume and stratification of rumen contents normal, no abomasal reflux).

Patients with generalised peritonitis, tumors in the area of the abomasum or abscess formation in the liver or close to the ROO were excluded in our study.

III) RESULTS AND DISCUSSION

1. Inflammatory adhesions at the reticulum

During exploratory ruminotomy in all patients the extent of adhesions on the reticular floor, at the reticular wall and around the ruminal atrium were semi-quantitatively assessed.

Inflammatory adhesive alterations on the reticular floor were found in RPT-, AD- and PD-patients being considerably more extensive in the patients with AD and PD than in patients with uncomplicated RPT. Adhesions in the area of the reticular wall were almost exclusively found in patients with AD and PD; these adhesions were evenly distributed over the lateral, cranial and medial wall. No differences were found between patients with AD and PD in respect of the extent and localization of adhesions. Inflammatory changes were found in none of the patients at the cardia or at the level of the ROO.

Postmortal investigations were made in 19 patients which died or were slaughtered throughout the study period. In none of the patients was there evidence, that the main branches of the N. vagus were damaged.

Based on normal anatomical conditions[10,11] our results indicate that the main branches of the dorsal and ventral abdominal N. vagus were most likely not involved in the inflammatory processes in our patients. We therefore conclude that passage disturbances in our patients were not caused by vagal damage.

2. Forestomach motility

Rumen motility was examined by auscultation and palpation in the left dorsal flank. Reticular motility was determined by auscultation in the 6th or 7th intercostal space.

Compared to the healthy control animals (10 contractions/5 min) the number of rumen contractions was significantly reduced in all patient groups; no differences were observed between the groups of patients (mean of AD-, PD-, RPT-patients: 5 contr./5 min) .

The number of reticular contractions was slightly, but not significantly smaller in RPT-patients (3.5 contr./5 min) compared with control animals (5 contr/5 min). In patients with AD and PD almost no reticular contractions were found (AD: 0.6 contr./5 min; PD 0.3 contr./5 min).

The relation of reticular to ruminal contractions was about 1:2 in the control- as well as in the RPT-group. In contrast, in AD-and PD-animals this relation was about 1:10 according to the drastic reduction of reticular movements.

This failure may be due to an immobilization of the reticulum by the extensive adhesions. Results indicate that the reticulum cannot play a significant role in the selective retention of large particles in the reticulo-rumen.

A disturbed function of the intramural nervous system due to inflammatory mediators may also impair reticular motility[12]. Histological investigations were done on slaughtered patients of the

RPT-, AD- and PD-group. Especially in patients of the AD- and PD-group peri- and intraganglionic inflammatory cell infiltration associated with degenerative changes in ganglionic nerve fibers were observed in inflammatory altered reticular areas[12].

3. Particle distribution in the reticulo-rumen and in the faeces

Particles passing the ROO do not change in size during the passage in the lower gastrointestinal tract. By determining particle distribution in the faeces information on particle separation in the reticulum can be obtained[1,2]. Therefore, we estimated in a faeces sample the occurence of particles larger than 5 mm semi-quantitatively. Particle distribution in the reticulo-rumen was assessed during ruminotomy.

An increased amount of large particles in the faeces was found in 73%, 40%, 40% of RPT-, AD- and PD-patients, respectively. Control animals had almost no particles larger than 5 mm in the faeces.

A poorly comminuted faeces in patients suffering from reticulo-peritonitis has been observed repeatedly[3]. Although an almost physiological frequence of reticular contractions was seen in our RPT-patients the first reticular contraction was obviously not strong enough to remove the long particles from the ROO by pushing them caudodorsally.

Due to the more extensive adhesions at the reticulum in patients with AD and PD one could expect a higher amount of large particles in faeces than in RPT-cows. However, we observed a considerably reduced amount of large feed particles in the forestomach contents of AD- and PD-cows compared to RPT-patients. This is most likely due to differences in feed intake between the patient groups. RPT-cows consumed nearly as much feed as the control animals while the feed intake of AD and PD animals was negligible. This may be the reason that an incrased amount of large particles was not found in faeces in all AD- and PD-patients. Furthermore, according to our criteria for patient selection, cows with AD und PD had a drasticly increased forestomach volume compared to RPT- and control animals. Thereby the liquid-matrix-level was considerably above the level of the ROO compared to RPT- or control animals. Thus, the probability of these large floating particles leaving the reticulo-rumen of AD- and PD-patients has to be lower than in RPT-cows.

4. Frothiness of the ruminal contents

In 82% of the AD- and 80% of the PD-patients the loss of stratification of rumen contents was accompanied by frothiness. According to the degree of frothiness, rumen contents developed a viscid consistency throughout in the reticulo-rumen. This observation is already well known from literature[4,5,6].

Several factors are assumed to be involved in the aetiology of frothiness in bloat[13]. There are indications that distribution of particles in the rumen fluid plays a role in the pathogenesis of frothy bloat[13]. However, their is still no convincing explanation.

5. Abomasal size and consistency

Size and consistency of abomasal contents were determined by palpation during exploratory ruminotomy.

Abomasal size was regarded as normal in control animals. The size of the abomasum was reduced in 59%, and it was normal in 41% of the AD-group. In the PD-group, the abomasum was enlarged in 80% of the patients; it was normal in 20%. The size of the abomasum was normal in all cows of the RPT-group.

Consistency of abomasal contents in the control animals was always liquid. In the AD- and RPT-group abomasal contents was mostly liquid (AD: 65%, RPT: 73%), partly pasty (AD: 35%, RPT: 27%). In patients of the PD-group consistency was liquid only in 20%; mostly pasty (60%) or even doughy (20%) abomasal contents was found.

Hoflund (1940) postulated that a failure of the ventral abdominal N. vagus causes disturbances of digesta passage at the pyloric level[10]. However, in vagotomized animals the intramural nervous system is able to maintain a nearly unchanged myoelectric activity of the abomaso-duodenal junction[14,15]. After complete thoracal vagotomy in sheep, transpyloric flow was undisturbed over months[16,17]. Nevertheless, in these animals, an essential precondition for a normal abomasal outflow of digesta was a physiological consistency of abomasal contents (dry matter: < 4%; particle size: < 3mm)[16,17,18]. Also from healthy sheep it is reported, that hydrodynamic factors of the abomasal contents, especially viscosity, are considerably involved in the regulation of transpyloric digesta flow[18]. Even small increases in viscosity cause a serious reduction in abomasal outflow[18].

Thus, a damage to the N. vagus seems not to be involved in AD- and PD-patients. However, in nearly all patients with abomasal reflux (PD-group) an increased size of the abomasum and a firm consistency of the abomasal contents was observed. We assume that the inhibition of abomasal outflow in our patients was caused by the drastic increased viscosity of abomasal contents. A higher vicosity of abomasal contents may be the consequence of an increased viscosity of rumen contents related to altered particle distribution and developing frothiness throughout the reticulo-rumen.

6. Contractility and size of the reticulo-omasal orifice (ROO)

The occurence of an abnormal size and contractility of the ROO (enlarged, low tonus) in patients with a disturbance of digesta passage is regarded as a distinct sign of a damaged vagal nerve[5,6,7]. Therefore we estimated by palpation the size and the contractility of the ROO in our patients during exploratory ruminotomy.

In most patients, size and contractility were comparable to that in control animals. However, an enlarged ROO was found in 35% of the AD-, in 20% of the PD-, in 15% of the RPT-cows. Abnormal findings at the ROO were neither connected with the occurence of abomasal reflux nor with the degree of comminution of the faeces.

In sheep[19] and in lambs[20] spontaneous contractions of the ROO are created by the intramural nervous system. The spontaneous activity of the ROO is modulated by excitatory as well as inhibitory effects of the vagus nerves. Moderate stimulation of the vagus nerves evoke closure, whereas pronounced stimulation leads to loss of spontaneous activity and tone[19,20].

Therefore, the enlarged ROO with a low tone in some of our patients may result from a high tone of an intact N. vagus probably related to stimuli of the slowly adapting mechano-receptors in the forestomach wall by increased rumen size. It is an interesting observation that forestomach volume increases in these patients although the ROO is widely open. This may be due to an insuffient negative pressure in the omasal chanal and/or by inhibition of digesta passage due to an increased viscosity of rumen contents.

IV) CONCLUSIONS

We conclude from our observations that in patients suffering from traumatic reticulo-peritonitis and HOFLUND's syndrome (AD as well as PD-group), the disturbances in digesta passage are primarily caused by a considerable and continous failure of the selective retention of particles in the reticulo-rumen. The failure of the particle separation process is related to the immobilisation of the reticulum due to adhesions. The disturbances in digesta passage result in an altered viscosity of ruminal and abomasal contents. Abomasal contents change from normal (liquid) to a viscosy (pastey) consistency. Thereby transpyloric digesta flow decreases, accumulation of digesta and enlargement of the abomasum occurs, and finally, abomasal reflux is evident.

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SUMMARY:

Forty-two dairy cows suffering from traumatic reticuloperitonitis were studied. Related to the clinical picture and results of clinical biochemistry 15 cows showed a uncomplicated reticuloperitonitis. In 27 cows HOFLUND's syndrom was present. Abomsal reflux occured in 10 of these cows. In none of the patients was there evidence of a damage to the main vagal nerves. The obtained results indicate that: 1.) A failure of the selective retention of particles in the reticulo-rumen in patients suffering from traumatic reticuloperitonitis occurs. This failure is due to the immobilisation of the reticulum by inflammatory adhesive processes. 2.) The disturbance in particle separation in the forestomachs is involved in the pathogenesis of HOFLUND's syndrome. 3.) The distinction between patients with an anterior or a posterior disturbance of digesta passage as syndroms of different pathogenesis is not justified. The primary disease-causing process is the developing of a disturbance of digesta outflow from the reticulo-rumen.

ZUSAMMENFASSUNG:

Es wurden 42 an Reticuloperitonitis traumatica erkrankte Milchkühe, von denen 27 Patienten gleichzeitig das klinische Bild des HOFLUND-Syndromes aufwiesen, sowie 10 gesunde Kontrolltiere untersucht. Bei 10 der am HOFLUND-Syndrom erkrankten Kühe wurde ein abomasaler Reflux festgestellt. Bei keinem der am HOFLUND-Syndrom erkrankten Kühe lagen Hinweise auf eine ursächliche Beteiligung des N.vagus am Krankheitsgeschehen vor. Klinische, laborklinische und Befunde der explorativen Ruminotomie werden beschrieben und diskutiert. Aus den Befunden wird geschlossen, daß bei Patienten mit Reticuloperitonitis, infolge der durch adhäsive Prozesse eingeschränkten Haubenmotilität die Separation von Partikeln in den Vormägen gestört ist. Dieses scheint eine entscheidende Rolle in der Pathogenese des HOFLUND-Syndroms zu spielen. Ferner, eine Störung des transpylorischen Ingestaflusses, gekennzeichnet durch das Auftreten eines abomasalen Refluxes, definiert nicht ein eigenständiges Krankheitsbild, sondern ist als Eintritt in ein neues Erkrankungsstadium des HOFLUND-Syndroms aufzufassen.

RESUMEN:

Se analizaron 42 vacas de ordeño con diagnóstico de retículo peritonítis traumática. 27 de éstos presentaron en forma evidente el cusdro clínico del sindrome de HOFLUND. Como control se consideraron 10 animales sanos. En 10 vacas con el sindrome de HOFLUND se constató un reflujo abomasal, pero ninguna de las 27 presentó lesiones que indiquen participación de N. vago. Se informan y se discuten los hallazgos de los exámenes de laboratorio y de las rumenotomías exploratorias. De los hallazgos se concluye que en pacientes con retículo-peritonitis se producen adherencias a nivel del retículo que entraban la separación de las patículas a nivel de los preestómagos. Esto podría desempeñar un papel importante en la patogenia del sindrome de HOFLUND. En consecuencia un bloquéo del reflujo transpilórico como consecuencia de un reflujo abomasal, no define una entidad patológica, sino que es considerado como una fase del sindrome de HOFLUND.