MORPHOLOGICAL INVESTIGATIONS OF THE FORESTOMACH IN COWS SUFFERING FROM HOFLUND SYNDROME

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Since HOFLUND`s explanation (1) of a functional stenosis of either the bovine forestomach or the pylorus due to damages of the vagal nerve in 1940, this hypothesis is discussed controversially. In addition disturbances of the beforementioned extrinsic nerve system, mechanical influences are discussed to be the origin of this disease (2,3).
As an extrinsic control system the vagal nerves are responsible for a coordionated contraction of the ruminant forestomach. These nerves originating from the gastric centre in the medulla oblongata are consisting of predominantly afferent fibres and some efferent, preganglionic nerve fibres (4). The efferent fibres are communicating with intramurally located ganglia. Recently published data present that the intramural neurons, also named as intrinsic nerve system are able to initiate reticulo-ruminal contraction (9,10).
Purpose of this study was to evaluate morphological alterations of vagal and intramural nerve fibres, intramural neurons and inflamamtory changes in the wall of the forestomachs and abomasum of cows suffering from HOFLUND`s Syndrome.

MATERIALS AND METHODS

Four different groups of animals were investigated:
1. cows without any clinical signs of a digestive tract disease (n=10). 2. patients with "vagus indigestion" at the level of the reticulo-omasal orifice (cranial functional stenosis) (n=7); 3. patients with "vagus indigestion" at the level of the pylorus (pyloric fuctional stenosis) (n=6); 4. patients with traumatic reticuloperitonitis without any sign of a failure in ingesta passage (n=6). In group 2 and 3 only those cows were selected, which had a cranial or pyloric functional stenosis in combination with inflammatory, adhesive processes at the reticulum.
Tissue sammples were obtained from seven defined tissue areas:
A: right N. vagus beside the esophageus at the level of the apertura thoracis cranialis; B: Cardia; C: reticular groove; D: ventral part of the reticular-omasal orifice; E: omasal channel; F: pylorus G: altered areas in fundus reticuli. Not knowing at the beginning of the investigations where exactly to take the samples of the fundus reticuli in the control animals, additional tissue samples were taken from 7 cows not having any disease of the alimentary tract.
Tissue samples were fixed in 10% buffered formalin and tissue sections were stained with hematoxylin-eosin and with a modified silver impregnation (UNGEWITTER) (5). Additionally in the reticulum an immunohistochemical reaction with antibodies against neurofilament protein 200kD (ABC-method, working solution 1 : 400) was performed.

RESULTS

Macroscopic findings

Whereas in the control animals no macroscopic lesions in the forestomach were observed, all animals of group 2, 3 and 4 had inflammatory alterations in varying degrees at the reticulum. In none of the animals inflammatory or tumorous changes were found along the vagal nerves. The amount of adhesions at the reticulum is described in detail elsewhere (6). In contrast to the animals with reticuloperitonitis, which mainly had inflammatory adhesions at the floor of the reticulum, the animals of group 2 and 3 had extensive adhesions as well at the reticular floor as at the wall of the fundus reticuli. Adhesions were found in comparable amounts at the medial, the lateral and the cranial wall of the reticulum.

Histological findings

The vagal nerves examined at their thoracical parts and at the cardia did not reveal any differences within the 4 groups. Only in some instances axon swelling or swelling of the myelin sheat were observed in all groups.

In tissue samples from the forestomach intramural ganglia were only present between the tunica muscularis (plexus myentericus), a plexus submucosus was only found in the pyloric region. In all groups a small amount of degenerating neurons, characterized by vacuolisation of the cytoplasm or an increased argyrophilia and cell pycnosis was present.

Within the fundus reticuli the findings in the control group were comparable to those of the other forestomach localisations. In groups 2,3 and 4 subacute and chronic inflammatory alterations and granulation tissue formation started out from the serosa. Especially in both groups with failure in ingesta passage inflammation extended into the tunica muscularis separating the longitudinal and circular muscle layer. Inflammatory cells predominantly mononuclear but also granulocytic cells infiltrated into ganglia. About 1/3 to 1/2 of all neurons were surrounded by inflammatory cells and a statistically not significant diminished amount of neurons related to the surface of the tunica muscularis was observed. Within the ganglia unmyelinated nerve fibres showed axon swelling and disorganization of fibre bundles.

DISCUSSION

Traumatic reticuloperitonitis is regarded to be the most common cause of HOFLUND syndrome in cattle (7). In accordance with the literature in our cases inflammatory changes were observed predominantly at the reticulum, but we could not find any preferential site at the reticulum wall. In contrast to the group with acute reticuloperitonitis (group 4) lesions spread to all parts of the wall of the fundus reticuli. Disturbances of the motility of the reticulum seem to be a major cause in the pathogenesis of HOFLUND syndrome (6). Under the aspect of experimental data revealing the induction of a "vagus indigestion" by sectioning certain nerve branches,.the vagal nerves at the reticulum or the esophageal groove are discussed to be affected due to the inflammatory processes (1). Other authors ascribe the impaired stomach motilitiy to mechanical factors resulting from adhesions. Only in a few clinical cases an involvement of the vagal nerve was demonstrated (8) and quite often only adhesive processes were observed. Examination of vagal nerves at the cardia and of a main branch of the thoracic vagal nerve did not reveal any difference to the healthy control group. Intramural ganglia are important nervous structures responsible for the comunication between efferent vagal

nerve fibres and effector cells, i.e. in the case of the forestomach for a coordinated motility. In our cases with HOFLUND syndrome no alteration of ganglia in the cardia, esophageal groove, omasal orifice and channel was observed. But in the altered areas of the reticular wall a peri- and intraganglionic infiltration with inflammatory cells combined with degenerative changes in the nerve fibres and a probable loss of neurons suggest a possible role of these nervous structures to induce the immobilisation of the reticulum.

SUMMARY

The pathogenesis of HOFLUND syndrome is discussed in the literature controversially. Damge of vagal nerves due to inflammatory processes or mechanical factors resulting from inflammatory adhesive alterations in the forestomach wall are the generally discussed causes of the disease. Purpose of these investigations were to evaluate alterations of intamural ganglia, nerve fibres and N. vagus in cows suffering from Hoflund syndrome. These were carried out in four groups of cows: 1) a healthy control group 2) animals with cranial functional stenosis 3) patients with pyloric functional stenosis 4) cows with acute reticuloperitonitis. Macroscopically most striking were adhesive chronic inflammatory processes at the reticulum floor and wall, which extended significantly more in those animals with functional stenosis than in those with reticuloperitonitis. In no case macroscopical or histological alterations of the thoracical vagal nerve and cardia surrounding nerves were observed. Microscopically in 5 defined areas (cardia, esophageal groove, omasal orifice, omasal channel, pylorus) of the forestomach/abomasum no differences between the groups were observed. Within areas of the reticular wall altered by chronic inflammation a peri- and intraganglionic inflammatory cell infiltration associated with degenerative changes in ganglionic nerve fibres was observed. It is concluded that apart from mechanical causes inflammatory alterations of the ganglionic neurons may play an important role in the immobilisation of the reticulum and therefore in the pathogenesis of HOFLUND syndrome.

ZUSAMMENFASSUNG

Die Pathogenese des Hoflund Syndromes wird kontrovers diskutiert. Vornehmlich werden entweder Schäden des N. vagus infolge entzündlicher Veränderungen oder mechanische Faktoren als Resultat chronisch adhäsiver Prozesse als Ursachen der Erkrankung angesehen. Im Rahmen dieser Studie sollten Störungen der intramuralen Ganglien, intramuraler Nervenfasern und des N. vagus bei Tieren mit dem klinischen Bild des HOFLUND syndroms untersucht werden. Die Untersuchungen wurden an 4 Gruppen durchgeführt: 1) klinisch gesunde Tiere; 2) Kühe mit vorderer funktioneller Stenose; 3) Kühe mit hinterer funktioneller Stenose; 4) Patienten mit Retikuloperitonitis traumatica ohne Anzeichen einer Passagestörung. Makroskopisch auffälligster Befund waren in den Gruppen 2,3 und 4 chronisch-entzündliche, adhäsive Prozesse im Bereich des Fundus reticuli. Während sie sich bei den Tieren mit Retikuloperitonitis im Bereich des Haubenbodens befanden, erstreckten sie sich bei den Tieren mit Passagestörung auf die Wand des Retikulums. In keinem Fall konnten makroskopische oder histologische Alterationen weder des thorakal gelegenen N. vagus noch der die Kardia umgebenden Nerven beobachtet werden. In fünf definierten Abschnitten des Vormagen/Magen (Kardia, Schlundrinne, Psalteröffnung, Psalterkanal, Pylorus) konnten keine Unterschiede bezüglich der Ganglia zwischen den Gruppen festgestellt werden. In den durch entzündlich-adhäsive Prozesse veränderten

Bereichen der Netzmagenwand zeigte sich eine peri- und intraganlionäre Entzündung mit degenerativen Veränderungen ganglionärer Nervenfasern. Aus diesen Ergebnissen kann gefolgert werden, daß neben mechanischen Ursachen entzündliche Veränderungen in den Ganglien eine wichtige Rolle bei der Immobilisierung des Netzmagens und damit in der Pathogenese des HOFLUND Syndroms spielen können.

RESUMEN

La patogénesis del síndrome de HOFLUND está discutida de modo controversial. En primer lugar se consideran como causa de la enfermedad bién lesiones del nervio vago como consecuencia de alteraciones inflamatorias ó bién factores mecánicos como resultado de procesos crónicos adhesivos. En este estudio se investigan trastornos de ganglios intramurales y del nervio vago en animales con los rasgos clínicos del síndrome de HOFLUND. La investigación se lleva a cabo en cuatro grupos: 1. animales sanos desde el punto de vista clínico; 2. vacas con estenosis anterior funcional; 3. vacas con estenosis posterior funcional; 4. pacientes con reticuloperitonitis traumática sin síntomas de trastorno de pasaje. El resultado mas llamativo de carácter macroscópico fueron procesos crónicos-inflamatorios adhesivos en la parte del fondo del retículo en los grupos 2,3 y 4. Mientras que en los animales con reticuloperitonitis se encontraban en la parte basal del fondo reticuli, en los animales con trastornos del pasaje se encontraban en los uros del retículo. En ningún caso se podían observar alteraciones macroscópicos ó histológicas ni en la parte torácica del nervio vago ni en los nervios alrededor del cárdias. En 5 localizaciones definidas del pre-estómago/estómago (cárdias, gotera esofágica, orificio omasal, canal omasal, píloro) no se podían encontrar diferencias respecto a los ganglios entre los grupos. En las partes del muro del fondo reticuli alteradas por procesos inflamatorios adhesivos se observaba una inflamación peri- e intraganglionar con alteraciones degenerativas de fibras nerviosas ganglionares. De estos resultados se puede concluir que, aparte de causas mecánicas, también alteraciones inflamatorias en los ganglios pueden jugar un papel importante en la inmovilización del retículo y de este modo en la patogénesis del síndrome de HOFLUND.

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