



Sveriges lantbruksuniversitet  
Swedish University of Agricultural Sciences

**Faculty of Veterinary Medicine  
and Animal Science**  
Department of Clinical Sciences

# **Peritonitis in Horses – a Retrospective Study of 69 Cases Admitted to a University Hospital During a Ten Year Period**

*Zandra Lundberg*

*Uppsala  
2014*

*Degree Project 30 credits within the Veterinary Medicine Programme*

*ISSN 1652-8697  
Examensarbete 2014:41*



# **Peritonitis in Horses – a Retrospective Study of 69 Cases Admitted to a University Hospital During a Ten Year Period**

## **Peritonit hos Häst – en Retrospektiv Studie av 69 fall behandlade på ett Universtetssjukhus under en Tioårsperiod**

*Zandra Lundberg*

**Supervisor:** *Pia Haubro-Andersen, Department of Clinical Sciences*

**Assistant Supervisor:** *Johan Bröjer, Department of Clinical Sciences;  
John Pringle, Department of Clinical Sciences*

**Examiner:** *Miia Riihimäki, Department of Clinical Sciences*

*Degree Project in Veterinary Medicine*

**Credits:** *30 hec*

**Level:** *Second cycle, A2E*

**Course code:** *EX0736*

**Place of publication:** *Uppsala*

**Year of publication:** *2014*

**Number of part of series:** *Examensarbete 2014:41*

**ISSN:** *1652-8697*

**Online publication:** <http://stud.epsilon.slu.se>

**Key words:** *peritonitis, horse, abdominocentesis, primary, secondary*

**Nyckelord:** *peritonit, häst, bukhinneinflammation, primär, sekundär (key words in Swedish)*

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## SUMMARY

Peritonitis is a potentially life-threatening disease in horses. With no published work from Sweden, the objective of the study was to describe the occurrence and demographics of horses with peritonitis and to evaluate the outcome of treatment in terms of short-term survival during a ten-year period in a large clinic in Sweden.

Data were examined in a retrospective manner in 69 horses diagnosed with and treated for peritonitis at Universitetsdjursjukhuset (UDS) in Uppsala between 2002-2012. Demographic data examined included age, breed, gender, history, duration of illness before arriving at UDS, reason for seeking veterinary care, initial clinical findings (general state of health, heart rate, respiratory rate, mucous membrane appearance, rectal temperature, abdominal sounds, rectal examination, nasogastric tube results), abdominocentesis results (abdominal fluid analysis including visual inspection, leukocytes and protein, cytology, bacterial culture and sensitivity pattern), complete blood count (CBC), Serum amyloid-A (SAA) upon presentation and a follow up, plasma fibrinogen, plasma protein and albumin, treatments, length of hospitalisation and outcome.

All medical records with the diagnosis of peritonitis were extracted from the medical records system Trofast. All records with any other diagnosis code referring to trauma such as rectal tear or ruptured uterus, recent abdominal surgery, external trauma or rupture in the gastrointestinal tract were excluded. To be included in the study, the peritoneal fluid should contain more than 20.000 cells/ $\mu$ L, have a peritoneal protein value of  $>30$  g/L; or have a significantly changed peritoneal fluid sample (orange with increased turbidity or worse) in cases where no data on cells or protein were available.

The horses were divided into two groups; one where the peritonitis was deemed to have an idiopathic aetiology (primary peritonitis) and the other group where the peritonitis had a possible aetiology such as intestinal parasites, impaction etc. (secondary peritonitis). Of the 69 horses in total that matched the criteria for inclusion, 59 survived to be discharged (86%). Of the primary cases, three out of 21 were euthanized (14%) and of the other 48 horses with anticipated secondary peritonitis, seven were euthanized (15%). There was no significant difference between the two groups regarding short-term survival.

There were significant differences in clinical parameters between the survivors and non-survivors. Non-survivors presented with pyrexia ( $p<0.0085$ ) and depression before coming to the clinic more often than survivors ( $p<0.0050$ ), they had higher heart rates ( $p<0.0001$ ), more often gastric reflux ( $p<0.0007$ ), presented with a higher degree of altered abdominocentesis results when visually examined ( $p<0.0001$ ) and more often presented with normal neutrophil morphology ( $p=0.0004$ ) when analysing the complete blood count. Surprisingly, there were two clinical parameters positively associated with survival. Survivors were more prone to display colic before arriving at the clinic ( $p=0.0013$ ) and depression upon presentation at the clinic ( $p=0.0171$ ) than non-survivors.

## SAMMANFATTNING

Peritonit, även kallad bukhinneinflammation, är en potentiellt livshotande sjukdom som drabbar hästar. Då ingen studie är utförd under svenska förhållanden vad gäller denna sjukdom var målet med studien att beskriva förekomst och demografiska förhållanden hos hästar med peritonit under en tioårsperiod på ett stort hästdjursjukhus i Sverige. Vi ville även utvärdera utfallet av behandlingen gällande korttidsöverlevnad, dvs. att de skrevs ut från kliniken.

Data undersöktes retrospektivt från 69 hästar som diagnosticerats med och behandlats för peritonit på Universitetsdjursjukhuset (UDS) i Uppsala mellan 2002-2012. Data som undersöktes inkluderade ålder, ras, kön, sjukdomsduration, anamnes, initiala kliniska fynd (allmäntillstånd, hjärtfrekvens, andningsfrekvens, slemhinneutseende, temperatur, ljud från buken, rektalundersökning och näs-svalg-sondning), bukpunktat (bukvätskeanalys inkluderade okulär bedömning, leukocyt- och proteinmängd, cytologi, bakterieodling och resistensbestämning av bakterier), hematologi, Serum amyloid-A (SAA) vid ankomst och vid uppföljning, plasma-fibrinogennivåer, plasmaprotein- och albuminnivåer, behandlingar, längd på sjukhusvistelse och utfall.

Alla journaler med diagnosen peritonit valdes ut ur journalsystemet Trofast. Alla journaler som även hade en diagnoskod som hänvisade till trauma såsom rupturerad uterus, nyligen utförd bukkirurgi, externt trauma eller ruptur i magtarmsystemet exkluderades. För att inkluderas i studien skulle peritonealvätskan innehålla mer än 20.000 celler/ $\mu\text{L}$ , ha ett proteinvärde  $>30$  g/L eller ha ett signifikant förändrat peritonealvätskeprov (orange med ökad turbiditet eller värre) i de fall där det inte fanns någon data om celler eller protein.

Hästarna delades in i två grupper; en där peritoniten ansågs ha en idiopatisk etiologi (primär peritonit) och en annan grupp där det fanns en möjlig etiologi såsom invärtes parasiter, förstoppning etc. (sekundär peritonit). Av de 69 inkluderade hästarna överlevde 59 stycken (86%) för att bli utskrivna från kliniken. Av de som hade primär peritonit avlivades tre av 21 (14%) och av de 48 som hade sekundär peritonit avlivades sju stycken (15%). Det fanns ingen signifikant skillnad mellan de båda grupperna vad gäller korttidsöverlevnad.

Det fanns signifikanta skillnader i de kliniska parametrarna mellan de som överlevde och de som avlivades. De som avlivades uppvisade oftare feber ( $p<0.0085$ ) och nedsatt allmäntillstånd i anamnesen ( $p<0.0005$ ) än de som överlevde, de hade högre hjärtfrekvens ( $p<0.0001$ ), oftare magsäcksöverfyllnad ( $p<0.0007$ ), uppvisade i högre grad ett visuellt förändrat peritonealvätskeprov ( $p<0.0001$ ) och hade oftare normala neutrofiler ( $p=0.0004$ ) på hematologin. Två kliniska parametrar var positivt associerade med överlevnad. De som överlevde visade oftare tecken på kolik innan ankomst till kliniken ( $p=0.0013$ ) och nedsatt allmäntillstånd vid ankomst till kliniken ( $p=0.0171$ ).

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### *Abbreviations*

SAA – serum amyloid A

WBC – white blood cell

CBC – complete blood count

NSAID – non-steroidal anti-inflammatory drugs

CRI – constant rate infusion

## **INTRODUCTION**

This study was initiated due to the fact that there seem to be quite a large number of horses in the clinic at Universitetsdjursjukhuset (UDS) being diagnosed with peritonitis, which seems to otherwise be rare in this part of the world (Dart & Bischofberger, 2011). There also seems to be a favourable prognosis for these animals in the clinic and to find out if this was accurate, this retrospective study was initiated.

Doing this study at a referral hospital such as UDS meant that there were many patient records available for analysis. During a normal year, approximately 4000 patients are seen, diagnosed and treated at the clinic.

There has been no Swedish research published in this subject, which makes it even more interesting to present our situation and our approach to treatment when it comes to peritonitis.

### **Aim**

The aim of the study was to describe the occurrence, demographics and clinical parameters of horses with peritonitis and to evaluate the outcome of treatment in terms of short-term survival. Possible differences between survivors and non-survivors were investigated.

In that context, it was also interesting to determine how many of the patients diagnosed with peritonitis could be termed primary (no known aetiology) and if that had any impact on short-term survival.

One specific hypothesis was that the treatment with NSAIDs due to any other illness could be related to or even cause peritonitis.

After a presentation of peritonitis and treatment thereof, the results of the study will be presented.

## **LITERATURE REVIEW**

### **Peritonitis**

Peritonitis is a condition where the mesothelial lining of the peritoneal cavity is inflamed. The inflammation can occur due to mechanical trauma, chemical stress or infectious agents (Reed *et al.*, 2010). Peritonitis is characterised by the exudation of fibrin, protein, inflammatory cells and serum into the peritoneal cavity (Hillyer & Wright, 1997; Davis 2003).

Peritonitis is divided into categories according to the inciting cause – primary or secondary, the degree of involvement of the peritoneum – generalised or localised, the severity and duration of clinical signs – peracute, acute or chronic and the presence of bacteria – septic or sterile (Rose & Hodgson, 1993).



Among the different aetiologies of peritonitis there are gastrointestinal causes such as intestinal ischemia, intestinal perforation, rectal tear, surgery, intestinal neoplasia, haemorrhage and intestinal parasites (Dyson, 1983; Hillyer & Wright, 1997, Davis, 2003). Non-gastrointestinal causes include ruptured bladder, vaginal tear, disseminated (bastard) strangles, acute pancreatitis, neonatal septicaemia and penetrating wound to the abdomen (Hillyer & Wright, 1997, Davis, 2003).

### *Clinical signs*

Clinical signs of peritonitis include fever, abdominal discomfort, reluctance to move, signs of endotoxemia, increased heart rate and respiratory rate, decreased borborygmia, and dehydration (Rose & Hodgson, 1993; Hillyer & Wright, 1999; Davis 2003; Reed *et al.*, 2010)

### *Diagnosis*

To diagnose peritonitis, the abdominocentesis and examination of peritoneal fluid is imperative (Reed *et al.*, 2010). It is the composition and change in peritoneal fluid that determines whether or not the horse has peritonitis since the clinical signs often presented can be attributed to a great number of conditions not requiring the same treatment. Ocular examination as to colour and turbidity should be performed as well as cytological examination regarding increase in white blood cell count, differential and the presence of bacteria. Biochemical analysis regarding total protein should be analysed and bacterial culture should be performed (Reed *et al.*, 2010).

The cut-off value for clinical diagnosis of peritonitis varies in different publications. Normal WBC in peritoneal fluid is <9000 cells/ $\mu$ L and protein levels range between 1-34g/L (Bach & Ricketts, 1974). According to Orsini & Divers (2008) and Reed *et al.* (2010), the normal WBC in peritoneal fluid is <5000 cells/ $\mu$ L and protein levels should be <25g/L. Other published authors have, in their studies, included all horses with WBC >5000 cells/ $\mu$ L (Hillyer & Wright, 1997; Henderson *et al.*, 2008; N6grádi *et al.*, 2011; Watts *et al.*, 2011). It is up to the clinician to decide which of these elevated cut-off values to use on a daily basis.

It is important to include history as part of the clinical examination of these horses. They have often shown signs of fever, depression, colic or anorexia. These symptoms may be general and/or intermittent but should point the clinician in the direction of an abdominocentesis.

Peritonitis is deemed primary when no aetiology can be found during the clinical evaluation of the horse. It is believed that the peritonitis in these horses is due to the impaired host defences, which make it possible for bacteria to enter the peritoneal cavity and cause peritonitis (Hanson, 1999).

### *Treatment*

It is important to treat any horse suspected with peritonitis aggressively and promptly (Davis, 2003) since treatment does not vary significantly between different aetiologies.

Treatment consists of different strategies to eliminate the primary problem, inhibit any further development of endotoxemia, restoring electrolyte and fluid imbalances and manage the pain associated with peritonitis. According to reference literature, broad-spectrum antimicrobial treatment should be initiated upon confirmation of peritonitis and even prior to bacterial culture results since the culture takes some time and there's the risk of a false negative result (Hawkins *et al.*, 1993; Davis, 2003; Reed *et al.*, 2010).

### *Treatment of endotoxemia*

Many horses with peritonitis present with signs of endotoxemia and it is important to inhibit any further development of endotoxins (Henderson, 1999; Davies, 2003).

One of the most commonly used treatments for horses with inflammation or infection causing the release of endotoxins is NSAIDs (non-steroidal anti-inflammatory drugs). They inhibit cyclooxygenase (COX) and thereby inhibit the production of prostanoids (Hanson, 1999; Davis, 2003; Reed *et al.*, 2010). Prostanoids are a biologically active group that can be divided into prostaglandins, prostacyclins and thromboxans and they are all part of the inflammatory response (<http://lipidlibrary.aocs.org/Lipids/eicprost/index.htm>, 2013-11-29). Thus by treating the animals with NSAIDs, the inflammation is suppressed. NSAIDs also provide analgesia for horses that present with abdominal discomfort (Rose & Hodgson, 1993; Matthews *et al.*, 2001; Davis, 2003).

The hypothesis that NSAIDs could be related to the development of peritonitis was based on the fact that they can cause gastrointestinal irritation and ulcers as a side effect (FASS *vet.* 2013). Gastrointestinal irritation could perhaps be a solitary and sufficient factor to initiate the process of peritonitis.

There are many other treatments suggested, amongst others, the endotoxin-binding agent Polymyxin B which can be combined with the antimicrobial treatment, anti-inflammatory corticosteroids, antioxidants to help with oxygen-derived radicals and lidocaine to provide analgesia and enhance the inhibitory effect of the flunixin on mucosal barrier function (Reed *et al.*, 2010). These treatments will not be further investigated or presented in this essay.

When treated with NSAIDs, it's important to keep the animal from becoming dehydrated or restore any loss of fluid. Fluid therapy should be administered if the underlying condition, causing the endotoxemia, poses a risk of causing dehydration since NSAIDs have a potential nephrotoxic effect if the animal is dehydrated or hypovolemic (FASS VET. 2013).

Treatments of peritonitis that also can be considered include abdominal drainage and lavage, abdominal surgery, treatments to resolve impaction and anthelmintic treatment if indicated.

### *Prognosis*

Depending on the causing factor, survival rates vary. The prognosis for horses with gastrointestinal rupture is grave and animals with peritonitis due to abdominal surgery have a high mortality rate, up to 56% (Hawkins *et al.*, 1993; Davis, 2003; Reed *et al.*, 2010).

However, horses with peritonitis due to *Actinobacillus* spp. have a good prognosis, usually with 100% survival rate (Matthews *et al.*, 2001; Watts *et al.*, 2011). Prognosis depends on the underlying cause of the illness and the patient should therefore be treated on an individual basis.

### **Abdominocentesis**

Abdominal paracentesis is essential when diagnosing peritonitis. In their article regarding paracentesis from 1974, Bach and Ricketts describe the method of performing paracentesis as a diagnostic tool in the horse. Presented in the article are the normal constituents of peritoneal fluid, among others, levels of nucleated cell count, distribution of white blood cells and protein levels.

Another article describing the course of action needed to diagnose peritonitis and explaining abdominal paracentesis in detail and with images is “Peritonitis in the horse” by Hillyer & Wright (1997). This can be of aid to the clinician since the pictures in the article are illustrative and in colour.

There are not many articles published in this field. For this study, eleven articles were identified using the databases Web of knowledge and Science Direct and the search words were “horse”, “peritonitis”, “paracentesis” and “study”. All eleven articles have their own inclusion and exclusion criteria and the results are therefore difficult to compare. Some have included cases with ruptured intestine, others peritonitis due to abdominal surgery and others have focused solely on a single bacterial aetiology.

The articles mentioned above have all been of great help when writing this essay even though not describing the exact same phenomenon. In this study, the cut-off value for white blood cells in peritoneal fluid was set at a higher level than all of the articles mentioned above to ensure that all cases are in fact peritonitis in need of treatment. Also, the cut-off value for protein levels in peritoneal fluid was set at a higher level in this study than all of the other articles referred to. The cut-off values are stated in materials and methods.

### **Acute phase proteins**

As a response to inflammation, the body synthesises a great number of *acute phase proteins*, mainly in the liver. When shifting focus in the presence of inflammation, the liver decreases the synthesis of albumin and hypoalbuminemia may be the result. This shift can be detected by taking a simple blood sample where low levels of albumin may be detected if the acute phase response is strong and has persisted for a while. Acute phase proteins of interest are fibrinogen and serum amyloid-A (SAA) (Reed *et al.*, 2010). These proteins increase due to inflammation. Fibrinogen is rapidly present in the bloodstream but then has a slow increase, whereas SAA increases rapidly and substantially in the presence of inflammation (Jacobsen & Andersen, 2007).

## **MATERIALS AND METHODS**

The material consisted of medical records from the Horse Clinic at Universitetsdjursjukhuset (UDS) in Uppsala between January 2002 and December 2012 where the final diagnosis had been noted as peritonitis or acute purulent peritonitis in the medical records system Trofast. The diagnosis is entered by one of the treating veterinarians using codes and the diagnosis code is then available for selection and data collection. Using this timeframe made it possible to have records of full years and as recent data as possible.

### **Selection criteria**

The first retrieval, based on the two diagnosis codes of interest being acute purulent peritonitis and peritonitis, resulted in 108 cases during this time period. In the system of Trofast, the veterinarian can choose to note one or more diagnose codes for one patient. This first retrieval was based on the diagnosis being peritonitis noted as first, second or third diagnosis for that patient. Based on the diagnosis code, the horses that had a history of trauma resulting in peritonitis were then excluded since the aim was to find the patients with primary peritonitis. The traumatic causes include ruptured uterus, external trauma, abdominal surgery, ruptured gastrointestinal tract, complications due to castration etc. One donkey was diagnosed with peritonitis but was excluded from the study due to potential species differences in clinical parameters.

Based on the above literature review, cut-off values were selected for the number of leukocytes and protein in the peritoneal fluid. To be included in the study the peritoneal fluid should have a nucleated cell count:  $>20000$  cells/ $\mu$ L and total protein content:  $>30$ g/L. If one of these criteria were met, the patient was included.

If a record did not have any values for protein or WBC in the peritoneal fluid, the case was included if the peritoneal fluid had a significant increase in turbidity and significant change in colour (orange or hemorrhagic). If the record did not meet any of these three criteria, it was excluded from the study.

### **Data collection**

The data collected included age, breed, gender, duration of illness before arriving at UDS, reason for seeking veterinary care, prior treatment with NSAIDs, initial clinical findings (general state of health, heart rate, respiratory rate, mucous membrane appearance, rectal temperature, abdominal sounds, rectal examination, nasogastric tube results), abdominocentesis results (abdominal fluid analysis including leukocytes and protein, cytology, bacterial culture and sensitivity pattern) complete blood count (CBC), Serum amyloid-A (SAA) upon presentation and a follow up, plasma fibrinogen, plasma protein and albumin, treatments, length of hospitalisation and outcome. Long-term follow-up for horses discharged from the clinic were not obtained.

## **Diagnostic criteria for primary vs. secondary peritonitis**

Horses with aetiologies, which could explain a secondary peritonitis, such as impaction, intestinal parasites, ileus and abnormal rectal findings were allocated to one group termed secondary peritonitis. The remaining cases, where no aetiology could be identified, were allocated to the primary peritonitis group.

## **Demographic data for comparison of populations**

Gender, age and breed data were collected from the entire hospital population during the same period of time to investigate the proportions of the values when compared to the cases with peritonitis in the clinic. Horses with multiple entries to the hospital were allowed once a year.

## **Statistics**

Age, breed and gender proportions were compared using Fisher's exact test. Clinical parameters, such as pyrexia, colic, depression, heart rate, respiratory rate, mucous membranes, rectal exam, visual alteration of the abdominocentesis, gastric reflux and positive bacterial culture were compared between non-survivors and survivors using Fisher's exact test. Significance was set at  $p < 0.05$  for each comparison.

## **RESULTS**

A total of 108 horses matched the initial selection criteria having been diagnosed with peritonitis during the period of 2002-2012 at UDS. Any patient with a recent history of abdominal surgery was excluded and any patient with ruptured intestine, uterus or complication after castration was excluded. Of the remaining 80 horses, 11 horses were excluded due to the fact that they did not make the inclusion criteria regarding the levels of protein or WBC in the abdominocentesis or the colour of the abdominocentesis was not altered enough. Left in the group were 69 horses that met the criteria stated above.

In the group deemed primary or idiopathic, 21 horses were included (30%). In the group with secondary peritonitis, 48 horses were included (70%).

The descriptive results of both groups (primary and secondary) will be presented together. Any differences in results will be presented in the discussion.

### ***Gender, age, breed***

#### *Gender*

Twenty-seven females, 36 geldings and six intact males were included in the study. There was no significant difference between the gender distribution of horses with peritonitis and the general hospital population.

#### *Age*

Range of age was 6 months to 30 years, average age 11.4 years and median age 11 years. For the distribution, see figure 1. The age distribution appears to be normal, though a small number of cases in this study made the distribution uneven.

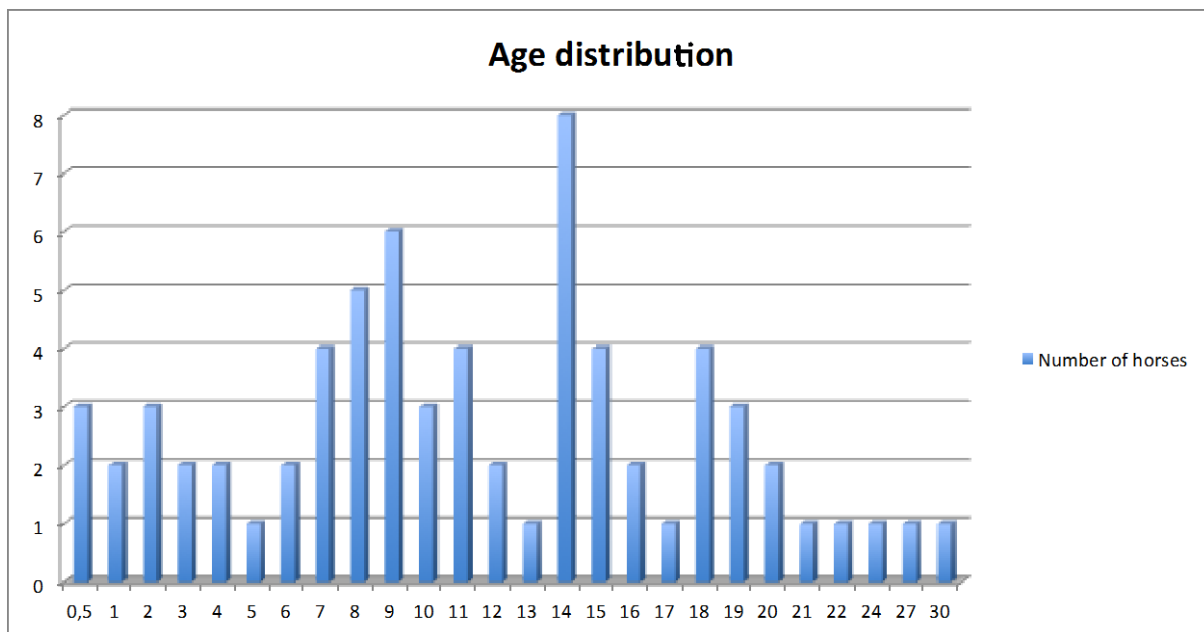


Figure 1. Distribution of age in the study

### Breed

Figure 2 shows the distribution of breeds in the case group. The distribution of breeds diagnosed with peritonitis was not reflective of the population treated at the clinic. The proportion of Icelandic horses was larger in the study group (16 %) vs. 9 % in the population group ( $p = 0.050$ ).

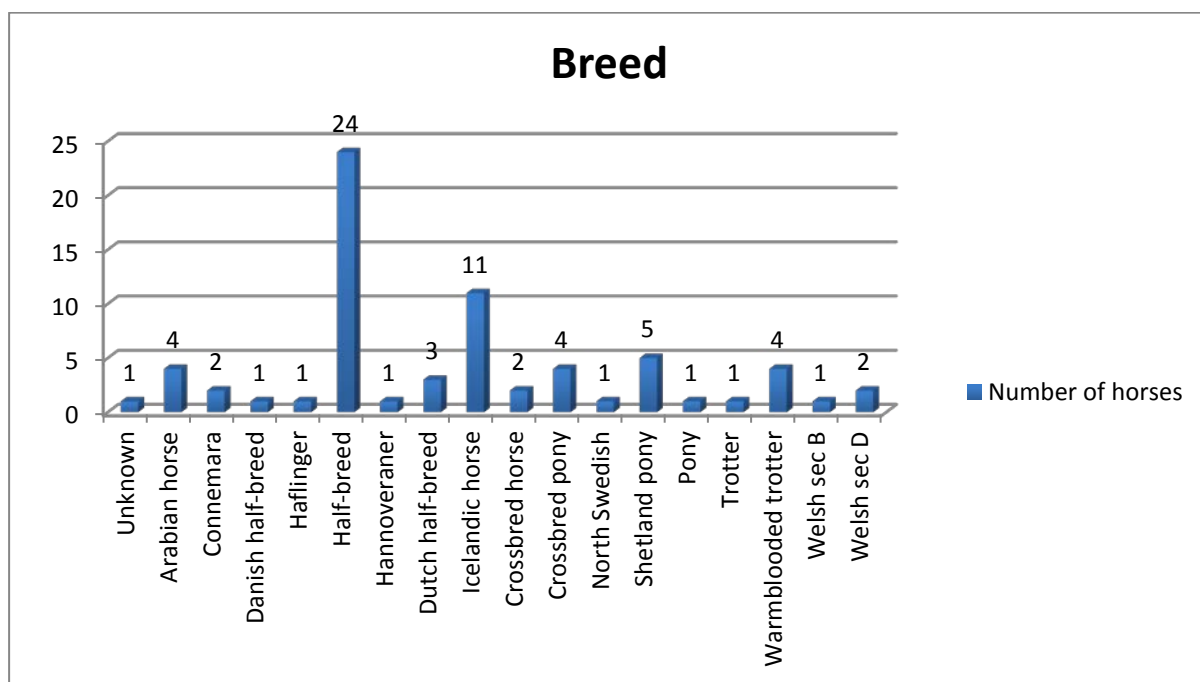


Figure 2. Distribution of breeds in the study

## History

The horses had been ill before arriving at the clinic between one day and two months. Most of them had only been clinically ill for one day (64%). Out of all 69 horses, 61 had been examined by a referring veterinarian who made an evaluation of the clinical status (88%). Many of these 61 horses had been treated by the referring veterinarian, either for a longer period of time or just before referring it to the clinic. There were treatments such as NSAIDs, metamizole, antibiotics, fluids and liquid paraffin via a nasogastric tube administered in the field. Out of the 69 horses coming to the clinic 44/69 cases (64%) had showed signs of depression, 48/69 cases (70%) had a history of colic, 43/69 cases (62%) had been anorexic and 53/69 cases (77%) had suffered from pyrexia ( $>38,2^{\circ}\text{C}$ ). Non-survivors presented with fever ( $p<0.0085$ ) and depression ( $p<0.0050$ ) more often than survivors. Survivors however, more often showed signs of colic in their history ( $p=0.0013$ ). No significant difference could be seen between non-survivors and survivors regarding anorexic behaviour.

Before coming to the clinic the horses had been ill for between one day (64%) and two months. 96% had been ill between one and eight days. The two cases having been ill between one and two months had had intermittent fever and colic symptoms, see figure 3.

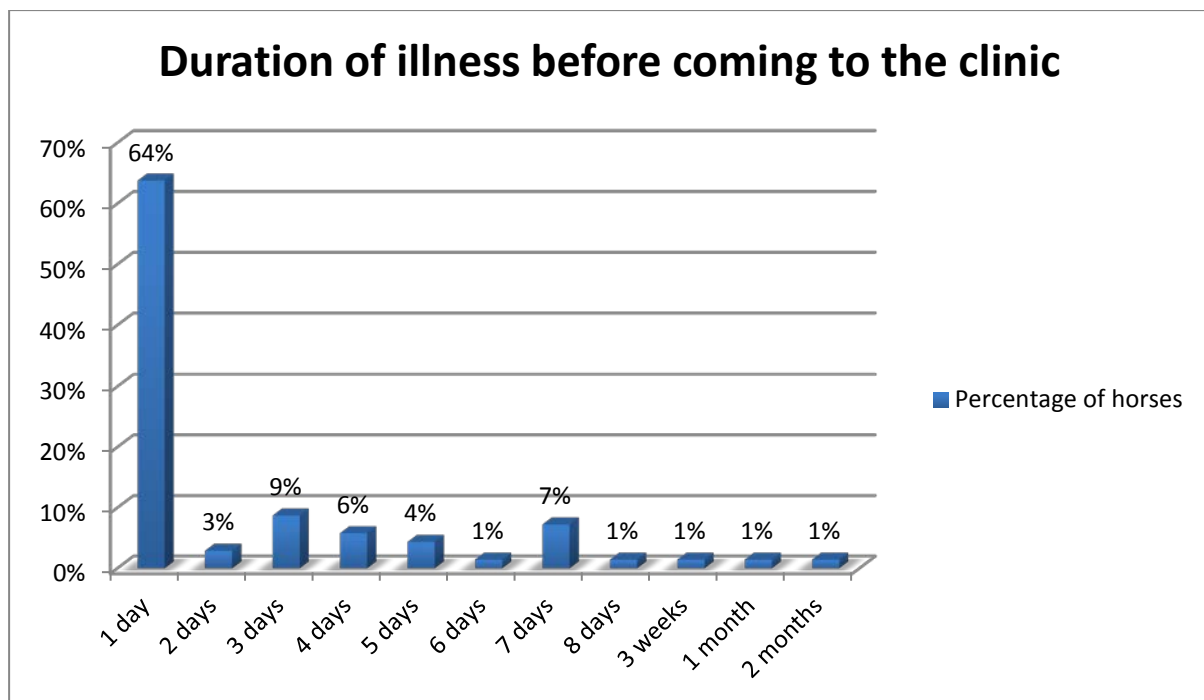


Figure 3. Distribution of duration of illness prior to arriving to UDS.

## NSAIDs in the history

Non-steroidal anti-inflammatory drugs, such as flunixin meglumine and meloxicam, were given to 21 horses (30%) prior to coming in to the clinic. Two of those horses (3%) had been treated for other issues such as suspected laminitis and lameness for a period of five days three weeks prior and eight days four weeks prior. The remaining 19 horses were given NSAIDs between 1-5 days to treat the symptoms they later came to UDS for. For four of the

horses, treatment with NSAIDs is unknown. Twenty horses were treated with metamizole prior to transport in to the clinic. Twenty-four horses were not treated with any form of NSAIDs or pain relieving substance.

**Physical examination**

Upon arrival 59/69 horses showed signs of depression compared to the 44 cases that had shown such signs before coming to the clinic. The sign of depression upon presentation was in the study significant for survival (p=0.0171). Pyrexia was seen in 42/68 horses examined. Abdominal discomfort was seen in 48/69 horses, the same number of horses with a history of colic, see table 1.

Table 1. Difference in number of horses with clinical signs before and after arrival at the clinic

**Variable**

2/68      62

Tachycardia (> 44/min) was present in 48/69 horses (73%), tachypnoea (> 20/min) in 28/67 horses (42%), abnormal appearance of the mucous membranes (not pink) in 42/69 cases (85%), pyrexia (> 38,2 °C) in 42/68 cases (62%), abnormal rectal examination (anything that wasn't regarded as normal such as impaction, swollen intestines, suspected volvulus etc.) in 33/62 horses (53%) and reflux during nasogastric tubing 9/50 horses (18%). Non-survivors presented with tachycardia (p<0.0001) and gastric reflux (p<0.0007) more often than survivors.

Borborygmia were recorded upon presentation in all cases except one, see figure 4.

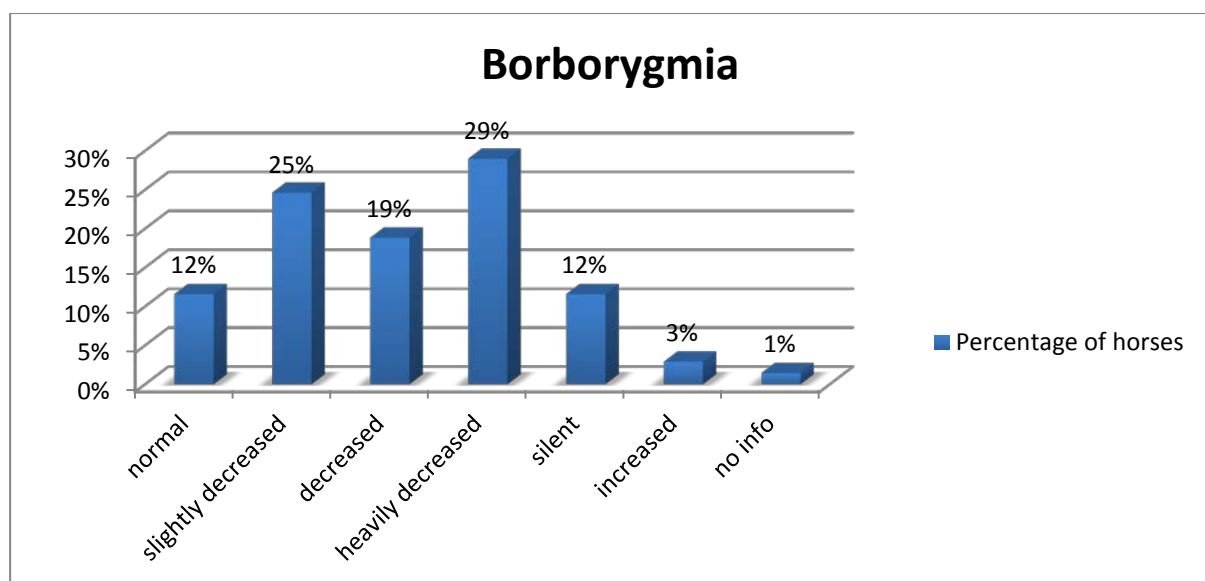


Figure 4. Abdominal sounds in the horses upon the first clinical examination



## Abdominocentesis

The results of visual inspection of the peritoneal fluid were recorded in all cases but one. The first evaluation regarding colour and turbidity is noted in figure 5. The peritoneal fluid was not considered normal in any of the horses evaluated. In one horse, the fluid described as yellow was noted as abnormal in the medical records of that horse and is therefore a category on its own even though it could have been a normal variation in colour. One horse did not have any information regarding visual inspection of the peritoneal fluid written in the medical record. Non-survivors presented with a more severe alteration of the abdominal fluid than survivors when visually examined ( $p < 0.0001$ ).

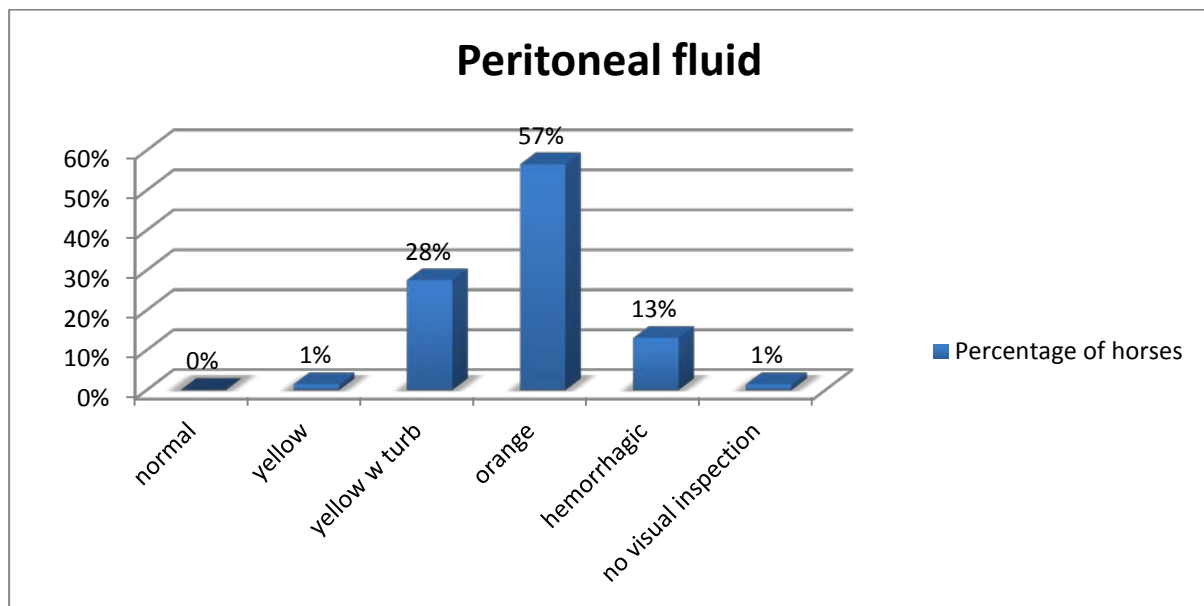


Figure 5. Results of the visual inspection of peritoneal fluid.

## Cytology

Leukocyte concentration was evaluated in 61/69 cases (88%). Values ranged from 13,200 – 486,000 cells/ $\mu$ L. Out of the 59 horses that had a value of  $>20,000$  cells/ $\mu$ L, eight were euthanized. Eight horses did not have a value regarding leukocytes recorded in their medical records. Two of these horses were euthanized.

Neutrophil percentage out of the leukocytes in the peritoneal fluid was noted in 52/69 cases (75%). Not all cases with leukocyte values recorded had noted the neutrophil percentage, and not all cases with neutrophil percentage measured had noted leukocyte concentration. Range of neutrophil percentage was 73-99%.

The peritoneal fluid was analysed for total protein concentration in 49/69 cases. Analysis was performed in the laboratory when analysing the peritoneal fluid for leukocytes and neutrophil concentration. Using the cut-off value of  $>30$ g/L, 42/49 horses (86%), had a value of  $>30$ g/L (range 31-67g/L). Out of these 42 horses, six were euthanized (14%). No horse with a total

protein value of <30g/L was euthanized. Out of the 20 horses lacking peritoneal protein values, four were euthanized (20%).

Manual cytological examination and gram-staining of the peritoneal fluid to detect bacteria was performed in 45/69 cases (63%). The cases that did not have a manual examination were tested with either a Lasercyte machine to determine the constituent cells of the fluid or not at all due to various factors such as not being able to run the Lasercyte or the machine not being available. Bacteria, both intracellular and extracellular, were found in 12/45 cases (27%).

*Microbial culture*

Of the 69 cases, 54 were sent for microbial culture. All of the samples taken were sent for both for anaerobe and aerobe culture. Of the 12 cases where bacteria were identified during cytology, bacteria grew in eight of them (67%), one sample was not sent for inoculation and in the two remaining cases no growth was seen. Out of the 33 cases where no bacteria could be seen in cytology, 29 were sent for inoculation (88%). Of these 29 samples, 20 were negative while nine cases showed bacterial growth (31%).

The peritoneal fluid samples with bacterial growth showed a wide diversity of bacteria, see figure 6 below. Thirty-two cases did not show any bacterial growth (46%). All of these 32 horses survived. Out of the three horses that showed bacterial growth and were euthanized, one had an infection with *β-Streptococcus*, one presented an infection with *E-coli* and one horse presented with a mixture of bacteria including *Bacteroides species*, *β-Streptococcus* and *Fusobacterium spp.* Fifteen cases were not analysed for bacterial growth. Seven of these horses were euthanized (47%). The lacking of bacterial analysis in some of the cases was due to the fact that they were euthanized shortly after arriving at the clinic, five out of seven horses within four days, and therefor no result could be obtained.

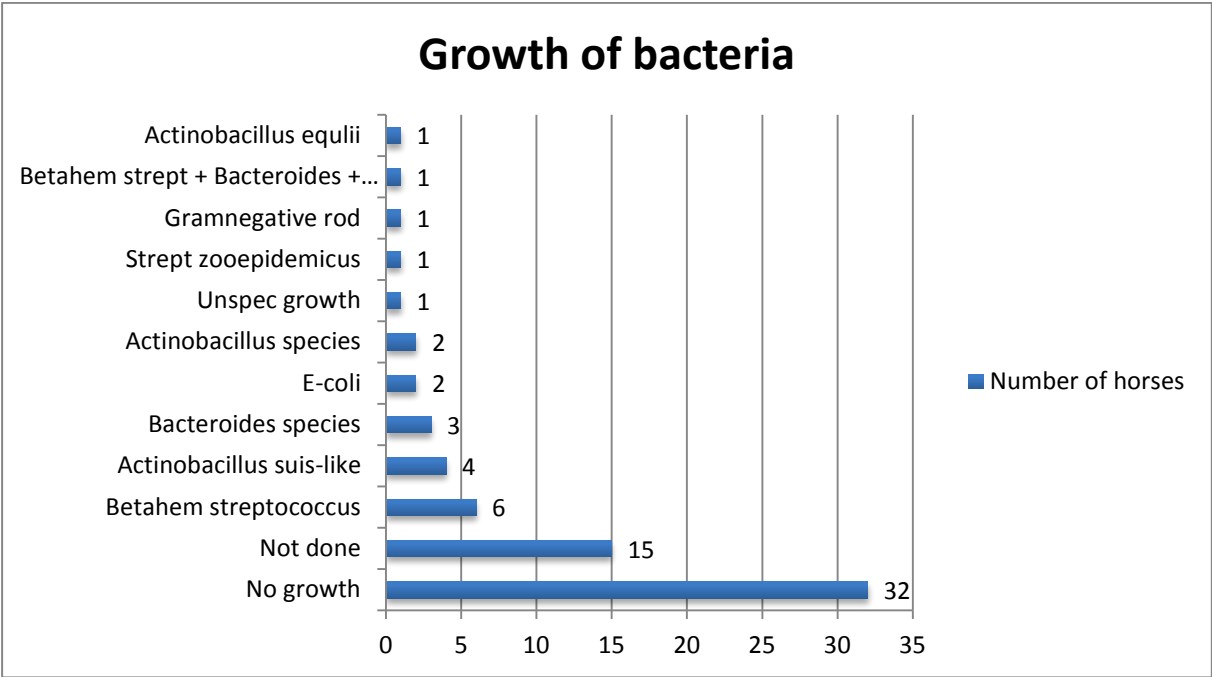


Figure 6. Distribution of bacterial cultivation in all 69 cases of peritonitis.

## Blood analysis

### Proteins

The acute phase protein serum amyloid-A (SAA) was tested in 44/69 cases (64%) to determine the level of inflammation in the horse. UDS has not been testing SAA for the entire ten-year period and therefore not all of the horses are expected to present with a value. A normal SAA value is <5 mg/L. In the study, a first test of SAA was noted (44/69) as well as a last test before leaving the clinic (39/69). All of the horses presented with an elevated SAA value (277 - >1250 mg/L), see figure 7 below. Out of the ten horses that were euthanized, only four were tested for SAA concentration. These four ranged between 340 - >1250 mg/L.

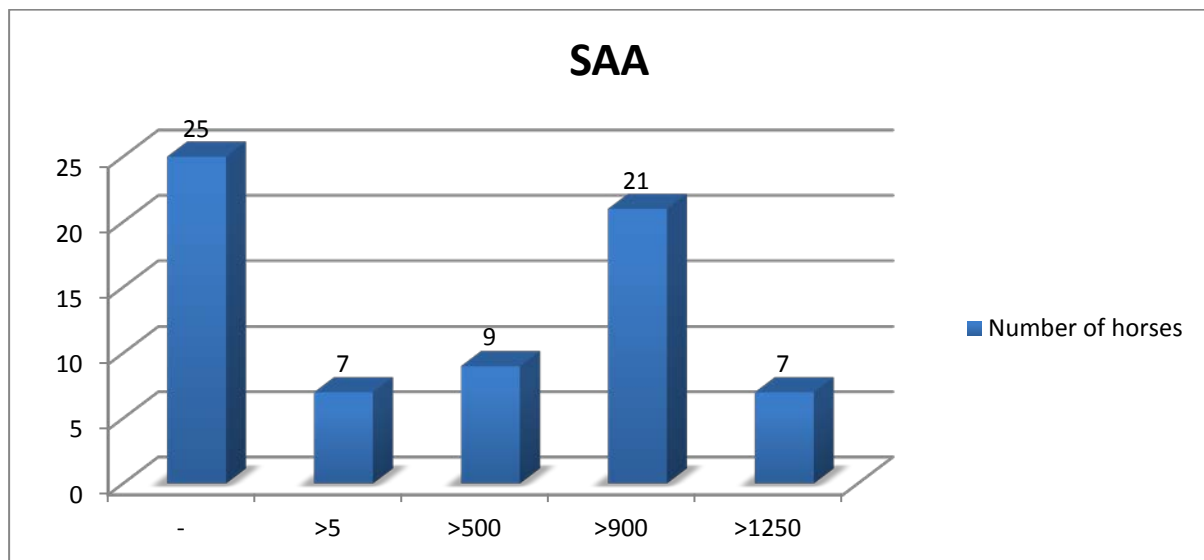


Figure 7. Distribution and range of serum amyloid-A in the 69 cases (- = not tested).

A final SAA test was performed in 39/69 cases (57%) before either being discharged from the clinic or euthanized. 6/39 (15%) had a normal SAA of <5 mg/L, the remaining 33 horses had a value between 5,3 - >1250 mg/L. Out of the ten horses euthanized, two had a value recorded for their final SAA. One of these horses went down from >1250 to 20 mg/L and one went down from >900 to 293 mg/L. The first horse mentioned above was euthanized due to development of laminitis after 16 days in the clinic. The other horse stayed for a total of 12 days and got better at first but then deteriorated and it was decided that it should be euthanized.

In general, the SAA decreased during the horses stay at the clinic but there were three horses that had the same values both times, these horses were tested at home to see that the value decreased before terminating antimicrobial treatment. One horses value increased at the clinic, from 277 to 599 mg/L, most likely due to the first test being analysed in the early stages of the illness. SAA was used to evaluate the response to treatment in the horse and to determine when treatment could be terminated.

Serum fibrinogen was also tested as a marker of inflammation. Values should be in the range of 1,8-4,2 mg/L. 54 out of 69 horses were tested (78%) and ranged from 2-10,6 mg/L. 41 horses had a value of >4,2 mg/L (76%) and out of these 41 horses, four were euthanized. Three horses with normal values were euthanized and the remaining three were not tested for levels of fibrinogen.

Total protein and albumin concentrations were also investigated in this study. Normal total protein ranges from 50-66 g/L and 34/69 horses (49%) were tested. Out of the 34 horses, 25 were in the normal range (74%). Five horses had <50 g/L and four horses had >66 g/L. Albumin has a normal range of 32-39 g/L. 31/69 horses (45%) were tested and hypoalbuminemia could be seen in 16/31 horses (52%). The rest were within normal range, no horse had hyperalbuminemia.

### *Haematology*

The white blood cell count (WBC) was recorded and neutrophil levels and their morphology were selected as important factors in this study. The samples analysed were from the first complete blood count (CBC) taken at the clinic. Out of the 69 horses, 59 (86%) had values or notes written in their medical record regarding the neutrophils and their morphology, see table 2.

*Table 2. Concentration and morphology of neutrophils in the 69 cases upon presentation*

<b>Neutrophils</b>	<b>N (%)</b>	<b>Euthanized (n)</b>	<b>Euthanized (%)</b>
No value	10/69 (15%)	2	20
Normal	20/69 (29%)	5	50
Neutropenia	18/69 (28%)	2	20
Neutrophilia	7/69 (10%)	0	0
Left shift	13/69 (17%)	1	10
Toxic	1/69 (1%)	0	0
<b>Total</b>	<b>69 (100%)</b>	<b>10</b>	<b>100%</b>

The 13 cases noted only with left shift or toxic neutrophils were normal regarding the amount of neutrophils, they only presented with a change in morphology. Four of the 19 cases with neutropenia also showed toxic changes or left shift. Three of the cases with neutrophilia showed signs of toxic changes to the neutrophils and left shift. Out of the ten horses euthanized, five (50%) had normal neutrophil levels. Non-survivors were more prone to display normal neutrophil morphology (p=0.0004) than survivors in this study.

### **Treatments**

#### *Supportive treatments*

All horses but one were treated with flunixin meglumine during their stay in the clinic. Flunixin meglumine was used as pain relief, anti-pyretic treatment and to prevent or aid in cases of suspected endotoxemia.

62 horses (90%) were given intravenous (IV) fluid therapy for between one and ten days. To some of the fluids there were additives, such as potassium chloride (KCl) and glucose, added depending on the clinical parameters of the horse. Water was given via nasogastric tube to one horse. Six horses were not given any fluids at all.

### *Antimicrobial treatment*

Antibiotics were administered in all cases, whether microbial culture or not, see table 3. In the clinic only Benzylpenicillin and Gentamicin are used as the treatment of choice, usually as a combination but sometimes Benzylpenicillin is used solely.

Duration of antimicrobial treatment in the clinic ranged between one day (the horses that were euthanized after only one day) and 23 days.

Duration of treatment at home was not possible to determine since most of the patients that were discharged with a continuous regimen of antibiotics were encouraged to take follow-up tests of SAA and abdominocentesis with the aid of their local veterinarian before subsiding treatment. Treatment was ended when those parameters were close to normal or normal.

*Table 3. Antimicrobial treatments used at the clinic to treat the patients and out patient treatment after leaving the clinic*

<b>Antimicrobial treatment</b>	<b>Number of horses n</b>	<b>Euthanized n=10</b>
<b>Benzylpenicillin</b>	<b>8</b>	
<b>Benzylpenicillin + Gentamicin</b>	<b>52</b>	<b>9</b>
<b>Benzylpenicillin + Trimetoprim-Sulfadiazine*</b>	<b>3</b>	
<b>Benzylpenicillin + Gentamicin + Trimetoprim-Sulfadiazine*</b>	<b>2</b>	
<b>Benzylpenicillin + Metronidazole*</b>	<b>2</b>	
<b>Benzylpenicillin + Polymyxin B</b>	<b>1</b>	
<b>Gentamicin</b>	<b>1</b>	<b>1</b>

\* = out patient treatment

### *Other treatments*

Other treatments during the horses stay in the clinic included gastric ulcer medication (omeprazole), analgesics (butorphanol, metamizole, lidocaine), polymyxin B, treatment for impaction (mineral oil via nasogastric tube with water or with feed, Glaubers salt) electrolyte solutions (potassium or potassium-chloride in IV-fluids, plasma), treatment for hyperlipemia (insulin, glucose) and topical anti-thrombotic ointment (heparin and hydroxyethyl salicylate gel, ketoprofen gel). Treatments after discharge from the clinic included anthelmintics (fenbendazole) and gastric ulcer medication (sucralfate) as well as continuation of some of the other treatments (heparin and hydroxyethyl salicylate gel, mineral oil).

### **Complications**

Complications that occurred during the hospitalisation included jugular vein thrombosis/thrombophlebitis, laminitis, suspected abdominal ulcers and intra-abdominal adhesions.

### **Length of hospitalisation**

Length of hospitalisation ranged between 1-25 days for all horses. Average length for survivors was 12,1 days, median length was 11,5 days and the range was 4-25 days. One horse was seen on outpatient basis and that was the horse that was hospitalised for four days. The duration of treatment for that horse is unknown. For non-survivors the range was 1-18 days, average 5,7 days and median length three days. The one horse hospitalised for 18 days developed laminitis during its stay and was euthanized due to intractable pain and old age.

### **Post-mortem findings**

Results of a post-mortem were available in 3/10 non-survivors (30%). Findings included focal, suppurative peritonitis with intramural abscess and pseudo-obstruction of the colon in one horse, fibrinous peritonitis with multi-focal pyogranulomatous inflammation and multiple renal infarctions in another and incarcerated small intestine with acute peritonitis, mesenteric abscesses, thrombotic arteritis, renal infarction and severe Ascariasis in the small intestine.

### **Survival rates**

Out of the 69 horses included in the study, 59 were discharged from the clinic (86%). Out of the ten horses that were euthanized, six horses were euthanized due to lack of response to treatment. Two other horses were euthanized in relation to the development of a large intestinal volvulus and two cases were euthanized due to financial restraints and development of laminitis respectively.

When separating the primary peritonitis from the secondary, it shows that 3/10 horses euthanized (30%), were in the primary group. Out of these three horses, only one was euthanized due to lack of response to treatment. The others were euthanized due to laminitis and old age and the other due to financial restraints. When comparing the survival rates between the primary and secondary peritonitis groups with Fisher's exact test, there is no significant difference between the two groups ( $p=1.000$ ).

One horse that was discharged from the clinic was euthanized one week later due to severe deterioration of condition. It was later revealed that it had a perforated jejunum.

## **DISCUSSION**

Out of the 69 horses included in the study, 59 survived (86%). It has been reported to be between 43-86% in other studies (Dyson, 1983; *Hawkins et al.*, 1993; *Henderson et al.*, 2008; *Nógrádi et al.*, 2011). The differences in these reported results are most likely due to the different inclusion and exclusion criteria (such as gastrointestinal ruptures, recent abdominal surgery etc.) included in the different studies. This study is similar to the one done by

Henderson *et al.*, (2008) in which the horses with suspected rupture of the gastrointestinal tract were excluded as well as any horse with recent abdominal surgery or recent castration. The survival rates being exactly the same at 86% is indicative of the short-term survival of horses with non-traumatic peritonitis.

Other studies and articles on the subject of peritonitis in horses (Mair *et al.*, 1990; Hillyer & Wright, 1997; Henderson *et al.*, 2008) have chosen cut off values above WBC 5000 cells/ $\mu$ L in abdominal fluid and protein >25g/L. To be certain that the horses in this study had severe forms of peritonitis, the cut-off values were set at higher intervals (WBC >20.000 or protein >30g/L). Selecting relatively higher cut-off values could infer a risk of achieving a higher mortality rate due to the fact that the horses with only mild forms of peritonitis were excluded. This does not seem to be the case in this study since the survival rate was very good (86%).

Eight variables (fever, depression in the history, increased heart rate, gastric reflux, visually altered peritoneal fluid, normal neutrophil morphology, colic in the history and depression upon presentation) that were of significant difference between survivors and non-survivors were discovered when using the Fischer's exact test. Non-survivors presented with fever ( $p < 0.0085$ ) and depression in the history ( $p < 0.0050$ ) more often than survivors, they had higher heart rates ( $p < 0.0001$ ), more often gastric reflux ( $p < 0.0007$ ), presented with a higher degree of altered abdominocentesis results when visually examined ( $p < 0.0001$ ) and more often presented with normal neutrophil morphology ( $p = 0.0004$ ) when analysing the complete blood count. There were two variables positively associated with survival. Survivors were more prone to display colic before arriving at the clinic ( $p = 0.0013$ ) and depression upon presentation ( $p = 0.0171$ ). Except for tachycardia being significant in both this study and in the one published by Hawkins *et al.* (1993), these differences were not seen in other studies using the same model (Hawkins *et al.*, 1993; N6grádi *et al.*, 2011). This is only an initial screening of possible prognostic indicators, and a multivariate statistical analysis should be applied in order to avoid confounding errors bound to repeated testing for significance.

It is often impossible to determine the underlying cause of peritonitis (Hawkins *et al.*, 1993). Primary peritonitis doesn't seem to have an underlying cause, however, it is still a disease that affects horses, sometimes with fatal outcome. The reasons for developing primary peritonitis might be an underlying disease that we cannot identify or it may be due to the horses general condition, meaning that if the horse is slightly weaker for period of time, peritonitis may develop. Perhaps it can be caused by a change in feed, feed of inferior quality or by stress (including new feed, new environment, other horses, other routines etc.) rendering the host defences impaired. These are factors that are difficult to determine since there is no way of knowing exactly how long it takes for a horse to react in a negative way to a stress factor (by initiating an inflammation in their abdomen) or if a horse will react at all.

One aim of this study was to investigate if primary peritonitis was different in terms of short-term survival compared to the second group, which was thought to have an underlying cause of the peritonitis. However, even though the secondary group had suspected aetiologies, it was not possible to determine whether the abnormal findings initiated the peritonitis, if they

arose because of the peritonitis or if they were irrelevant to the illness. There was no difference in mortality between the groups of horses that suffered from primary and secondary peritonitis; they both had a mortality of about 15% ( $p=1.000$ ). This means that the separation of the two groups is unnecessary to determine the outcome of the patient in the clinical day-to-day work. However, this only applies to this study, the distinction between the two groups might be of greater importance in other studies.

A skewed horse population around the UDS clinic could cause a reason for an apparent high occurrence of horses of a specific breed, gender or age. Such demographic factors might have an influence on the occurrence of certain diseases. This is not easy to control, and therefore we had to use the entire hospital population as a reference population.

When analysing age, breed and gender distributions of the entire hospital population and comparing the data with the patient distribution in the study, there were some signs of aberration from the normal distribution. The age distribution did not follow the normal distribution curve found at UDS. There were fewer horses between the ages of 10-13 in the study compared to the overall population coming to the clinic. This is most likely due to the small sample in this study compared to the much larger number of horses treated at UDS (4000-6000 horses/year) and the difference in the age distribution curve is therefore not significant. This was not tested using Fisher's exact test.

Breed distribution showed signs of a higher probability to develop peritonitis in Icelandic horses (15% vs. 9%) and a lower probability for Warm-blooded trotters (8% vs. 15%) and Half-breeds (34% vs. 40%) to develop peritonitis. However, only the increased risk of Icelandic horses was statistically significant, and this result should be retested in a more advanced statistical model, which simultaneously investigates the influence of age, breed, gender, and other factors, such as different clinicopathological findings. The findings regarding these different breeds might be due to Icelandic horses being more sensitive to develop peritonitis and trotters and Half-breeds being more resistant. Usually Icelandic horses are viewed as a bit more robust of a breed so it is difficult to find an explanation for these findings. However, out of the horses euthanized, only one was an Icelandic horse (1/11) and five (50%) were Half-breeds (5/24) so the Icelandic horses seem to be more resilient with treatment.

Gender almost followed the normal distribution at the clinic. Therefore we did not analyse this any further.

The most common clinical signs upon presentation were pyrexia (77%) and signs of colic (70%). More than 60% also presented with signs of depression and anorexia.

Out of the 48 horses with signs of colic in their history, five were euthanized (10%) and out of the remaining 21 non-colic horses, five were euthanized (24%). There seems to be a positive correlation between colic in the history of the horses and survival ( $p=0.0013$ ) in this study. *Nota bene*, the significance of this should be evaluated in the light of this being the subjective information regarding the horse's history from owners and caretakers. Their appreciation of colic might differ from that of a veterinarian evaluating the horse at the clinic. There was no



significant difference between survivors and non-survivors when comparing colic upon presentation at the clinic ( $p=0.1366$ ).

Signs of colic, both in the history and upon presentation, were seen in 70% of the horses in this study compared to 50% (Henderson *et al.*, 2008), 63% (Dyson 1983) and 31% (Nógrádi *et al.*, 2011) respectively. Mair *et al.*, (1990) found that 81% of the cases in their study presented with signs of colic, both chronic, low-grade pain and recurrent bouts of acute pain.

In this study, pyrexia was noted in 62% of the cases upon arrival at the clinic. In their history, 77% of the horses had suffered from pyrexia. Pyrexia was not a consistent finding in a study published in 2011 by Nógrádi *et al.* It was also only seen in 28% of the horses evaluated in a study written by Henderson *et al.* (2008). However, they had chosen a cut-off value of  $>39^{\circ}\text{C}$  (Nógrádi *et al.* 2011) and  $>38,5^{\circ}\text{C}$  (Henderson *et al.*, 2008) respectively. Having chosen the same cut-off value of  $>38,5^{\circ}\text{C}$  Mair *et al.* (1990) found that 71% of their cases presented with pyrexia.

The improvement that can be seen regarding pyrexia upon arrival compared to the history (table 1) can most likely be assigned to the administration of NSAIDs or metamizole, which have an antipyretic effect, given before transport to the clinic. Forty-one horses (59%) were treated with NSAIDs, metamizole or both before transport, four of the horses may have been treated but the medicine administered in those cases is unknown. Many of the horses had higher temperatures after the initial evaluation at the clinic; these temperatures were not recorded in this study.

The NSAIDs might have masked other clinical signs such as abdominal discomfort or any other discomfort but this is not possible to determine in this study.

No evidence could be found that supported the theory of the possibility of NSAIDs causing peritonitis. Only two horses had been treated for a period of time for other conditions before coming to the clinic to be diagnosed with peritonitis. The other 19 horses treated with NSAIDs were treated for the symptoms for which they later came into the clinic and therefore are not suspected of causing the peritonitis.

Nógrádi *et al.* (2011) found that most horses had abnormal mucous membranes (82%) and in our study, we could confirm the same phenomenon where abnormal mucous membranes were present in 85% of the cases. Henderson *et al.*, (2008) could however only present 3/65 cases (5%) with abnormal mucous membranes. There was no difference between survivors and non-survivors regarding abnormal mucous membranes.

Depression upon presentation as a sign of peritonitis was seen in 86% of the horses in our study. It was only seen in 49% (Henderson *et al.*, 2008) and in 17% of the cases (Nógrádi *et al.*, 2011) respectively. Perhaps this is because the owners of the horses in our study have tried to treat the illness out of practice as a first attempt and when that fails the horses come to the clinic in a worse condition than in the other studies. It is possible that the transport made them more depressed, but it is also possible that the deterioration was due to a subjective

opinion of the receiving veterinarian or the fact that it wasn't mentioned or recorded as a part of the history of the horse.

Gastric reflux was in this study correlated with a higher mortality ( $p < 0.0007$ ). This is however not a common clinical sign in horses with peritonitis (Rose & Hodgson, 1993; Hillyer & Wright, 1999; Davis 2003; Reed *et al.*, 2010). The gastric reflux may be associated with other severe conditions that might lead to a less favourable outcome for the horse. However, it is important for any clinician to know that a horse presenting with gastric reflux and peritonitis might have a less favourable prognosis.

Many of these clinical signs can be displayed in relation to other illnesses so it is therefore imperative to perform paracentesis and analyse the abdominal fluid to be able to diagnose peritonitis.

Bacterial cultures were done in 54/69 cases (78%) in this study. The most common finding was no growth in 32/54 cases (60%). The most common bacterial finding in the peritoneal fluid in this study was *β-Streptococcus*, 6/54 (11%) followed by *Actinobacillus suis-like* in 4 cases of 54 (8%). These findings are not consistent with what is seen in other studies. Both Nógrádi *et al.* (2011) and Hawkins *et al.* (1993) found that *E-coli* were the most commonly isolated bacteria. Other studies show that *Streptococcus zooepidemicus* and *Bacteriodes fragilis* are the most commonly cultured bacteria (Mair *et al.*, 1990). These differences are most likely because of the different inclusion criteria where the authors above have included horses with gastrointestinal rupture and abdominal surgery in their history. There was no significant difference between survivors and non-survivors regarding positive bacterial results, both groups had 30% positive cultures. In this study, there is a mix between anaerobic bacteria and facultative anaerobic and aerobic bacteria. These are bacteria that can be found in the gastrointestinal tract but also can be found in relation to other illnesses such as strangles, pyogranulomas in internal organs and as viral infectious complications (Equine influenza etc.).

When analysing the clinical signs and the laboratory findings in the group of horses deemed primary (21/69), nothing in their history, clinical signs or clinicopathological findings could separate them from the other horses upon presentation except for the fact that they had a normal rectal exam and no gastric reflux. They presented with much the same findings as the rest of the horses in the study. They seemed more prone to show signs of colic (81% vs. 70% in the whole study) and pyrexia (70% vs. 62%) but this was not statistically significant. No difference could be detected when determining signs of depression (86%).

The finding of intestinal parasites was in this study a parameter used to divide the horses into the two groups. It is important to note that when analysing faecal samples for intestinal parasites, a negative result might be a false negative and a positive result might not be of significance to the peritonitis. The significance of this was not possible to determine in this study.

Normal neutrophil morphology was in this study associated with increased mortality ( $p = 0.0004$ ) since five horses presented with normal neutrophils and were euthanized. This

might be due to the fact that the horses presenting with normal neutrophils have not yet responded to the illness because it is early in the process, they might also suffer from the per-acute form of peritonitis and therefore haven't been able to mobilise a significant defence upon presentation. One of these five horses was euthanized due to financial restraints.

SAA has never been evaluated in other published articles about peritonitis. In this study it appeared not to be a significant tool for determining the outcome of the patients since only four of the horses euthanized were tested and they ranged, much as the survivors, from >1250-340 mg/L in their first test. However, UDS has not been using SAA as a marker for inflammation for the entire ten-year period and therefore the results might be inconclusive in this study. A second SAA test was performed in two of the horses euthanized and they showed great signs of improvement (from >1250 to 20 mg/L and from >900 to 293 mg/L). Follow-up tests such as SAA and abdominocentesis for cytological examination were recommended in most cases to determine when antimicrobial treatment could be terminated. This was usually performed out of the clinic, which made it difficult to determine the length of treatment with antibiotics at home and the final outcome of the patients.

In the literature there are other methods of treatment discussed and performed besides the ones used at UDS and presented in this study (intravenous fluids, NSAIDs, antimicrobial treatment and treatment for any underlying condition that might be related to the peritonitis). These treatments include abdominal lavage, which seems to be a method of choice for some clinicians but not proven to be a successful treatment in clinical trials (Reed *et al.*, 2010). Even though lavage doesn't seem to have great effect, there could be of some use in the clinic to drain more of the abdominal fluid to discard of bacteria and inflammatory cells more efficiently.

The use of lidocaine infusion could perhaps be a treatment that could be introduced at the clinic more often when faced with endotoxemia and severe pain. In clinical trials the horses with endotoxemia seem to have a favourable outcome when treated with lidocaine CRI (Reed *et al.*, 2010).

## **CONCLUSIONS**

Survival rate for horses diagnosed with primary peritonitis is 86% at Universitetsdjursjukhuset (UDS) in Uppsala and primary peritonitis is seen in one out of three patients diagnosed with non-traumatic peritonitis here. No difference could be seen in terms of short-term survival between the groups of primary and secondary peritonitis in this study.

There were eight clinical parameters (fever, depression in the history, increased heart rate, gastric reflux, visually altered peritoneal fluid, normal neutrophil morphology, colic in the history and depression upon presentation) statistically significant in this study to give an indication regarding the outcome of the patients. These can now be monitored more closely when encountering new patients presenting with the suspicion of peritonitis.

There was no evidence of peritonitis being caused by NSAIDs in the study.

## **ACKNOWLEDGMENTS**

A special thanks to my supervisor and assistant supervisors for always taking the time to answer any and all questions. A special thanks to Mikael Eklund in the Veterinary Library for your infinite knowledge in the world of digging up old articles at record speed.

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