Acta vet. scand. 1996, *37*, 203-211.

Acute Post-Treatment Enterocolitis in 13 Horses Treated in a Norwegian Surgical Ward

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Larsen, J., N.I. Dolvik, and J. Teige jr.: Acute post-treatment enterocolitis in 13 horses treated in a Norwegian surgical ward. Acta vet. scand. 1996, 37, 203-211 – Case histories, results of laboratory analyses, treatment regimens and outcome are described for 13 horses that developed acute enterocolitis after various surgical treatments. Twelve of the 13 horses had been cast in lateral recumbency, and 10 of those 12 had received general anesthesia. Eleven horses had been treated with antimicrobial drugs. Most horses (11) developed diarrhea 2 days after initiation of treatment, with a variation from 1 to 5 days. The most apparent symptoms were fever, up to 40.5°C, elevated heart rate, discoloration of mucous membranes, and diarrhea. Dehydration, acidosis and leukopenia were marked at the time clinical signs occurred. Packed cell volume and total serum protein values were elevated. Therapy was directed toward replacement of fluid losses, restoration of acid-base balance, and counteraction of inflammation. Seven horses (54%) died or were euthanized. At necropsy, pathological changes showed large variations between individuals. *Salmonella* spp. were not isolated from any of the horses, and no common causative agent was identified. Preventive efforts to avoid post-treatment enterocolitis have been taken with regard to preoperative routines, premedication and anesthesia, with apparent success.

colitis; diarrhea; endotoxemia; acidosis; sodium bicarbonate; postoperative complications.

Introduction

Acute colitis occurs sporadically in the overall horse population *(Palmer* 1992). *Jaksch* (1982) reported an incidence rate of approximately 1% of all types of gastroenteritis among his patients. However, the incidence of colitis can be high on certain premises for given periods of time, especially in stressful environments like racing stables or equine hospitals *(Rooney et al.* 1963, *Wierup* 1977). The rapid onset and high mortality (up to 80%) make this disease a devastating clinical and economic problem *(Kraft* 1985, *Staempfli et al.* 1991, *Verter & Wedell* 1991, *Palmer* 1992). Currently, several different infectious agents are known or suspected to be capable of causing acute colitis in adult horses *(Palmer* 1992, *Radostits et al.* 1994): *Salmonella* spp., *Ehrlichia risticii, Clostridium* spp., *Aspergillus* spp., *Mucor* spp., and nematodes of the subfamily *Cyathostominae.* Additionally, colitis can be associated with the use of certain antibiotics, certain toxins, and overdosage of non-steroidal anti-inflammatory drugs *(Palmer* 1992, *Radostits et al.* 1994). Colitis caused by *Ehrlichia risticii* infection (Potomac horse fever) and some cases of *Salmonella* enterocolitis seem to occur without apparent stress *(Whitlock* 1986, *Palmer* 1992), but many

reports of acute colitis in adult horses, from different parts of the world *(Graham et al.* 1920, *Rooney et al.* 1963, *Cook* 1973, *Baker & Leyland* 1973, *Owen* 1975, *Wierup* 1977, *Nielsen & Vibe-Petersen* 1979, *Jaksch* 1982, *Kraft* 1985), emphasize the importance of stress factors, such as transportation, racing, abrupt changes in feed, preexisting disease states, antibiotic treatment, or surgery, for the development of the disease. In a large percentage, perhaps as high as 50% of clinical cases, it has not been possible to identify any causative agent *(Whitlock* 1986, *Palmer* 1992).

This paper describes the seasonal occurrence, clinical signs, clinicopathological and postmortem findings, and treatment of secondary acute colitis in horses developed after treatment for other unrelated diseases. The relationship to previously described forms of acute colitis is discussed, and our efforts to prevent the disease are presented.

Materials and methods

Animals studied

During the years 1989-92, 13 horses treated in our surgical unit developed acute colitis 2-5 days after the initiation of treatment. With one exception, all cases occurred in winter: From November 1989 through February 1990, 5 of a total of 43 anesthetized horses developed diarrhea. From the middle of December 1990 to early February 1991, 5 more cases occurred with an additional case on the 6th of April; a total of 50 horses were anesthetized between December 10, 1990 and April 10, 1991. The remaining 3 cases occurred in December 1991 and January 1992, when a total of 19 horses were anesthetized. The total number of anesthetized horses in 1991 and 1992 were 140 and 153, respectively.

Of the 13 affected horses, 8 were Standardbreds, 3 Cold-blooded trotters, 1 Thoroughbred, and 1 Icelandic pony. Five were geldings, 4 were stallions and 4 were mares. Ages ranged from 1 to 13 years. Age distribution and outcome are presented in Fig. 1.

Case histories

All of the affected horses had been sedated with detomidine[a] intravenously at dosages of 5 to 10 μg/kg. Ten of them were cast with guaiphenesin and sodium thiopental intravenously prior to general anesthesia with nitrous oxide and halothane. Three horses did not go through inhalation anesthesia: The first of these horses received sodium thiopental only, the second was cast with guaiphenesin without general anesthesia, and the third one was treated standing.

Eleven horses, 8 of which had been anesthetized, received antimicrobial treatment: 8 horses received a combination of procaine penicillin and streptomycin i.m., and 3 received potentiated sulphonamides, i.v. and orally. Four of the horses treated with antimicrobials had also received a combination of ramifenazone and phenylbutazone[b] intravenously, at a dose rate of approximately 11 mg ramifenazone and 6 mg phenylbutazone per kg per day on one or two occasions.

The horses were treated primarily for the following problems: 4 of them had jaw fractures or cheek teeth removed. Three horses had infectious arthritis due to intra-articular injections, and one had infectious tendovaginitis due to a punctured wound. Two horses went through osteochondrosis operations. One horse had sinusitis and was trephined, and one had a bone sequestrum on the lower limb. The last horse was castrated and the spermatic cords ligated under general anesthesia.

[a] Domosedan®, Orion Corp. Farmos, Turku, Finland.

[b] Tomanol®, Essex Pharma GmbH, Konstanz, Germany.

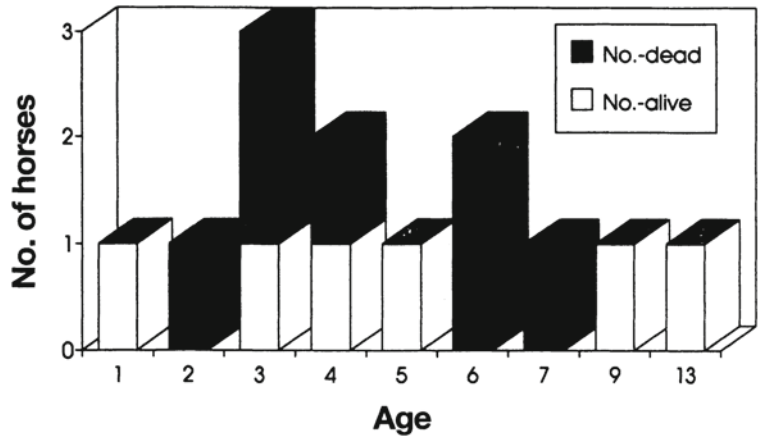

Figure 1. Age distribution and clinical outcome of 13 horses with acute post-treatment enterocolitis.

Laboratory analysis

Blood samples were drawn from the external jugular vein into sterile plain and EDTA-containing vacutainer tubes for biochemical and hematological evaluation. Arterial blood was taken from the transverse facial artery for acid-base and blood gas analysis. From 3 horses, blood samples were drawn into specially prepared pyrogen-free heparinized vacutainer tubes for endotoxin analysis. Basically, samples were taken before therapy was instituted.

Hematology was evaluated with the Technicon H*1 automatic hematology system, and a Technicon RA-1000 was used for biochemical analysis. Serum sodium was measured with an ion-selective electrode on the AVL 982 Electrolyte analyzer. Plasma potassium together with the acid-base and blood gas variables were analyzed on a Radiometer ABL4 acid-base laboratory. Plasma L-lactate was measured enzymatically.[c] Plasma endotoxin was measured using a Limulus amoebocyte lysate assay.

[c] Lactate fully enzymatic, Test-combination, Boehringer Mannheim, Germany.

Necropsy procedures

Seven horses were submitted for necropsy. Four of these had been euthanized, and 3 had died. They all went through a routine necropsy procedure. Sections for microscopical examination were stained with hematoxylin and eosin. Selected sections were also stained using the Martius scarlet blue (MSB) method.

Bacteriology

Feces (and, after necropsy, intestinal contents) was cultured on blood agar plates aerobically and anaerobically. Selenite broth was used as enrichment medium.

Results

Clinical signs

Most horses (11) developed diarrhea 2 days after initiation of treatment, with a variation of 1 to 5 days. A short period of fever preceded the diarrhea with temperatures up to 40.5°C. Mild to moderate colic was common. In most cases, diarrhea was profuse and foul-smelling and often tested positive for occult blood. The heart rate was elevated up to 90/minute, the pulse was

Table 1. Affected variables in blood, serum and plasma.

Variable	Range	Reference range	Unit
PCV	0.30 - 0.70	0.28 - 0.47	l/l
Total protein	63 - 86	56 - 74	g/l
WBC	1.8 - 10.9	5.7 - 11.2	G/l
Neutrophils	0.5 - 8.9	2.7 - 7.5	G/l
Lymphocytes	0.5 - 2.9	1.6 - 4.3	G/l
PaO2	97.8 - 112.2	98.8 - 132.1	mmHg
PaCO2	30.0 - 39.6	36.7 - 50.7	mmHg
Arterial blood pH	7.161 - 7.380	7.316 - 7.419	
SBE	−18.1 - −1.5	−3.0 - +4.2	mEq/l
Na	126 - 139	139 - 147	mmol/l
K	2.9 - 5.1	3.7 - 4.5	mmol/l
Lactate	1.3 - 6.7	0.4 - 1.3	mmol/l
AST	120 - 1270	120 - 380	SF-units
Total bilirubin	28.0 - 112.4	16.8 - 62.2	μmol/l
Fibrinogen	1 - 5	< 3	g/l

PCV = packed cell volume; WBC = white blood cells; SBE = standard base excess;
AST = aspartate amino-transferase.
Reference values are based on data from 46 clinically healthy horses examined at the Norwegian College of Veterinary Medicine *(Larsen* 1991). The same laboratory methods and equipment were utilized when the reference ranges were established.

weakened, and respiratory rates were increased. Mucous membranes showed marked reddish-blue discoloration, with prolonged capillary refill time. The skin, muzzle, and extremities were cold to the touch, and some horses were "cold-sweating" heavily. Due to the rapidity of the dehydration, typical signs like sunken eyes and tenting of the skin were not prominent. Splashing and sometimes tingling sounds could be heard on auscultation of the abdomen. Jugular vein thrombosis after venipuncture or catheterization became a serious problem in several horses. In one particular horse, which had to be destroyed for humane reasons, bilateral jugular vein thrombosis lead to swelling of the head, which caused great hindrance to respiration, mastication, and swallowing. Some horses exhibited a markedly increased bleeding tendency after venipuncture or arterial puncture. Laboratory tests for coagulation parameters were not performed.

In the surviving horses, fever lasted for just a few hours and formed feces usually reappeared on the following day. Appetite was always reduced, but food intake was generally better in the survivors than in the non-survivors.
Some of the non-surviving horses had persisting or relapsing fever, the diarrhea persisted, and the horses were generally more depressed.

Clinical pathology
Dehydration, acidosis, and leukopenia were marked at the time clinical signs occurred.
The ranges of some of the affected variables in blood, serum, and plasma are shown in Table 1. Extreme values were not observed in each and every horse, but the PCV and total protein were generally high due to dehydration.
Leukopenia was common with lymphopenia, neutropenia with a left shift, and the appearance of toxic neutrophils.
All horses except one had moderate to severe

non-respiratory acidosis indicated by large negative values of the Standard Base Excess. Arterial blood pH values were kept closer to the reference range because of respiratory compensation, although some values were quite low. Sodium loss was a very important factor for the development of acidosis with lactate accumulation contributing only moderately. Initially, hyperproteinemia due to dehydration also contributed to the acidosis. As the disease proceeded, large amounts of protein were lost in the feces resulting in hypoproteinemia. Total protein values down to 38 (survivor) and 36 g/l (non-survivor) were recorded.

Blood samples from 2 of 3 horses tested positive for endotoxin with values of 0.1 (non-survivor) and 0.2 ng/ml (survivor), respectively. The negative sample came from a surviving horse.

Treatment

Therapy was directed towards replacement of fluid loss, counteracting inflammation, restoration of acid-base and electrolyte balance, and counteracting intestinal fluid secretion.

Large amounts of acetated Ringer's solution were given i.v., up to 60 l per day.

The amount of sodium bicarbonate needed to correct acidosis was found using the following formula: $-1 \times$Standard Base Excess$\times 0.3 \times$body –weight (in kg), which estimates the whole-body base-deficit in mmol. Sodium bicarbonate was then given i.v. as a 4.2% solution for immediate correction of the acidosis. One to 2 litres of 1.1% potassium chloride were mixed with the sodium bicarbonate solution to prevent hypokalemia. The mixture was given by free gravity flow through a 13-gauge catheter. In spite of acidosis, plasma potassium levels were low in most of these horses, strongly suggesting large potassium deficits. Additional blood samples were taken immediately after sodium bicarbonate administration, to assess the effect of this therapy. These samples showed increases of the standard base excess values into the reference range, and the maximal plasma sodium concentration recorded was 148 mmol/l. After correction of the acidosis, fluid therapy was continued with acetated Ringer's intravenously. Some horses also received isotonic potassium chloride or a balanced electrolyte solution with a stomach tube. However, because of the increased bleeding tendency in many cases, we tried to keep the number of nasal tube passages low.

For intestinal protection and absorption of enteral toxins, either activated charcoal, cultured milk or barium sulphate was given by stomach tube. Dosages were 400-500 grams of activated charcoal, 5-6 l of cultured milk or 3-5 l of barium sulphate, repeated every 6-12 h. The effects of these different treatments were roughly equal.

Flunixin meglumine was given i.v. as an antiinflammatory agent. The dosage was 0.3-1.1 mg/kg 2-4 times daily.

Pathological findings

The necropsy revealed conspicuous lesions in the intestinal tract of 6 horses. The small intestine and, to some extent, the large intestine, in these animals were dilated and filled with watery greyish contents. The intestinal mucosa looked pale in all except one case, which had evident hyperemic changes in the cecum and colon. Microscopical examination of the intestines revealed small necrotic and thrombotic lesions in the mucosa of the large intestines. The adrenal cortices of at least two cases showed hemorrhagic and necrotic changes.

The intestinal lesions of the seventh necropsy case included only a marked edema in the wall of the colon for a length of 2 feet. The microscopical examination of these lesions showed, besides edematous changes, infiltration of eosinophilic and mononuclear cells in the mucosa

and submucosa. This horse had been castrated, and 1 of the ligatures had failed to occlude the spermatic artery. There was a substantial loss of blood into the abdominal cavity, and the combination of hemorrhage and colitis had led to severe shock and death less than 24 h after surgery. It was assumed that coagulation failure due to the colitis greatly increased blood loss, but it is also likely that the hemorrhage was an important factor for the development of the colitis.

Bacteriology

Bacteriological examination of feces or intestinal contents did not reveal any infectious agent common to these horses. *Clostridium perfringens* was isolated from 2 horses; only once, however, were the numbers large enough to suggest a diagnosis of intestinal clostridiosis. In some of the other cases, *E. coli* was isolated in relatively large numbers. An abnormal flora containing large numbers of *Bacillus spp.* was found in 1 case. *Salmonella* spp. were not detected.

Discussion

The distribution of breed, sex, and age of the affected horses seems to match the general distribution among the patients in our clinic. The clinical findings in these horses are similar to the disease called "Colitis X" or "exhaustion shock" *(Rooney et al.* 1963, *Rooney et al.* 1966). There is also a similarity in the fact that it, in most cases, has not been possible to identify any causative agent with certainty *(Rooney et al.* 1963, *Rooney et al.* 1966, *Hermann* 1985, *Kraft* 1985).

The resemblance of *Salmonella* enterocolitis is apparent *(Murray* 1992), and the clustering of cases suggests an infectious etiology. However, Salmonellosis is a notifiable disease in Norway and rarely occurs in horses and farm animals, including poultry *(Anon.* 1992, 1993, *Kapperud et al.* 1994).

In enzootic areas, a fatal acute colitis without gross pathological lesions of the cecal and colonic mucosa would suggest a diagnosis of Potomac horse fever (Equine monocytic ehrlichiosis) *(Cordes et al.* 1986). However, the occurrence of Potomac horse fever has never been sufficiently documented in Europe.

Equine colitis can be looked upon as a multifactorial disease which can be initiated by specific infectious and toxic agents, stress, use of antibiotics, disruption of the indigenous intestinal microbial flora, and, possibly, impairment of intestinal oxygenation and motility *(Argenzio* 1978, *Nielsen & Vibe-Petersen* 1979, *Whitlock* 1986, *Palmer* 1992, *Radostits et al.* 1994). Endotoxin is probably crucial in the pathogenesis of colitis *(Murray* 1988, *Palmer* 1992). The endotoxin levels detected in 2 of the horses, correspond well with the findings reported by *King & Gerring* (1988) and *Fessler et al.* (1989). As the endotoxin may be present in the circulation only for a short period of time *(Meyers et al.* 1982), its absence in the third horse does not exclude the presence of or importance of endotoxemia in this animal.

A contributory effect of phenylbutazone to the development of the colitis in the horses studied should not be completely ruled out, even though cecal and colonic ulceration was not prominent. Profuse diarrhea with severe toxemia due to massive colonic ulceration and necrosis has been reported after high doses of phenylbutazone *(Collins & Tyler* 1984).

Our treatment was fairly in accordance with the therapy suggested by other authors *(Whitlock* 1986, *Murray* 1990, *Palmer* 1992). Compared to these guidelines, our corrective therapy against acidosis seems to be rather aggressive. The extremely rapid sodium loss, reflected in the low serum sodium concentrations measured, justifies the relatively rapid sodium re-

placement. Abnormally high serum sodium levels were not recorded, even immediately after treatment with hypertonic sodium bicarbonate solution. The intravenous infusion of hypertonic sodium bicarbonate solution seemed to be of great benefit to the affected horses. The correction of acidosis via restoration of the strong ion difference of the extracellular fluid made the horses much more bright and alert, and their appetite increased. Additionally, it is possible that this hypertonic solution prevented or reduced cell swelling due to extracellular fluid hypotonicity similar to the effect of hypertonic saline *(Bertone* 1991).

The rapidly developing hypoproteinemia was an indication for plasma therapy. However, commercial equine plasma products are not readily available in Norway. Time did not always allow the preparation of plasma from the blood of a suitable donor.

Preventive efforts

As we did not know what specifically had caused the diarrhea in these horses, we had to improve our general preoperative, anesthetic, and postoperative procedures to reduce the risk of compromizing normal intestinal and hepatic function. Several routines were evaluated and improved:

– Feeding. We reduced preoperative fasting, and refeeding was started as soon as possible. At present, horses are fed hay, but no concentrates, on the day before surgery. No morning feed is given on the day of surgery, but the horses have access to water.

Fasting reduces small intestinal absorption of carbohydrate *(Breukink* 1974) and may alter the indigenous microflora of the cecum and colon *(Meyer* 1991). Upon refeeding, excessive amounts of rapidly fermentable carbohydrate may reach the cecum, and a significant reduction of luminal pH with abrupt changes of the intestinal flora may result *(Whitlock* 1986).

– Premedication. Prior to anesthesia, detomidine was routinely used for premedication at a usual dose rate of 10 micrograms per kg. Detomidine can cause bradycardia and heart block for a short period shortly after intravenous administration *(England et al.* 1992). Unfortunately, many horses were given guaifenesin and thiopental sodium for induction of anesthesia almost immediately after the injection of detomidine. Such a procedure may lead to rapid reductions of cardiac output and tissue perfusion, which also affects the intestine.

From 1992, acepromazine was used for premedication in most cases. More important than the choice of drugs, however, is to allow sufficient time to elapse between premedication and induction of anesthesia, so that heart rate, cardiac output, and blood pressure can stabilize. Administered at the correct time, about 20 min prior to induction, detomidine is considered a safe drug, and α_2-agonists are currently back in routine use.

Some of the horses that developed colitis had received sulfadoxin/trimethoprim intravenously shortly before or after the administration of detomidine. These drugs should not be given concurrently due to the risk of serious cardiac arrythmia *(Dick & White* 1987, *Taylor et al.* 1988). The importance of this procedure with respect to the development of colitis is not known, but it is possible that it may have lead to temporary cardiovascular alterations in some horses.

– Anesthesia. Horses are now preoxygenated before induction of anesthesia, and they are rapidly intubated. The use of artificial or assisted ventilation with adequate monitoring greatly reduces the risk of hypoxia and hypercapnia. The use of nitrous oxide for inhalation anesthesia is now minimal.

– Horses at risk. It is important to avoid all kinds of additional stress during treatment of horses already in great pain. If possible, we now

try to treat horses with infectious arthritis or tenosynovitis in the standing position under sedation.

Our efforts to prevent the occurrence of post-treatment colitis seem to have succeeded, as this disease is presently rare in our clinic: From February 1992 through June 1994, only 1 additional case has occurred.

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Sammendrag

Akutt enterocolitt oppstått hos 13 hester etter kirurgisk behandling.

Kliniske symptomer, laboratorieresultater, behandling, forløp og patologiske forandringer er beskrevet hos 13 hester som utviklet akutt enterocolitt etter ulike kirurgiske inngrep. Tolv av de 13 hestene hadde vært sedert og kastet i sideleie, hvorav 10 hadde blitt lagt i generell anestesi. Elleve av de 13 hestene hadde blitt behandlet med antimikrobielle midler. De fleste hestene (11) fikk symptomer 2 dager etter innsatt behandling, med en variasjon på 1 til 5 dager. De viktigste symptomer var feber opp mot 40.5°C, rask hjerteaksjon, misfargede slimhinner og diaré. Det var tydelig dehydrering, acidose og leukopeni ved utbrudd av de kliniske symptomer. Behandlingen bestod hovedsakelig i å erstatte væsketap, gjenopprette syre-basebalansen og motvirke inflammasjonsprosessen. Syv hester (54%) døde eller ble avlivet. Obduksjonsfunnene viste store individuelle variasjoner. *Salmonella* spp. ble ikke påvist hos noen av hestene, og intet felles etiologisk agens ble funnet. Preventive tiltak, som så ut til å ha god effekt, ble iverksatt med hensyn til preoperative rutiner, premedikasjon og anestesi.

(Received March 13, 1995; accepted March 7, 1996).

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