Equine cyathostomosis: case reports

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ABSTRACT: Twelve clinical cases of cyathostomosis in horses treated at the Equine Clinic University of Veterinary and Pharmaceutical Sciences in Brno, the Czech Republic, between the years 1999 and 2008 are described in this report. Six cases (50%) were hospitalized in the period from 2007 to 2008. Eleven of them were hospitalized in the period from December to March. Only one case was admitted in June, but the clinical signs had appeared for the first time in January. All horses described in these cases were younger than six years of age. Diarrhoea as a predominant clinical sign was present in four horses and colic in four horses. One horse showed both colic and diarrhoea whilst three horses had weight loss and subcutaneous oedema. Metabolic acidosis was found in three horses, eight patients had leucocytosis. Hypoproteinemia was found in four horses, hypoalbuminemia in seven horses, hypokalemia in three horses and increased alkaline phosphatase (ALP) activity in five horses. Seven horses recovered, one horse died and four horses were euthanized.

Keywords: cyathostome infection; small strongyles; diarrhoea; colic; hypoalbuminemia

Small strongyles (cyatostomes) were previously considered as parasites with minimal pathogenic influence on the equine organism. With increasing levels of resistance, small strongyles have become a limiting factor in anthelmintic treatment within the horse population. The increasing incidence of these parasites in the gastrointestinal tract of horses has lead to the increasing occurrence of clinical cyathostomosis to such an extent that it is considered an emerging infection (Lyons et al., 2000).

Cyathostomosis particularly affects young horses (≤ 6 years) during the late winter and early spring months (Reid et al., 1995). Clinical signs include diarrhoea (Mair et al., 1990; Love et al., 1992; Mair, 1993), subcutaneous oedema, intermittent colics and weight loss (Murphy et al., 1997; Smets et al., 1999). Several different clinical syndromes connected with cyathostome infection are distinguished by Mair (2002):

1. Acute protein loosing diarrhoea, which can be followed by a chronic stadium.
2. Recurrent diarrhoea that can occur in aged horses as well.
3. Rapid weight loss and subcutaneous oedema, which is a consequence of hypoalbuminemia.
4. Seasonal „malaise syndrome". The main clinical signs are lethargy, inappetence and variable faecal consistency.
5. Non-specific colics. There is a presumed significance of small strongyles in the aetiology of recurrent colics.
6. Small strongyles are probably the agents of non-strangulated intestinal infarcts and caecal tympany (Mair and Pearson, 1995).
7. On the basis of the contemporary occurrence of caecocolic or caecocaecal intussusceptions and larval cyathostomosis in several horses, the connection between these two pathological conditions is presumed (Mair et al., 2000; Love, 2002).

Diagnosis of cyathostomosis is difficult because the faecal egg counts are often very low and clinical (diarrhoea, weight loss, colic, subcutaneous oedema, pyrexia) as well as laboratory signs (neutrophilia, anaemia, hypoalbunemia, hypoproteinemia, elevation of β globulins, elevation of alkaline phosphatase, decreased blood ions) can be variable.

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To establish a proper diagnosis, it is necessary to prove the presence of larvae in the gastrointestinal tract – in the faeces, in the intestinal mucosa biopsy or during necropsy (Love et al., 1999; Lyons et al., 2000; Mair, 2002; Kaplan and Matthews, 2004).

Therapy for clinical cyathostomosis consists of five daily doses of fenbendazole followed up by a single dose of ivermectin (Love and McKeand, 1997; Duncan et al., 1998), or alternatively only one dose of moxidectin (Deprez and Vercruysse, 2003). The later was proved to cause less damage to the intestinal mucosa due to inflammation (Steinbach et al., 2006). Corticosteroids are recommended for larval cyathostomosis treatment, together with supportive therapy in severe cases of fluid loss, hypoproteinemia and acid-base imbalance (Love and McKeand, 1997; Mair, 2002).

The aim of this retrospective study was to evaluate the prevalence of clinical cyathostomosis at the Equine Clinic of the University of Veterinary and Pharmaceutical Sciences in Brno in the period from January 1999 to November 2008, to specify seasonality and clinical signs of this disease, describe changes in haematology and biochemistry, assess response to therapy and the clinical outcome of the disease.

Case description

History of patients. Altogether 12 horses with cyathostomosis were included in the retrospective study. All horses were treated at the Equine Clinic of the University of Veterinary and Pharmaceutical Sciences in Brno between January 1999 and December 2008. Basic data and history of these horses are summarised in Table 1.

Examinations performed and diagnostic criteria. A clinical examination was performed on all horses admitted to the clinic including a rectal palpation (except in case 3 because of the small body size of the horse), followed by haemathology, bio-

Table 1. Basic data and history of horses with cyathostomosis treated at the clinic from January 1999 to December 2008

Data of the horse				History		
Case No.	breed	sex	age	clinical signs	previous treatment	date of admission
1	Thoroughbred	mare	4 years	diarrhoea, weight loss, inappetence	–	March 1999
2	Thoroughbred	mare	6 years	diarrhoea for 6 months, weight loss	–	June 1999
3	Shetland pony	gelding	4 years	intermittent colic, inappentence	–	February 2002
4	Czech Warmblood	mare	1.5 year	profuse diarrhoea, weight loss	penicillin	December 2003
5	Friesian horse	stallion	2.5 years	intermittent diarrhoea, subcutaneous oedema, weight loss	penicillin	January 2004
6	Thoroughbred	mare	4 years	intermittent colic, diarrhoea, inappetence	–	February 2006
7	Dutch Warmblood	stallion	1 year	intermittent colic, weight loss	flunixin meglumine	January 2007
8	Slovak Warmblood	stallion	7 months	diarrhoea, subcutaneous oedema, weight loss, inappetence	moxidectin, praziquantel	January 2008
9	Slovak Warmblood	stallion	7 months	diarrhoea, subcutaneous oedema, weight loss, inappetence	moxidectin, praziquantel	January 2008
10	Paint horse	stallion	2.5 years	diarrhoea, inappetence	penicillin, flunixin meglumine, ivermectin, praziquantel	January 2008
11	Czech Warmblood	mare	3 years	colic, diarrhoea	–	March 2008
12	Friesian horse	stallion	5 years	intermittent colic, weight loss, diarrhoea for 2 weeks, inappetence	penicillin, flunixin meglumine	March 2008

chemistry and base-acid balance. Ultrasonography of the abdomen was performed in cases 7 and 12.

Faecal examination including flotation of intestinal worm eggs with macroscopic and microscopic examinations of faeces (searching for small strongyle larvae) was performed on all horses.

Rectal biopsy was performed in three horses during the rectal examination using forceps for transendoscopic biopsy. Histopathology revealed non-specific intestinal inflammation in cases 1 and 4 and eosinophilic inflammation in case 2.

Horses were included in the study on the basis of typical clinical signs and finding larvae in the faeces intravitally and/or by histopathologic examination of the sample of intestinal mucosa.

Treatment. Ten patients were treated with anthelmintics as a part of the therapy at the clinic. Nine of them (cases 1, 2, 4–6, 8–11) were treated by repeated administration of larvicidal doses of fenbendazole (10.0 mg/kg b.w.). This treatment was followed up by a single dose of ivermectin (0.2 mg/kg b.w.) in six horses (cases 4–6, 8, 9, 11). Moxidectin (0.4 mg/kg b.w.) was administered to one horse (case 3) without any previous treatment with fenbendazole. Anthelmintic therapy was not used in two horses (cases 7 and 12) and these horses were euthanized due to an unfavourable prognosis.

Patients 4, 8, 9, 10 and 11 were treated with corticosteroids, dexamethasone (0.1 mg/kg b.w. intravenously) and prednisolone (1.0 mg/kg orally in a descending course) in addition to the anthelmitics. Sulfonamides potentiated by trimethoprim (20.0 mg/kg b.w. intravenously) were administered to five horses (cases 1, 4, 5, 10 and 11). Intravenous fluid therapy (Ringer's solution with the addition of potassium chloride and sodium bicarbonate) was used in four horses (cases 4, 5, 10 and 11). One horse was given a plasma infusion because of severe hypoproteinemia (case 4). Cimetidine (7.7 mg/kg b.w. intravenously) as an antiulcerous treatment was administered to three horses (cases 4, 5 and 8).

Celiotomy was performed in two horses – case 6 (caecal impaction) and case 7 (caecocaecal intussusception).

RESULTS

Altogether 1497 horses with gastrointestinal diseases were admitted and treated at the clinic in the period from January 1999 to December 2008. Twelve horses with the diagnosis of clinical cyathostomosis represent 0.80% of all gastrointestinal cases. Eleven of them were hospitalized in the period from December to March (one in December, five in January, two in February and three in March). Only case 2 was admitted in June, but the clinical signs had appeared for the first time in January in this horse. Six cases (50%) were hospitalized in the period from 2007 to 2008.

Cyathostomes larvae were found in the faeces intravitally in nine cases (cases 3–6, 8–12). In five patients the presence of small strongyles was proved by histopathology *post mortem* (cases 1, 5, 7, 10 and 12). In these patients, caecal and ventral colon inflammation was found and encysted L4 larvae of small strongyles were present in the mucosa of the large colon. In case 2 the diagnosis of "cyathostomosis" was based on histopathology of the rectal biopsy and regress of clinical signs after the beginning of treatment.

Clinical signs and disease outcome are summarized in Table 2. Acute diarrhoea as a predominant clinical sign was present in three horses (cases 1, 4, 10) and chronic intermittent diarrhoea in one horse (case 2). Colic as a predominant clinical sign was present in four horses (cases 3, 7, 11 and 12). One horse showed colic and diarrhoea (case 6), three horses (cases 5, 8, 9) had non specific clinical signs – weight loss, subcutaneous oedema and inappetence.

Caecal impaction was found rectally in case 6 and confirmed by celiotomy. Caecocaecal intussusception was found in case 7 by ultrasonography and confirmed by celiotomy. Caecocolic intussusception was found by ultrasonography and confirmed by necropsy in case 12.

Seven horses (58%) recovered; five horses (42%) were euthanized or died.

Blood analyses

Metabolic acidosis was found in three horses (cases 4, 5 and 10; pH 7.24–7.28). PCV was elevated in case 4 (0.58 l/l) and decreased in case 3 (0.15 l/l). Eight patients had leucocytosis varying between 14.9–38.0 G/l (cases 1, 3, 4, 5, 7, 8, 9 and 10), one of them had eosinophilia 3% (case 1). Hypoproteinemia was found in four horses (cases 1, 4, 5 and 12; range 31.5–47.3 g/l), hypoalbuniemia in seven horses (cases 1, 4, 5, 8, 9, 10 and 12; range 12.6–21.6 g/l) and hypokalemia in three

horses (cases 4, 5 and 10; range 1.93–3.10 mmol/l). Increased alkaline phosphatase (ALP) activity was found in five horses (cases 5, 7, 8, 9, 11; range 291–1 350 IU/l).

DISCUSSION AND CONCLUSIONS

It was possible to distinguish several clinical forms of cyathostomosis as described by Mair (2002) within our group of horses. However, strict division is impossible since some horses showed several clinical signs depending on the phase of the disease.

In 25% of cases, the predominant clinical sign was profuse diarrhoea with an acute onset (cases 1, 4, 10). Additional signs included colic, pyrexia and subcutaneous oedema (case 4, Figure 1). Clinical diagnosis was only suspected in case 1 because of the negative result of faecal examination. The diagnosis was confirmed by histopathology. In contrast a high number of larvae were found in the faeces in cases 4 (Figure 2) and 10. Similarly Love et al. (1992) established the diagnosis ante mortem in only seven out of 14 horses with chronic diarrhoea caused by cyathostome infection.

The prognosis of larval cyathostomosis with the clinical manifestation of severe diarrhoea is uncertain. In the report of Love et al. (1992) five horses

Figure 1. Hypoproteinemia – associated subcutaneous oedema in case No. 4

Table 2. Clinical signs and disease outcome of horses with cyathostomosis treated at the clinic from January 1999 to December 2008

Case No.	Clinical signs	Duration of disease after beginning the treatment	Clinical outcome
1	profuse diarrhoea	8 days	euthanasia
2	chronic intermittent diarrhoea	5 days	recovery
3	intermittent colic	14 days	recovery
4	diarrhoea, subcutaneous oedema, dehydration, febrilia (39.3°C), inappetence	27 days	recovery
5	weight loss, subcutaneous oedema, febrilia, (39.8°C), inappetence	3 days	exitus
6	intermittent colic, diarrhoea	6 days	celiotomy, recovery
7	colic	2 days	celiotomy, euthanasia
8	weight loss, subcutaneous oedema	11 days	recovery
9	weight loss, subcutaneous oedema	11 days	recovery
10	diarrhoea, subcutaneous oedema, inappetence	5 days	euthanasia
11	intermittent colic	6 days	recovery
12	colic, inappetence	1 day	euthanasia

survived and nine died. Van Loon et al. (1995) described two cases of larval cyathostomosis in regularly dewormed horses. One of them died, the second one was euthanized despite intensive treatment. Murphy et al. (1997) reported three cases of larval cyathostomosis with diarrhoea; one horse recovered and two were euthanized. In our report only one of three horses recovered (case 4); the other two horses were euthanized (case 1 and 10).

25% of cases in our group (5, 8 and 9) were presented because of rapid weight loss, subcutaneous oedemas and mild intermittent diarrhoea in some of them. Similarly in the studies of Smets et al. (1999) and Peregrine et al. (2006) reporting 24 cases, weight loss and colic were the second most common clinical signs of cyathostomosis after diarrhoea. In case 5 of our group homeostasis was severely affected despite the fact that the horse did not exhibit diarrhoea. Similarly to the report by Murphy et al. (1997), faecal output was reduced in this horse, but, unlike these previous cases, our patient died. Clinical signs in this horse were most probably caused by endotoxin resorption as a consequence of damage to the intestinal barrier.

In 25% of horses (cases 3, 6, 11) the predominant clinical sign was intermittent colic. Case 3 also showed inappetence and a reluctance to move. The anaemia that was found in this patient was probably caused by bleeding into the intestine. Another two horses from this group had mild intermittent diarrhoea as an additional clinical sign. All horses from this group excreted cyathostome larvae massively (Figure 3, case 11) and the diagnosis was established intravitally in these cases. All of them recovered.

Recurrent diarrhoea as a syndrome of cyathostomosis was described previously in five aged ponies and all except one responded to therapy by oral ivermectin (Mair, 1993). In our study recurrent diarrhoea was observed in a six year old Thoroughbred mare (case 2). The patient suffered with diarrhoea which lasted six months without resulting in any alteration of the general status and clinical signs disappeared after one dose of fenbendazol (10 mg/kg b.w.).

Caecocolic or caecocaecal intussusceptions were found in two cases (case 7 and 12). In both horses the diagnosis of cyathostomosis was confirmed by necropsy and large amounts of larvae were found in the caecal and colonic mucosa (Figures 4 and 5). The association between cyathostomosis and intestinal intussusception has been speculated upon by Mair et al. (2000) on the basis of four cases but this hypothesis has yet to be confirmed.

Haematology and blood biochemistry were performed on all patients. Hypoalbuminemia was present in seven horses (case 1, 4, 5, 8, 9, 10 and 12) from our group. Three horses presented normoproteinemia with hypoalbuminemia (case 8, 9 and 10). Similarly Love et al. (1992) observed hypoalbuminemia and hypoproteinemia in 11 out of 14 horses with diarrhoea caused by cyathostome infection and Peregrine et al. (2006), in 5 of 6 horses with cyathostomosis. Smets et al. (1999) found a significant relationship between hypoproteinemia and the presence of cyathostome L4 in the faeces of horses.

Leucocytosis was observed in 66% of cases in our study. Love et al. (1992) found neutrophilia in 8 of 14 cases with diarrhoea caused by cyathostome

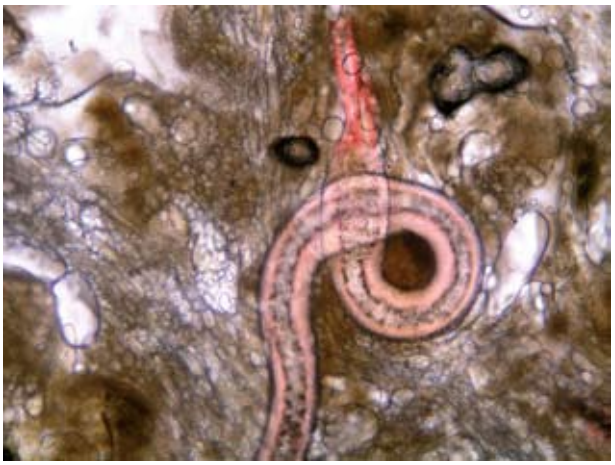

Figure 2. Cyathostoma larvae in microscopic examination of faecal smears in case No. 4

Figure 3. Cyathostoma larvae in faeces in case No. 11

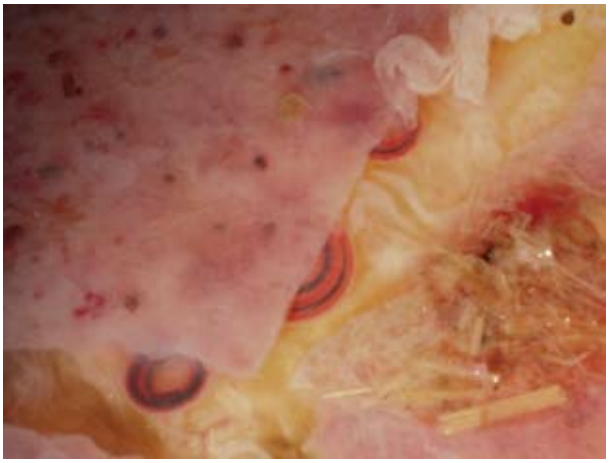

Figure 4. Cyathostoma larvae in caecal mucosa in case No. 7

Figure 5. Caecocolic intussusception, *post mortem* examination (case No. 12)

infection. Eosinophilia and anaemia were found in one horse. Metabolic acidosis was observed in two horses with profuse diarrhoea (case 4 and 10). In case 5 (the horse with inappetence and weight loss) the acidosis was probably the consequence of severe endotoxemia.

Increased ALP activity was observed in five horses. All of them except one had ALP activities in the range 291–357 IU/l. A dramatic increase (1 350 IU/l) was found only in the horse suffering from inappetence, subcutaneous oedema and weight loss (case 5). Murphy et al. (1997) described increased ALP in three out of four cases with cyathostomosis, Love et al. (1992) in six out of 14 cases with chronic diarrhoea caused by cyathostomes infection and Murphy and Love (1997) in one out of nine ponies infected experimentally. These results suggest that increased activity of ALP is not a consistent laboratory finding in horses with cyathostomosis.

Clinical cyathostomosis usually affects one or only a few horses in a herd. The reason for this fact is probably individual predisposition of some horses as well as the patchy distribution of parasites in the herd (Mair, 2002). In most of our cases also, only one horse from the herd was affected. There was only one exception – horses 8 and 9 originated from the same stud.

A primary mechanism of larval cyathostomosis is synchronic larval migration from the large colon wall to the lumen causing damage to the mucosa. Routine anthelmintic treatment or a stressful situation are considered to be the most important stimuli initiating the migration (Reid et al., 1995). In our group, there was no patient with a history of the onset of clinical signs after anthelmintic therapy. In only three horses (8, 9 and 10) anthelmintics were administered as a part of treatment before their hospitalization. In patients 8 and 9, a combination of moxidectin-praziquantel was administered. Since no improvement was apparent, a five day treatment with fenbendazol was carried out at the clinic, followed by a single application of ivermectin. In case 10, a combination of ivermectin-praziquantel was administered after the onset of clinical signs but this treatment did not influence the clinical course of the disease.

The incidence of clinical cyathostomosis in the Czech Republic is not high (0.80% from the group of patients with GIT disorders) but it is probably increasing, because whilst seven cases were hospitalized at the clinic in the period 1999–2007, five cases were recorded in the year 2008. One of the reasons for this fact could be the mild winter between years 2007/2008. The real incidence could be higher, however, because there is no effective way of determining the burden of larval cyathostomes in the wall of the large intestine of the horse ante mortem apart from surgical biopsy. A similar increase in incidence was observed by Peregrine et al. (2006), describing 24 cases of cyathostomosis in the years 1991–2003; 15 of them appeared during the period 2001–2003.

The results of this retrospective study suggest that cyathostomosis has become a more common disorder in the Czech Republic in recent years. The reason for this is probably the growing resistance of small strongyles to anthelmintic drugs and as a consequence an increase in small strongyle larvae numbers in the large colon in some horses. A diagnosis of cyathostomosis should be considered in

horses with diarrhoea, colic, weight loss or a combination of these clinical signs that appear in the winter or early spring months. Moreover, a negative result for the presence of larvae in faeces does not rule out the presence of parasites.

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