# Case Study - Peritonitis

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Peritonitis can pose a diagnostic and therapeutic challenge. Four cases of peritonitis are presented (Tables 1, 2 and 3), with discussion on the causes, investigation, treatment and prognosis.

## RESPONSE TO TREATMENT

### Case 1

Case one responded well to medication initially. On day 3, despite improving clinical signs (decreasing heart rate and temperature, improved demeanour and appetite), repeat abdominocentesis revealed a significant increase in white cell count (250 × $10^9$/l) with large numbers of degenerate neutrophils evident on smear. Similar samples were obtained from both the hernia sac and ventral midline. Abdominal ultrasound was unchanged. Haematology revealed an increase in white cell count (8.9 × $10^9$/l) and fibrinogen (1241 mg/dl). Oral metronidazole (15 mg/kg PO q 8h) was added to the treatment regimen, in addition to oral omeprazole(4mg/kg PO q 24h).

The colt showed improved demeanour and clinical parameters over the following 48 hours. Repeat abdominocentesis revealed fluid with a total protein of 15 g/l and a white cell count of 27.5 × $10^9$/l. After a five-day course of penicillin and gentamicin, the colt was discharged with a two-week course of oral metronidazole and trimethoprim sulpha (30 mg/kg PO q12h), plus reducing doses of NSAIDs and oral omeprazole. Due to the high worm egg count and poor worming history, anthelmintics were also prescribed. The hernia was surgically repaired at a later date following resolution of the peritonitis.

### Case 2

Case 2 responded well to medication and the pyrexia resolved within 24 hours. On day 3, the peripheral white cell count had decreased to 2.5 × $10^9$/l. Abdominal ultrasound was unremarkable, no free peritoneal fluid was imaged and none obtained by paracentesis. By day 5, the peripheral white cell count had increased to 6.8 × $10^9$/l and the horse was clinically normal. He was discharged with a ten-day course of oral enrofloxacin (7.5mg/kg PO q24h) and made an uneventful recovery.

### Case 3

Case 3 showed evidence of acute bowel rupture and was euthanased immediately. Post-mortem examination revealed rupture of the right dorsal colon.

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**TABLE 1: Presentation**

| | Case 1 | Case 2 | Case 3 | Case 4 |
|---|---|---|---|---|
| **Signalment** | 8-month-old Warmblood colt | 10-year-old Thoroughbred gelding | 16-year-old Thoroughbred gelding | 13-year-old Thorough Welsh gelding |
| **Presenting complaint** | Dull, depressed, mild colic, sweating | Mild colic, dull, depressed | Unresponsive colic of 24 hours' duration, with worsening clinical parameters | Mild colic, inappeten depressed |
| **Abnormalities on initial clinical examination** | Dull<br>HR 60 bpm<br>RR 40 brpm<br>T 40.1°C<br>Dark mucous membranes<br>Umbilical hernia, 4 cm wide (previously treated with an elastrator ring), which was thickened and painful on palpation | Dull<br>RR 26 brpm<br>T 39°C | Dull demeanour<br>Cold extremities<br>Purple, congested mucous membranes<br>Prolonged capillary refill time (>5 seconds)<br>Poor pulse quality<br>HR 90 bpm<br>RR 60 brpm<br>T 40°C<br>Large, distended viscus detected on rectal examination<br>Gritty feeling to serosal surfaces | No significant abnor detected |

**TABLE 2: Test results**

| Test | Parameter | Reference values | Case 1 | Case 2 | Case 3 | Case 4 |
|---|---|---|---|---|---|---|
| **Haematology** | White cell count (× $10^9$/l) | 5-10 | 5.3 | 10.1 | 1.0 | 3.6 |
| | Differential (%) | Neutrophils 40-60 Lymphocytes 30-34 Monocytes 0-6 Eosinophils 0-3 Basophils 0-1 | Neutrophils 90, of which 10 were band neutrophils Lymphocytes 10 | Neutrophils 87 Lymphocytes 13 | | Neutrophils 71 Lymphocytes 29 |
| | Fibrinogen (mg/dl) | <320 | 443 | 600 | 365 | 502 |
| **Peritoneal tap** | Gross appearance | | High volume, cloudy, turbid | Serosanguineous | Profuse, malodorous, green/brown fluid containing particles of ingesta | Small volume, cloudy and slightly discoloured |
| | White cell count (×109/l) | <5 | 150 | 74 Phagocytosed bacteria noted on smear | Not performed | 94 |
| | Total protein (g/l) | <25 | 50 | 30 | Not performed | 76 |
| | Culture results | | -ve | -ve | Not performed | -ve |
| **Additional tests** | | | Faecal egg count: 1000 epg | N/A | N/A | N/A |
| **Results of abdominal ultrasound** | | | Moderate amount of flocculent fluid within the hernia sac Reduced small and large intestinal motility Increased volume of anechoic peritoneal fluid | Increased volume of anechoic peritoneal fluid | Marked amounts of heterogeneous free peritoneal fluid Markedly thickened large bowel wall (6 mm) with mural tramlines | No abnormalities detected |

**TABLE 3: Initial treatment received**

| Case 1 | Case 2 | Case 3 | Case 4 |
|---|---|---|---|
| Gentamicin IV 6.6mg/kg q 24h | Gentamicin IV 6.6mg/kg q 24h | 4l hypertonic saline (7.4% NaCl) | Gentamicin IV 6.6mg/kg q 24h |
| Sodium penicillin IV 40,000 iu/kg q 6h | Flunixin meglumine IV 0.5mg/kg q 12h | IV during initial examination | Sodium penicillin IV 40,000iu/kg q 6h |
| Flunixin meglumine IV 0.5mg/kg q 12h | Procaine Penicillin IM 20mg/kg q 12h | | Flunixin meglumine IV 0.5mg/kg q 12h |
| | | | Metronidazole PO 15mg/kg q 8h |

**Case 4**

Case 4 showed a poor response to treatment, with on-going pyrexia, dullness and inappetence. On day 7 there was further deterioration, namely worsening pyrexia, tachycardia, tachypnoea and hyperaemic mucous membranes. Peritoneal fluid was very cloudy with a white cell count of 172 × $10^9$/l and protein of 52 g/l. Abdominal ultrasound showed increased peritoneal fluid and thickened mesentery on the left side.

Exploratory laparotomy was performed and revealed diffuse proliferative peritonitis over the intestinal surfaces (Fig. 1). There was a large 'knot' of adhered mid-jejunum with multiple adhesions between loops and a large caseous abscess in the centre. Multiple enlarged lymph nodes were present within the mesentery. The ileum could not be exteriorised. The horse was euthanased on the table and culture of aspirated material identified *Streptococcus equi* var. *equi*. Further questioning of the owner revealed an outbreak of 'strangles' on the yard several months earlier, although this horse had not shown any clinical signs at the time.

## DISCUSSION

Peritonitis is inflammation of the mesothelial lining of the peritoneal cavity, and may result from a mechanical, chemical or infectious insult to the parietal peritoneum (Table 4). Inflammation results in exudation of fibrin, serum, inflammatory cells and pus into the peritoneal cavity. It can be classified according to its causal agent (primary or secondary),

**TABLE 4: Causes of peritonitis**

| | |
|---|---|
| **Infectious/septic** | Compromised bowel (intestinal ischemia, proximal enteritis, intestinal perforation) or uterus allowing transmural movement of bacteria |
| | Abdominal abscess (usually associated with *Streptococcus equi* var *equi* in the UK, i.e. 'bastard strangles') |
| | Uterine rupture/perforation |
| | Metritis |
| | Cholangitis |
| | Septicaemia |
| **Non-septic** | Foreign body |
| | Neoplasia (abdominal/ovarian) |
| | Renal disease |
| | Rupture of intra-abdominal part of urinary tract |
| | Acute pancreatitis |
| **Parasitic** | Verminous arteritis |
| | Cyathostominosis |
| | Perforation from tapeworms or ascarids |
| **Traumatic** | Breeding or foaling injuries |
| | Blunt or penetrating abdominal trauma |
| | Ruptured diaphragm |
| | Splenic rupture |
| **Iatrogenic** | Following abdominal surgery |
| | Castration complication/scirrhous cord |
| | Biopsy (e.g. liver) |
| | Enterocentesis (e.g. during paracentesis) |
| | Caecal trocarisation |
| | Rectal tear (grade 3 or 4) |
| | Uterine perforation |

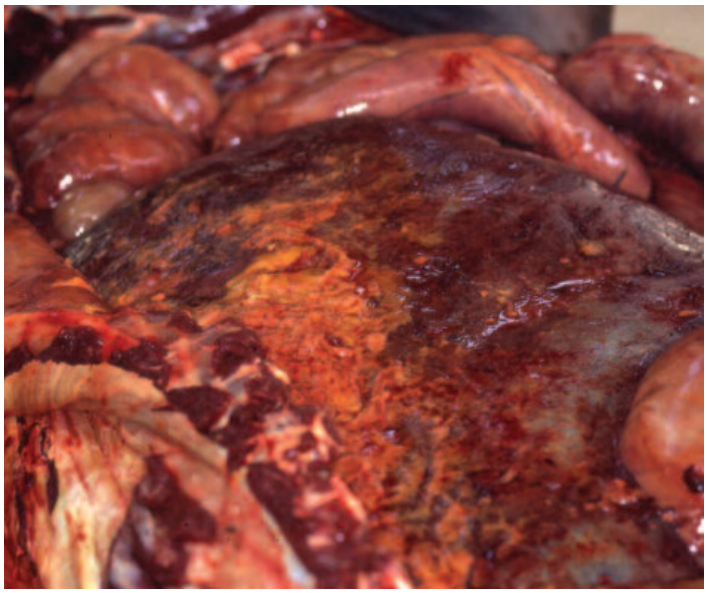

**Fig. 1:** *Diffuse fibrinous peritonitis - this image was taken at post mortem examination.*

onset and duration (peracute, acute or chronic), distribution (localised or diffuse) and bacterial involvement (septic or non-septic).

## Diagnosis

A full history and clinical examination are vital in both diagnosing peritonitis and ascertaining its cause. In a recent study of horses with peritonitis, a definitive cause was identified in only 23% of cases; idiopathic peritonitis was the most common diagnosis, as reflected in Case 2.

### *Clinical signs*

The clinical signs of peritonitis vary according to the primary disease process, onset and duration of the problem and the degree of peritoneal inflammation (Table 5). Dullness, depression and mild colic are non-specific signs, and were the main presenting signs for Cases 1, 2 and 4.

With generalised infection, the large shift of fluid and electrolytes from the vascular space into the peritoneal cavity can lead to severe dehydration and hypovolaemic shock. Septic peritonitis cases show the most severe clinical signs secondary to release of inflammatory mediators in response to bacterial toxins and, in the case of Gram -ve infections, due to the presence of endotoxins. These result in signs of endotoxaemia and shock may also be evident, as for Case 3.

### *Rectal examination*

Rectal examination may identify the underlying cause, e.g. neoplasia, abdominal abscessation or small intestinal strangulation. In periparturient mares, septic peritonitis secondary to uterine rupture or haemoperitoneum following arterial rupture should always be considered. In the case of periparturient haemorrhage, it may be days to weeks before clinical signs become apparent.

**TABLE 5: Clinical signs that may be associated with different forms of peritonitis**

| Peracute | Localised/Subacute/Chronic |
|---|---|
| (e.g. secondary to bowel rupture/rectal tear) | Chronic or intermittent colic |
| Severe toxaemia | Depression |
| Weakness | Anorexia |
| Depression or severe colic | Weight loss |
| Tachycardia | Intermittent fever |
| Tachypnoea | Ventral oedema |
| Circulatory failure | Exercise intolerance |
| Pyrexia (+/- depending on the degree of shock) | Decreased or absent intestinal sounds |
| Cold extremities | Mild dehydration |
| Sweating | Chronic diarrhoea (+/-) |
| Muscle fasciculation | Rectal examination may elicit pain on palpation of fibrinous or fibrous adhesions, ileus, intestinal impaction, masses (neoplasia or abscess); in most cases, however, there are no detectable rectal abnormalities |
| Weak peripheral pulses | |
| Red/purple mucous membranes | |
| Prolonged capillary refill time | |
| Decreased skin elasticity | |
| Parietal pain: reluctance to move, sensitivity to external abdominal palpation | |
| Ileus | |
| Painful urination/defecation | |
| Gritty feeling on serosal surfaces during rectal examination | |
| if extensive abdominal faecal contamination is present | |
| Collapse/death within several hours | |

**TABLE 6: Clinicopathological abnormalities vary depending on the time of onset and severity of peritonitis**

| Acute Septic Peritonitis | Chronic Peritonitis |
|---|---|
| Leukopenia | Leukocytosis |
| Haemoconcentration | Hyperfibrinogenaemia |
| Metabolic acidosis | +/- hyperproteinaemia with hyperglobulinaemia |
| Azotaemia | |
| Electrolyte imbalances | |
| +/- hypoproteinaemia | |

### *Clinicopathological abnormalities*

Clinicopathological abnormalities vary depending on the time of onset and severity of peritonitis (Table 6). Full haematology, biochemistry and peritoneal fluid analysis are indicated as part of the diagnostic work-up.

**Peritoneal fluid** is often increased in volume and may be grossly or microscopically abnormal (Fig. 2 and Table 7). A decrease in peritoneal fluid pH (<7.3) or glucose (<30 mg/dl) and an increase in fibrinogen concentration (>200 mg/dl) also suggest the presence of septic peritonitis.

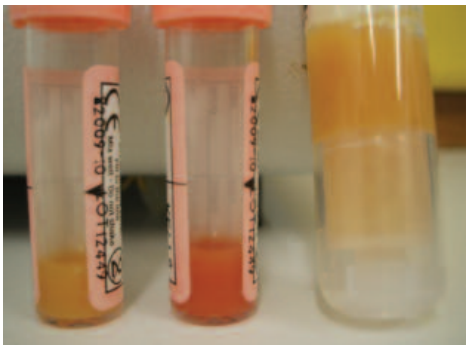

**Fig. 2:** *Abnormal peritoneal fluid.*

Culture of peritoneal fluid samples has a low sensitivity, only yielding growth in 9.5-32.5% of cases. In all three cases where performed, peritoneal fluid culture was negative. Positive cultures and cultures yielding more than one pathogen have been associated with a poor survival rate. The inability to culture bacteria in the presence of a Gram +ve stain is suggestive of an anaerobic infection. Often clinicians may choose not to culture samples but to provide broad-spectrum antibiotic cover.

### *Ultrasound*

Transabdominal and rectal ultrasound are a vital part of assessment for peritonitis, both in the diagnosis and to provide information about the aetiology. There is often a marked increase in the volume of peritoneal fluid, which may separate loops of small intestine and elevate them from the ventral abdominal floor (Fig. 3). A moderate increase in intestinal wall thickness, small intestinal distension and an increase or decrease in motility may be evident. Echogenicity of the peritoneal fluid increases with increasing cellular content; in chronic cases, fibrin may be seen floating freely within the peritoneal fluid and fibrin tags or abdominal adhesions may also be evident. Free gas in the abdominal cavity occurs in cases of bowel rupture or in the presence of gas-producing bacteria. Ultrasound examination can also identify the best place for collection of fluid and should be performed

**TABLE 7: Comparison of normal and abnormal peritoneal fluid**

| Parameter | Normal | Peritonitis |
|---|---|---|
| **Gross appearance** | Clear or slightly turbid, colourless to straw coloured | Thick, turbid, dark yellow to orange |
| **TP (g/l)** | <25 (usually <15) | >25 |
| **Total nucleated cell count (× 109/l)** | Adult: <5 (usually <2) | >10 |
| **Differential cell count (%)** | Neutrophils 20-90<br>Mononuclear cells/mesothelial cells 5-60<br>Lymphocytes 0-35<br>Eosinophils 0-5<br>Basophils 0-1 | High numbers of degenerate and non-degenerate neutrophils, intra- and extracellular bacteria<br>Plant material may be seen in cases of intestinal rupture<br>NB presence of plant material in the absence of other supporting clinical signs suggests inadvertent enterocentesis |

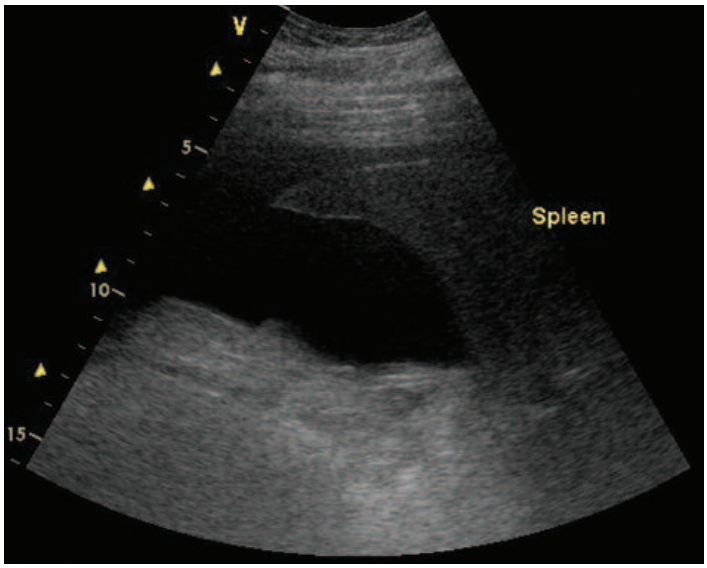


**Fig. 3:** *A marked increase in the volume of peritoneal fluid identified on ultrasound examination. (Image courtesy of Fernando Malalana MRCVS.)*

prior to paracentesis to reduce the risk of intestinal perforation when distended intestine has been palpated per rectum.

### *Laparoscopy/laparotomy*

Laparoscopy or laparotomy may aid both in detection and treatment of any underlying cause, especially as many cases of peritonitis are secondary to gastrointestinal insults. Laparoscopy is less invasive, but provides a limited view of the dorsal abdomen only and rarely facilitates treatment. Laparotomy requires a general anaesthetic but allows more comprehensive abdominal exploration and correction of most surgical intestinal lesions, plus intraoperative abdominal lavage or placement of drains for subsequent post-operative lavage. Peritonitis cases of unknown origin failing to respond to medical therapy warrant surgical exploration in order to obtain a more accurate diagnosis and prognosis, as in Case 4.

## Treatment

Treatment of peritonitis must be prompt and aggressive. Aims of treatment are to identify and resolve the underlying cause, minimise the inflammatory response and prevent long-term sequelae. This involves elimination of infection, restoration of fluid, protein, electrolyte and acid-base imbalances, reversal of endotoxaemia and ileus, control of pain and inflammation and nutritional support.

### *Fluid replacement*

When sequestration of fluid in the peritoneal cavity causes severe haemoconcentration and hypovolaemic shock, prompt restoration of circulating volume with crystalloids is required. In severe cases, hypoproteinaemia (plasma protein concentration <4.0 g/dl) may also be present, necessitating intravenous oncotic support in the form of colloids, such as 6% hydroxyethyl starch solutions (5-10 ml/kg q 24h), or hyperimmune plasma (4.4 ml/kg). Any electrolyte and acid/base imbalances should also be addressed.

### *Antimicrobial and anti-inflammatory therapy*

Antimicrobial therapy is critical in the management of septic peritonitis. In most cases, a mixture of Gram +ve and Gram -ve aerobes and anaerobes will be present, necessitating broad-spectrum antibiotic therapy. An appropriate therapeutic regime would consist of penicillin or ceftiofur, gentamicin and metronidazole. Antibiotics should be continued until the white blood cell count and abdominal fluid analysis are normal. Serum amyloid A (SAA) and fibrinogen can be monitored, with SAA showing a rapid return to normal following resolution of infection and fibrinogen showing a more delayed response.

Horses with peritonitis may also present with varying degrees of endotoxaemia. Treatment includes anti-inflammatory therapy (flunixin meglumine 0.25 mg/kg IV q 8h) and drugs designed to bind and inactivate endotoxin, including hyperimmune plasma and polymixin B.

### *Abdominal drainage*

Abdominal drainage may be beneficial, as it acts to remove excess fluid, bacteria, bacterial products, fibrin, blood and other foreign materials from the peritoneal cavity. In cases of diffuse peritonitis,

abdominal drainage is often combined with abdominal lavage, performed either surgically or via an in-dwelling ventral abdominal drain, placed under standing sedation and local anaesthesia. Abdominal drainage or lavage should not be attempted until any hypovolaemia has been corrected.

### *Additional treatments*

Additional treatment includes preventative measures for laminitis (frog supports, deep bedding, box rest) and appropriate anthelmintic treatment for cases with high faecal worm egg counts, presence of larvae in the faeces or poor de-worming history.

### Prognosis

Short-term survival rates vary from 53-86% and horses with idiopathic peritonitis survive for significantly longer periods than those with peritonitis of known aetiology. The prognosis is grave for horses with peritonitis secondary to gastrointestinal rupture and poor for abdominal neoplasia, abscesses, penetrating wounds to the body wall, urinary tract infection or obstruction and ileal hypertrophy. Septic peritonitis after abdominal surgery is also associated with high mortality. Peritonitis due to *Actinobacillus equuli* is generally associated with a very favourable prognosis after appropriate medical therapy. Long-term survivors may suffer from complications such as chronic recurrent colic, weight loss and pyrexia.

## CONCLUSION

Equine peritonitis is a potentially life-threatening disease that requires prompt diagnosis and aggressive treatment. Mortality rates can be as high as 59.7% and long-term sequelae, such as the formation of abdominal adhesions and internal abscesses, further reduce the survival rate. The prognosis is best in either idiopathic cases or where a treatable underlying cause is identified and early and aggressive treatment is instituted. Peritonitis should be considered in any horses displaying signs of abdominal pain and pyrexia.


FURTHER READING

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**CPD** CONTINUING PROFESSIONAL DEVELOPMENT SPONSORED BY BAYER ANIMAL HEALTH

*These multiple choice questions are based on the above text. Answers appear on page 59.*

**1. The most common cause of peritonitis in the UK is:**

a. Abdominal abscess due to *Strep. equi* var *equi*
b. Idiopathic
c. Larval cyathostominosis
d. Pancreatitis.

**2. Peritoneal fluid changes in cases of septic peritonitis include:**

a. Increased pH, decreased glucose, increased fibrinogen
b. Decreased pH, decreased glucose, increased fibrinogen
c. Increased pH, increased glucose, increased fibrinogen
d. Decreased pH, increased glucose, decreased fibrinogen

**3. Culture of peritoneal fluid in peritonitis cases may yield growth in up to:**

a. 33% of cases
b. 43% of cases
c. 53% of cases
d. 63% of cases.

**4. Haematology changes in acute peritonitis would include:**

a. Leukopaenia, anaemia, metabolic acidosis
b. Leukocytosis, haemoconcentration, metabolic acidosis
c. Leukopaenia, haemoconcentration, metabolic acidosis
d. Leukocytosis, anaemia, metabolic alkalosis