Septic peritonitis secondary to unilateral pyometra and ovarian bursal abscessation in a dog

A seven-year-old, female entire Labrador retriever was presented for acute-onset vomiting and lethargy, associated with weakness and generalised tremors. The clinical, radiographic, ultrasonographic and histopathological findings revealed septic peritonitis which occurred secondarily to unilateral pyometra and ovarian bursal abscessation. However, in this case, the initial clinical findings, blood parameters, radiographic and ultrasonographic findings did not allow a specific diagnosis. Repeat monitoring was required, and abdominocentesis proved to be the most useful diagnostic test, allowing a definitive diagnosis and the decision to be made as to whether or not to carry out exploratory surgery.

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Journal of Small Animal Practice (2002) **43**, 452–455

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INTRODUCTION

Early diagnosis of peritonitis is essential for successful treatment. Although septic peritonitis and pyometra are well known diseases in the dog, to the authors' knowledge there are no previous reports in the veterinary literature of septic peritonitis associated with unilateral pyometra and ovarian bursal abscess. This report describes the clinical, radiographic, ultrasonographic and histopathological findings in a case of septic peritonitis secondary to unilateral pyometra and ovarian bursal abscessation in a seven-year-old bitch. Management and treatment are also outlined.

CASE HISTORY

A seven-year-old, female entire Labrador retriever was presented for acute-onset vomiting and lethargy, associated with weakness and generalised tremors. The dog had no previous history of illness. Her last season ended two weeks prior to presentation.

On presentation, the dog appeared dull and depressed, and was obviously tachypnoeic (80 breaths per minute). Clinical examination revealed pink mucous membranes and a capillary refill time of less than two seconds. Reduced skin-fold compliance suggested moderate dehydration (8 to 10 per cent). There was pain on abdominal palpation, particularly of the cranial abdomen. The heart rate was 84 beats per minute and regular, the pulses were strong and no deficits were palpated. No vaginal swelling or discharge was noticed. Auscultation of the thorax revealed increased respiratory sounds associated with the tachypnoea. Rectal temperature was 38·5°C. Ophthalmological and neurological examinations were normal.

The dog was admitted to the authors' hospital for further investigations. After blood sampling (Table 1; day 0/T0 hours), intravenous fluid therapy (10 ml/kg/hour Hartmann's solution, Aqupharm 11; AnimalCare) and analgesic therapy (buprenorphine 0·01 mg/kg, Vetergesic; Alstoe) was initiated. Abnormal blood values included mild leucopenia ($5{\cdot}1 \times 10^9$/litre, thrombocytopenia (120×10^9/litre), and mildly increased amylase (36·0 µmol/litre). All other values were within normal limits. Thoracic radiographs were considered normal. Abdominal radiographs revealed the presence of faeces-filled large intestinal loops and a localised loss of radiographic contrast in the cranial abdomen. On abdominal ultrasonography, the liver, spleen and kidneys had a normal appearance, there was generalised intestinal ileus, and the pancreas could not be visualised. In the mid-abdomen a diffuse area of mottled hyperechogenicity without shadowing or reverberation was observed. It was thought to be inflamed omental fat. Distal to this area a fluid-filled, immobile tubular structure, 3 cm in diameter and 5 cm in length was visualised. The origin of this structure remained uncertain because it was not possible to establish a communication with the uterine horns distal to the bifurcation or with any intestinal loop. No free peritoneal fluid was observed.

On the basis of the clinical signs and elevated serum amylase, a provisional diagnosis of pancreatitis with associated focal steatitis and peritonitis was made. The tubular structure seen on ultrasound examination was thought to be dilated, fluid-filled intestine. Fluid therapy was continued

Table 1. Blood results on admission (day 0/T0 hours), 12 hours later (day 0/T12 hours) and at one (day 1) and three days (day 3) postoperatively

	Day 0/ T0 hours	Day 0/ T12 hours	Day 1	Day 3	Reference range
Red blood cells	6·97	6·41	5·25	5·99	5·50-8·50 $\times 10^{12}$/litre
Packed cell volume	0·47	0·43	0·37	0·40	0·39-0·55 litre/litre
Haemoglobin	16·9	15·4	13·4	14·9	12·0-18·0 g/dl
MCV	67·2	67·1	66·4	66·9	60·0-77·0 fl
MCHC	36·1	35·8	36·5	37·2	32·0-36·0%
Total WBC count	5·10	10·3	19·5	12·6	6·00-15·00 $\times 10^9$/litre
Neutrophils segmented	3·72	7·83	17·16	8·32	3·60-12·00 $\times 10^9$/litre
Neutrophils meta	0·00	0·41	0·00	0·25	0·00-0·00 $\times 10^9$/litre
Lymphocytes	0·97	1·24	0·78	2·14	0·70-4·80 $\times 10^9$/litre
Monocytes	0·26	0·83	0·39	1·39	0·00-1·50 $\times 10^9$/litre
Eosinophils	0·15	0·00	1·17	0·50	0·00-1·00 $\times 10^9$/litre
Basophils	0·00	0·00	0·00	0·00	0·00-0·20 $\times 10^9$/litre
Platelets	120	304			200-500 $\times 10^9$/litre
Total protein	61·9		46·6	58·8	58·0-73·0 g/litre
Albumin	30·8		19·2	21·7	26·0-35·0 g/litre
Globulin	31·1		27·4	37·1	18·0-37·0 g/litre
Bile acids	0·1				0·0-7·0 µmol/litre
Creatinine	86·0			105·0	0·0-106·0 µmol/litre
Glucose	5·0		5·80		3·0-5·0 mmol/litre
Urea	4·6			6·1	1·7-7·4 mmol/litre
AP	17·0				20·0-60·0 IU/litre
ALT	43·0				15·0-60·0 IU/litre
Amylase	36·0	15·8			15·0-28·0 µmol/litre
Lipase	51·0	122·0			13·0-200·0 IU/litre
Calcium	2·5				2·3-3·0 mmol/litre
Inorganic phosphate	1·1				0·9-2·0 mmol/litre
Sodium	143·0	142·0	142·0	145	139·0-154·0 mmol/litre
Potassium	4·0	3·5	3·5	4·6	3·6-5·6 mmol/litre
Chloride			123·5		3·48-5·8 mmol/litre
Thyroxine	40·3				15·0-48·0 µmol/litre

MCV Mean corpuscular volume, MCHC Mean corpuscular haemoglobin concentration, WBC White blood cell, AP Alkaline phosphatase, ALT Alanine aminotransferase

(Hartmann's solution, 10 ml/kg/hour), vital signs and blood parameters were monitored (Table 1), and all oral intake was withheld. Despite an initial improvement, the dog became more depressed and tachypnoeic. All vital signs remained within normal limits but the abdominal pain appeared to be more generalised. Repeat haematology (Table 1; day 0/T12 hours) showed a normal total neutrophil count ($10·3 \times 10^9$/litre) with a mild left shift ($0·41 \times 10^9$/litre). Serum amylase and lipase were within normal limits. Repeat abdominal radiographs revealed a generalised loss of contrast with a small intestinal sentinel loop suggestive of paralytic ileus (Fig 1). On repeat abdominal ultrasonography, a moderate amount of free peritoneal fluid was seen. Ultrasound-guided abdominocentesis was performed. The aspirated fluid was brown and turbid. Cytological examination (Diff Quik and Gram's stain) revealed the presence of many degenerate and toxic neutrophils. Gram-positive cocci in chains were observed both within neutrophils and extracellularly. The diagnosis of septic peritonitis was made and the dog was immediately referred for surgery. Abdominocentesis fluid was submitted for aerobic and anaerobic bacterial culture and sensitivity testing.

The bitch was anaesthetised by premedication with acetylpromazine 0·025 mg/kg intramuscularly (IM) (ACP; Arnolds) and morphine sulphate 0·35 mg/kg IM (Morphine; Medeva), induction with propofol 4 mg/kg intravenously (IV) (Propofol; Schering-Plough) and maintenance with isoflurane (Isoflo; Merial) and oxygen. A ventral midline coeliotomy from xyphoid to pubis was performed. A single intravenous dose of 1 mg/kg flunixin meglumine (Finadyne; Schering-Plough) was administered before induction. Antibiotic therapy comprising cefotaxime 30 mg/kg IV (Claforan; Roche), ampicillin 20 mg/kg IV (Penbritin; SmithKline Beecham) and gentamicin 6 mg/kg subcutaneously (Genticin; Roche) was also administered before induction.

A large amount of free haemorrhagic, purulent peritoneal fluid was present. The peritoneum was inflamed. All abdominal organs were normal except for the left ovary and left uterine horn. The left ovary was surrounded by inflamed omental fat and the ovarian bursa was fluid-filled; there were two separate fluid-filled segmental dilations in the left uterine horn. The right uterine horn and right ovary appeared normal on macroscopic examination (Fig 2). An ovariohysterectomy was performed. The abdomen was lavaged with 8 litres (250 ml/kg) of warmed sterile saline. The wound was closed routinely with 1 Prolene (Ethicon) simple continuous suture of the linea alba, 3-0 Monocryl (Ethicon) simple continuous suture subcutaneously and 4-0 Monocryl continuous horizontal mattress suture intradermally.

Postoperatively, intravenous fluid therapy with Hartmann's solution was administered at 3 ml/kg/hour with supplemental potassium (KCl 1·5 per cent; Martindale). Antibiotic therapy was continued with cefotaxime 30 mg/kg IV three times daily, ampicillin 20 mg/kg IV three times daily and gentamicin 6 mg/kg subcutaneously once daily. Analgesia was provided with morphine 0·2 mg/kg IV every four hours initially and later with buprenorphine 0·01 mg/kg IM three times daily. All vital signs, including urine output, remained within normal limits. The dog was discharged five days after surgery with a three-week course of oral amoxycillin/ clavulanate acid 15 mg/kg twice daily (Synulox; Pfizer) and 10 mg/kg metronidazole (Metronidazole; Cox Pharmaceuticals) twice daily. The dog made a full recovery.

Culture of the peritoneal fluid yielded a heavy pure growth of *Streptococcus canis* which was sensitive to a variety of antimicrobials. The uterus and ovaries were submitted for histopathology. On macroscopic examination, the left uterine horn was dilated and sacculated along its entire length. The mucosa was thickened and corrugated in appearance. Several soft multilobular masses were associated with the proximal uterine wall close to the left ovary. The right uterine horn was of normal diameter along its entire length. No obstruction was obvious at the base of the left horn. There was no evidence of rupture of the uterine wall. The ovaries contained multiple corpora lutea. Microscopic examination showed that both uterine horns had a moderate degree of cystic endometrial hyperplasia in association with varying degrees of metritis. The left uterine horn had a florid endometritis with a mixed, but predominantly mononuclear, cellular infiltrate. In some areas the inflammation extended through all layers of the

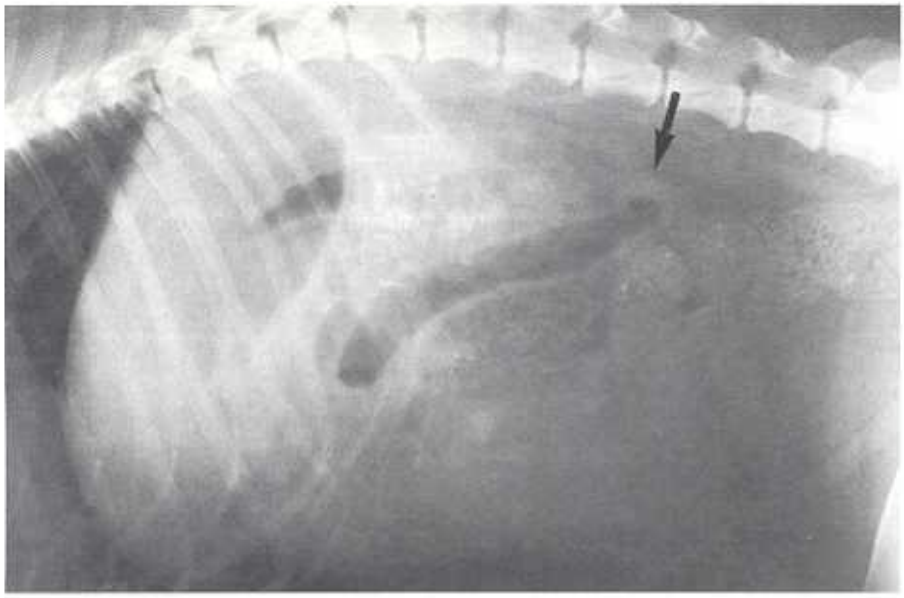

FIG 1. Right lateral abdominal radiograph, obtained 12 hours after admission, showing generalised loss of contrast and a small intestinal sentinel loop (arrow)

uterine wall, particularly in association with the masses at the proximal end. The masses were composed of necrotic adipose tissue with focal areas of degenerate polymorphonuclear cells. The right uterine horn had similar changes confined to the endometrium.

DISCUSSION

Generalised peritonitis, a diffuse inflammation of the peritoneal cavity, can be primary or secondary (Hosgood and Salisbury 1988). In this case, the peritonitis was apparently secondary to bacterial infection of the reproductive tract, but the route of peritoneal contamination is unclear. Since there was no evidence of rupture of the uterine wall, the periuterine abscessation and peritonitis might have arisen in one of two ways. Extension of infection through the uterine wall from infection in the lumen is one possibility. There was histopathological evidence of inflammation tracking through the uterine wall although there was no evidence of uterine rupture. Another possibility is retrograde spread of infection via the oviduct into the ovarian bursa and surrounding tissue. There was bursal abscessation and there is direct communication between the ovarian bursa and peritoneal cavity during ovulation. In adolescent girls there is a condition (so-called cocoon syndrome) where retrograde menstruation has been thought to lead to sclerosing encapsulating peritonitis (Foo and others 1978).

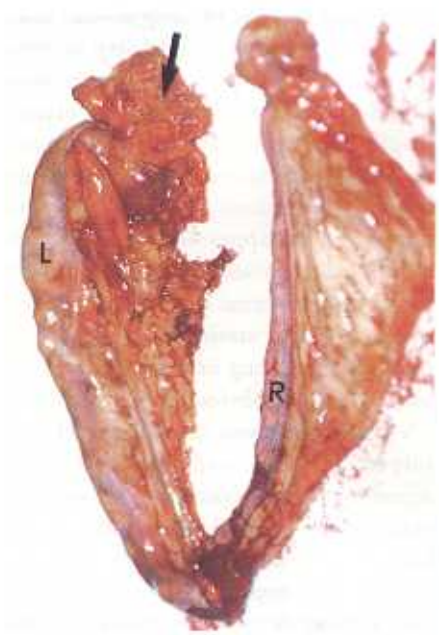

FIG 2. Resected reproductive tract showing the sacculated left uterine horn (L) and several soft multilobular masses of necrotic adipose tissue surrounding the left ovary (arrow). The right uterine horn (R) and ovarian bursa are normal

The reason for segmental left uterine horn pyometra rather than the more usual bilateral uterine horn pyometra remains unclear. There was no gross or histological evidence of obstruction at the base of the left uterine horn. Pyometra most frequently occurs two months postoestrus (Wheaton and others 1989); the early development of pyometra postoestrus, as seen here, is unusual. The most common source of bacteria in pyometra is faecal contamination of the genitourinary tract (Barsanti 1998). Given that the disease occurred so shortly after the end of the bitch's season, bacterial contamination during copulation, and an ascending vaginal infection in early pregnancy, cannot be excluded as a cause of the segmental infection.

Ruptured pyometra is an uncommon, but documented, cause of septic peritonitis in dogs and cats. However, to the authors' knowledge, there are no previous reports in the veterinary literature of unilateral pyometra associated with ovarian bursal abscessation and septic peritonitis.

Early diagnosis of peritonitis is essential for successful surgical treatment (Lunggren and Olsson 1972). Diagnosis was slightly delayed in this case because the initial clinical, radiographic and ultrasonographic signs were consistent with pancreatitis. The history and clinical signs in peritonitis are often vague and non-specific (Swann and Hughes 2000). Vomiting, depression and cranial abdominal pain are common findings in peritonitis, but are also seen in pancreatitis. Normothermia during hospitalisation, as in this case, is unusual in septic peritonitis (King 1994). Mild haematological changes such as those seen here (initial leucopenia and a mild left shift without generalised neutrophilia 12 hours post-admission), although uncommon, have been previously reported in generalised peritonitis (King 1994). An initial mild thrombocytopenia was observed in this case in contrast to the profound fall in platelets seen by Sugerman and others (1982) in dogs with septic peritonitis. There was no hypoalbuminaemia (Hardie 1989) and no significant elevations in serum alkaline phosphatase in this case in contrast to dogs with sepsis (Sugerman and others 1982) and animals with pyometra (Wheaton and others 1989). Elevated serum alkaline phosphatase is presumed to be secondary to intrahepatic cholestasis. Serum concentrations of glucose in this case were normal, in contrast to the transient hyperglycaemia followed by profound hypoglycaemia seen in most models of canine sepsis and endotoxaemia (Hardie and others 1985). The prevention of severe hypotension and shock have previously been shown to prevent hypoglycaemia in canine sepsis (Hardie and others 1985). In this case, all vital signs including mucous membrane colour, capillary refill time, heart rate, pulse quality and urine output remained within normal limits, making the presence of septic shock unlikely. If

shock had developed, hypotension, peripheral vasoconstriction and hypothermia would have been expected (Hardie 1989).

In this case, radiographic and ultrasonographic findings were consistent with peritonitis, but the decision to perform surgical exploration was confirmed by cytological examination of peritoneal fluid.

The treatment of peritonitis includes supportive care of the patient, correction of the inciting cause, drainage of the exudative material and specific antimicrobial therapy based on culture and sensitivity testing. Patient support may include fluid therapy to restore the haemodynamic status of the animal, support of plasma protein levels, support of serum glucose and electrolyte levels and nutritional support (Kirby 2002). Correction of the inciting cause of peritonitis involves thorough abdominal exploration and may require additional surgical procedures such as intestinal resection and anastomosis, mass removal, closure of a ruptured viscus or ovariohysterectomy, as performed here (Winkler and Greenfield 2000). There are no specific criteria reported to assist in case selection for primary coeliotomy closure versus open peritoneal drainage. The authors opted for primary coeliotomy closure after resection of the abscessed reproductive tract and copious peritoneal lavage. The abdomen was lavaged with warmed sterile saline. Well controlled experimental studies have shown no benefit to including antibiotics in the lavage fluid when parenteral antibiotics are administered (Schein and others 1988). The choice of antibiotics in the present case was empirical, and may be overwhelming. Cefotaxime was chosen for its broad-spectrum activity and ampicillin for its Gram-positive and anaerobic spectrum (with the exception of *Bacteroides* species). Gentamicin is effective against many resistant Gram-negative bacteria, but with the risk of dose-dependent nephrotoxicity and ototoxicity. Gentamicin was given in larger, less frequent doses (6 mg/kg once daily) to reduce this risk (Campbell and Rosin 1992). In this case, serum creatinine concentrations increased slightly but remained within normal limits; serum urea concentrations also remained within normal limits. Urine output was sufficient at all times.

Flunixin meglumine was administered as a single preoperative dose for its proven efficacy, without renal or gastrointestinal toxicity, in the treatment of acute lethal *Escherichia coli* peritonitis (Hardie and others 1985). Flunixin meglumine blocks the production of thromboxane and prostacyclin, which are important mediators in the inflammatory cascade (Hardie and others 1985). To the authors' knowledge, flunixin meglumine is the only non-steroidal anti-inflammatory drug (NSAID) with proven efficacy in septic peritonitis. Other NSAIDs may be equally efficacious, but any specific drug recommendation awaits experimental or clinical trials. The use of corticosteroids in sepsis remains controversial (Kirby 2002).

Once the results of bacterial culture were available, the antibiotic regimen was changed. Clavulanate-potentiated amoxycillin was selected for the treatment of *S canis*. Metronidazole was administered for anaerobic bacteria, although no anaerobes were isolated. The recovery of anaerobic bacteria in cases of septic peritonitis in humans is related to both initial sample handling and the rigours of anaerobic culture techniques (Laroche and Harding 1998).

Conclusions

Unilateral pyometra and ovarian bursal abscessation should not be overlooked as a possible cause of septic peritonitis. In the present case, initial clinical findings, blood parameters, radiographic and ultrasonographic findings did not allow a specific diagnosis. Repeat monitoring was required, and abdominocentesis proved to be the most useful diagnostic test, allowing both a definitive diagnosis and a decision to be made as to whether or not to carry out exploratory surgery.

Acknowledgements

The authors thank Mr M. Camburn for the anaesthetic management of this animal and C. Scudamore and L. Newman from, respectively, the Pathology and Microbiology Departments of the Royal (Dick) School of Veterinary Studies for diagnostic services.

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