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CASE REPORT
Priapism in a castrated cat associated with feline infectious peritonitis

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This report describes a case of feline infectious peritonitis (FIP) in a castrated cat which first presented with the unusual sign of priapism. Laboratory examinations showed increased serum protein content and decreased albumin/globulin ratio. Serum electrophoresis revealed increased α_2- and γ-globulin content. One month after the first examination, the cat died. At necropsy, histopathological evaluation of organs showed inflammatory granulomatous lesions compatible with non-effusive FIP and coronavirus-specific polymerase chain reaction confirmed the diagnosis. FIP antigen was demonstrated immunohistochemically in penile tissue.

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Priapism is defined as persistent penile erection in the absence of sexual stimulation. This erectile disorder is uncommon in domestic animals and results from the engorgement of the corpus cavernosum while the corpus spongiosum is less turgid than in a normal erection (Rochat 2001). Erectile tissue engorgement can either be the consequence of an increase in arterial blood flow or of a reduction in venous drainage (Bivalacqua and Burnett 2006). The aetiologies of this condition are various. Arterial overflow of the penis occurs when a traumatic event produces an arterial–cavernosal shunt; in man, venous outflow occlusion can follow haematological disorders, prostatic diseases, spinal cord trauma, use of pharmacological agents such as heparin and phenothiazines, or it can be idiopathic (Rochat 2001). In the veterinary literature other reported causes of priapism are spinal cord lesions associated with distemper, complications of castration and lower urinary tract diseases (Rochat 2001).

Permanent damage to erectile tissue is a likely consequence of priapism, with thrombosis of the cavernous spaces, endothelial and smooth muscle cell destruction and eventually corporal fibrosis following vascular stasis and corporal ischaemia (Bivalacqua and Burnett 2006).

The cases reported in the cat have been a consequence of castration (Orima et al 1989, Swalec and Smeak 1989), with funiculitis or urethritis as likely initiating factors (Swalec and Smeak 1989), or the effect of attempted mating, so that a traumatic origin can be hypothesised (Gunn-Moore et al 1995); vasculitis following suspected feline infectious peritonitis (FIP) was reported in a cat (Gunn-Moore et al 1995). In no cases did the condition respond to the attempted conservative treatments (penile lubrication and massage, corticosteroids, antibiotics and diuretics) and it was resolved surgically either with perineal urethrotomy (Swalec and Smeak 1989, Gunn-Moore et al 1995) or with corporal drainage (Orima et al 1989).

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A 2-year-old castrated male mixed breed cat was referred to the Veterinary Teaching Hospital of The University of Turin because of priapism of 2 weeks' duration, associated with reduction of activity and reduced appetite. In the months before presentation, the cat had shown some episodes of fever (39.5–40°C) that had subsided after a short course of antibiotics (long lasting amoxycillin, 15 mg/kg q 48 h). At 3 months of age, the cat had been tested for both feline immunodeficiency virus (FIV) antibody and feline leukaemia virus (FeLV) antigen (SNAP, IDEXX Lab, Westbrook, MA, USA) and had resulted negative; a trivalent and FeLV vaccine had then been administered twice, followed by annual revaccination.

At time of presentation the cat appeared in rather poor condition, with ruffled coat and mild ocular discharge. Physical examination was unremarkable, apart from the presence of an engrossed penis, of fibrous consistency, which was moderately painful at palpation. Abdominal ultrasonography did not show any abnormality. Unfortunately, it was not possible to collect and analyse urine, however, the cat did not show any signs of urinary tract inflammation/infection and urination occurred without effort.

Red blood cell count was normal at 8.9×10^{12}/l (reference interval: $5–10 \times 10^{12}$/l) and packed cell volume (PCV) was marginally elevated at 0.47 l/l (reference: 0.24–0.45 l/l), while white blood cell count was normal at 5.5×10^{6}/l (reference: $5.5–19.5 \times 10^{6}$/l). Serum biochemistry parameters (aspartate aminotransferase, alanine aminotransferase, creatine kinase, γ-glutamyltransferase, creatinine, urea, glucose, total bilirubin, triglycerides, total cholesterol, alkaline phosphatase, amylase and lipase) were within normal limits. Total serum protein content was increased (82 g/l, reference: 60–75 g/l), albumin was 27 g/l (reference: 23–39 g/l) and globulin was 55 g/l (reference: 22–50 g/l): as a consequence the albumin/globulin ratio was decreased to 0.49 (reference: 0.6–1.2). Serum protein electrophoresis revealed also increased α_2- and γ-globulin content (13.4 g/l and 29.4 g/l, with reference: 4.3–12.6; and 5.1–10.3, respectively). Serology for FIV, FeLV and *Toxoplasma gondii* (IgG and IgM) was negative.

FIP was suspected and the owner did not consent to further investigations. Surgical intervention for the treatment of priapism was not considered an option at the time. A palliative therapy with prednisone (2 mg/kg BID) was administered, in association with amoxycillin–clavulanic acid (15 mg/kg BID); penile lubrication was prescribed.

One month after the examination, the cat died.

Necropsy showed a moderate to severe serofibrinous chronic pleuritis and peritonitis. The macroscopic lesions of systemic organs were indicative of non-effusive FIP. Some nodular whitish lesions, from 0.3 to 1.5 cm in diameter, were visible on the cut surface of mesenteric and mediastinal lymph nodes, lung, liver, pancreas and kidney. The same nodular lesions were present on the submucosa of the penis.

Histopathological examination revealed a moderate thoracic and abdominal fibrinous serositis. A moderate degree of lymphoid depletion in both spleen and lymph nodes was present. Several perivascular lymphocytic infiltrates were visible in the pancreas, liver, lung and kidney, associated with multifocal necrotic areas and pyogranulomatous foci of inflammation. Severe and diffuse pyogranulomatous inflammation with some minimal necrotic areas infiltrated also the submucosa of the penis, associated with fibrinoid necrosis of the corpus cavernosus (Fig 1a and b).

Formalin-fixed, paraffin wax-embedded tissue samples from intestine, lymph nodes, spleen, liver, pancreas, kidney, lung and penis were tested for coronavirus antigen by immunohistochemistry that was performed using the Avidin–Biotin-Complex (ABC) technique with a commercially available kit (Vectastain Elite, Vector Laboratories, CA, USA), and using a monoclonal antibody against the feline coronavirus (FcoV) (kindly provided by Prof N.C. Pedersen, Davis, CA, USA) as previously described (Hsu et al 1981). Positive labelling for coronavirus was found in necrotic-proliferative foci of pancreas and kidney, in subpleural foci, and in the pyogranulomatous lesions of the penis (Fig 1c and d).

FIP is a fatal coronavirus-induced systemic disease in cats. The causative agent is a mutant FCoV generated by a mutation of the widespread enteric strain that gains the ability to replicate in macrophages and spreads through infected monocytes (Paltrinieri et al 2001). The cell-mediated immune response can be strong enough to eliminate the virus; weak cellular immunity can slow viral replication, resulting in the non-effusive FIP, characterised by proliferative inflammatory lesions. Effusive FIP develops in the absence of cell-mediated immunity, when antigen–antibody complex deposition and the release of vasoactive amines following complement fixation lead to vasculitis and plasma protein exudation (Andrew 2000).

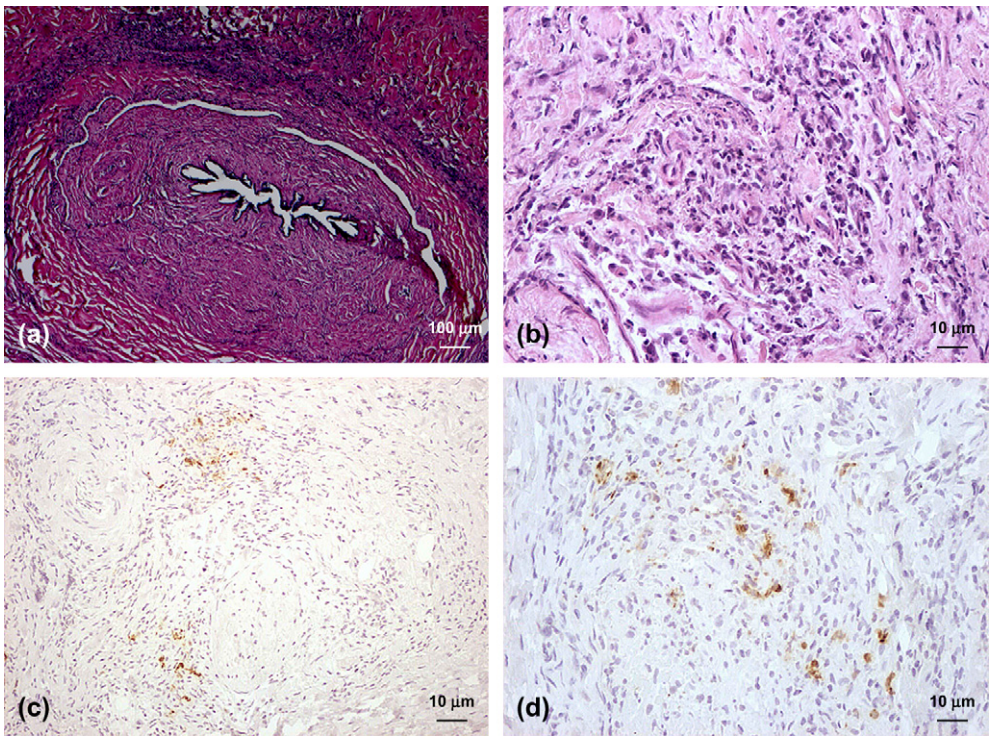

Fig 1. Cat penis. (a) Moderate to severe pyogranulomatous infiltrate surrounding the urethra. Haematoxylin and eosin (HE) stain. Original magnification, 20×. (b) Moderate to severe pyogranulomatous infiltrate in the submucosal layer of the penis. HE stain. Original magnification, 600×. (c) FCoV antigen expressed within necrotic-proliferative foci detectable in the penis. Immunohistochemistry, anti-FCoV antibody, ABC method; chromogen = diaminobenzidine (DAB); counterstain: Mayer's haematoxylin. Original magnification, 250×. (d) Higher magnification of a pyogranulomatous lesion in which viral antigen is immunohistochemically detectable within macrophages and, to a lesser extent, as a granular positivity scattered among cellular infiltrates. Immunohistochemistry, anti-FCoV antibody, ABC method; chromogen: DAB; counterstain: Mayer's haematoxylin. Original magnification, 400×.

In the case presented here, the first clinical signs were mild and the appearance of priapism was misleading. The ultrasound examination did not reveal any granulomatous lesions in the abdominal organs, and the serum biochemistry panel revealed no sign of dysfunction or organ damage. The ante-mortem presumptive diagnosis was based on the haematological and serum protein profiles that revealed a non-specific inflammatory pattern (Sparkes et al 1991); the execution of other tests, such as serum FCoV antibody titration or the measurement of α_1-acid glycoprotein, would have increased the likelihood of a correct diagnosis, though, in any case, only the detection of viral antigen in the histological lesions can confirm it (Addie et al 2004).

The involvement of the male genital tract in FCoV infection has previously been described as scrotal enlargement following abdominal effusion (Andrew 2000) or as orchitis (Sigurðardóttir et al 2001). The presumptive diagnosis of FIP in previous case of priapism in a systemically ill cat (Gunn-Moore et al 1995) could not be confirmed.

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