Defaecation syncope and pulmonary thromboembolism in a cat

NT WHITLEY[ab] and RL STEPIEN[a]

A 7-year-old male neutered domestic shorthair cat was referred for worsening gastrointestinal and haematologic abnormalities. Physical status deteriorated further despite intravenous crystalloids, blood transfusion and nutritional support. Cardiorespiratory signs developed and the cat died suddenly while straining to defaecate. Diffuse thrombosis, pulmonary thromboembolism, metastatic pancreatic carcinoma and histologic evidence of cardiomyopathy were present at necropsy. This is the first reported case of feline pulmonary thromboembolism associated with defaecation syncope. Predisposing factors for thrombotic disease in this case and aspects of human defaecation syncope are discussed. The risk of clot dislodgement by the Valsalva manoeuvre in patients with a thrombotic tendency is highlighted.

Aust Vet J 2001;79:403-405

Key words: Defaecation syncope, pulmonary thromboembolism, cat, DIC, pancreatic carcinoma.

APTT	Activated partial thromboplastin time
DIC	Disseminated intravascular coagulation
FDP	Fibrin degradation products
OSPT	One stage prothrombin time
PCV	Packed cell volume
PTE	Pulmonary thromboembolism
q 12 h	Every 12 hours
SC	Subcutaneously
TT	Thrombin time

Case report

A 7-year-old male neutered domestic shorthair cat (6.3 kg) was referred for evaluation of multiple problems. Profound weight loss, constipation, intermittent vomiting and lethargy had occurred over the previous 3 months. Radiographs following oral barium administration were reported to show delayed gastric emptying. Therapy with lactulose, multiple enemas (containing sodium citrate, sodium lauryl sulfoacetate and sorbitol), cisapride and glucocorticoids had been employed. At the time of referral, lactulose and cisapride had not been given for 2 weeks, and glucocorticoids had not been given for 2 days. During the days prior to referral, the vomiting had worsened, and abdominal pain, declining haematocrit and ecchymotic haemorrhages were reported. Feline leukaemia and feline immunodeficiency virus tests (whole blood ELISA tests, Idexx Laboratories Inc.) were negative.

On emergency referral, abnormal physical examination findings included extreme lethargy, tacky mucous membranes, with multiple ecchymotic haemorrhages, bruising at previous venipuncture sites, weak pulses, dehydration and apparent abdominal pain on palpation. Serum biochemistry revealed reductions in sodium (131 mmol/L, normal 148-157), chloride (104 mmol/L, normal 114-126) and albumin (26 g/L, normal 28-39) and increases in urea (12.9 mmol/L normal 5-12), glucose (16.8 mmol/L, normal 3.7-6.9), creatine kinase (666 U/L, normal 11-315) and alanine aminotransferase (318 U/L, normal 17-138). Haematology showed anaemia (PCV 16 L/L), thrombocytopaenia (41 x 10^9/L) with moderately enlarged platelets, and neutrophilia (20.8x10^9/L) with band forms present (0.9x10^9/L). Red cell morphology was characterised by mild anisocytosis and polychromasia, moderate poikilocytosis, and occasional schistocytes. Mean cell volume was normal and a reticulocyte count not available. A voided urine sample had a specific gravity of 1.045 and contained glucose (>111 mmol/L) and blood (2+). A coagulation profile showed prolongation in OSPT (19.7 sec), APTT (>110 sec) and TT (38.7 sec), and a low fibrinogen (0.37 g/L) compared with control values obtained from pooled plasma from healthy cats (12.3 sec, 11.4 sec, 21.1 sec and 0.99 g/L respectively). FDP concentration was normal (<10 mg/mL). An additional study was performed, in which equal volumes of plasma from the patient and a healthy control cat were mixed. The same coagulation assays were run on this mixed sample and results then compared with those obtained from the pooled control sample from normal cats. Failure of the APTT to correct completely with this manipulation suggested that presence of an endogenous coagulation inhibitor substance in the patient serum was more likely than an absolute factor deficiency. (APTT for mixed patient/healthy control sample 66.7 APTT for pooled control sample:16.4 sec).

Thoracic radiographs taken upon admission demonstrated obesity and caudal deviation of the gastric axis, but a normal intrathoracic viscera. Abdominal ultrasound revealed hepatic rounding, hyperechogenicity of the hepatic parenchyma, and a large volume of echogenic peritoneal fluid. The ultrasonographer found the intestinal tract difficult to evaluate and reported that it was all clumped in the centre of the abdomen. However, one bowel loop was isolated and measured and was within normal limits. Initial emergency therapy consisted of blood transfusion (50 mL of whole blood, matched for blood type), intravenous lactated ringers solution (25 mL/h), and administration of Vitamin K (18 mg SC q 12 h). Following the blood transfusion, abdominocentesis was performed, yielding a modified transudate with evidence of inflammation (characterised by an increased protein content and an increased number of mesothelial cells). A tentative diagnosis of chronic pancreatitis, complicated by abdominal effusion, coagulopathy and hepatopathy was made. Other differential diagnoses considered included abdominal neoplasia, hypoadrenocorticism, and partial gastrointestinal obstruction caused by neoplasia or a foreign body.

On day 2 of hospitalisation, PCV was 26 L/L and body weight 6.5 kg. Mild hypercholesterolaemia (5.5 mmol/L, normal 1.7-5.0) and hyperglycaemia (8.4 mmol/L, normal 3.7-6.9) were

[a]Department of Medical Sciences, Veterinary Medical Teaching Hospital, University of Wisconsin-Madison, 2015 Linden Drive West, Madison, WI 53706, USA

[b]Current address: Department of Veterinary Clinical Sciences, Evelyn Williams Building (B10), The University of Sydney, New South Wales 2006

present. A voided urine sample still contained blood but was negative for glucose. A cardiac gallop rhythm was detected on auscultation. The cat ate small amounts of food, after intravenous diazepam administration (0.5 mg/kg).

On day 3 PCV was 18 L/L and body weight 7 kg. The abdominal effusion was more pronounced and jugular distension was now present. Heart rate was 180/minute and femoral pulse strength was poor. Syringe feeding was commenced and the cat passed firm faecal pellets following administration of a chlorhexidine solution enema. A limited echocardiographic examination was performed. Abnormal echocardiographic findings included marked right atrial and right ventricular dilation, mild tricuspid regurgitation, thickening of the tricuspid valve and apparent hypercontractility (left ventricular shortening fraction 54%). These findings were interpreted as compatible with acute onset of right-sided congestive heart failure. The intravenous fluid rate was reduced to 12 mL/h and frusemide therapy was instituted at 1 mg/kg SC q 12 h.

On day 4 of hospitalisation, the cat's heart rate was 200/min. Therapeutic abdominocentesis removed 200 mL of fluid. Rectal temperature was 39.4°C. Nasal congestion and sneezing had developed but the cat ate very small amounts of food. Thoracic radiographs revealed mild generalised cardiomegaly, a small pleural effusion and enlarged pulmonary arteries and veins.

By the fifth day of hospitalisation the cardiac gallop rhythm was still present intermittently. Thoracic radiographs revealed worsening pleural effusion. Hypothermia developed (37°C). A nasogastric feeding tube was placed and feeding instituted. PCV was 21 L/L. Thrombocytopenia, hypofibrinogenaemia (0.70 g/L, control 1.04) and elevated FDP concentrations (>10 and < 40 mg/mL) suggested DIC; 25 mL of fresh frozen plasma was administered intravenously. Several hours later, the cat was observed to be straining to defaecate in its litter tray, then vocalised, became ataxic, cyanotic, collapsed and subsequently experienced cardiac arrest. Resuscitation attempts were not made.

Necropsy examination showed an adherent thrombus on the tricuspid valve apparatus. Another large thrombus occupied the lumen of one of the main pulmonary arteries. In addition, histopathological examination showed subacute, severe thrombosis of the portal vein, smaller pulmonary arteries and multiple small vessels in the myocardium and adrenal glands. Pathological cardiac findings included myofibres that were fragmented or contained vacuolated cytoplasm, and were separated by a clear space, sometimes containing loose fibrous connective tissue. These findings suggested cardiomyopathy. Multifocal, necrotising pancreatitis and pancreatic carcinoma were present, with extension of the neoplasm into the duodenum. Carcinoma metastases were present in the liver, lymph nodes and lung, and abdominal carcinomatosis was present.

Discussion

A recent retrospective study on feline PTE suggested possible predisposing conditions in all cases.[1] These authors also described clinical features and radiographic markers of PTE. A comprehensive list of the principle causes of PTE has been published by other authors.[2] In the case reported here, several factors may have been implicated in the genesis of the PTE. These include obesity, recumbency, glucocorticoid therapy, transient diabetes mellitus, the indwelling intravenous catheter, cardiac disease, pancreatitis, pancreatic carcinoma and DIC. It is probable that metastatic pancreatic carcinoma was the dominant pathological process responsible for altered coagulation in this patient. Several mechanisms have been suggested for inappropriate coagulation in cancer patients, including production of procoagulant proteins by tumour cells and monocytes, and elaboration of platelet proaggregatory substances by tumour cells.[3]

This cat's sudden death may have been caused by a cardiac arrhythmia triggered by the structural cardiac disease documented at necropsy, but the temporal association between attempted defaecation and cardiac arrest is compatible with defaecation-associated syncope. In people, defaecation syncope is classified as a situational syncope in the broader grouping of reflex-mediated vasomotor instability syndromes. Other examples of situational syncope identified in people include micturition-, cough- and swallowing-related syncope. Neural impulses from gut wall tension receptors are thought to be transmitted via vagal afferents to the CNS, resulting in hypotension and bradycardia.[4] The history of constipation in this cat and the straining observed prior to collapse may have caused significant activation of gut wall tension receptors, resulting in sudden hypotension and bradycardia.

Defaecation syncope associated with pulmonary embolism has also been documented in humans.[5-7] Increases in intrathoracic and intra-abdominal pressure during defaecation tend to reduce venous return and hence reduce cardiac output and peripheral blood flow (the Valsalva manoeuvre). Release of the Valsalva manoeuvre on completion of defecation and the attendant sudden increase in cardiac output and the 'vacuum effect' caused by the sudden reduction in intra-abdominal pressure may cause dislodgement of intravascular or intracardiac clots and subsequent pulmonary embolisation.[6] Pulmonary thrombi may have embolised from the portal vein, adrenal vessels or the tricuspid valve in this cat. Due to the high suspicion for DIC in this cat, it is also possible that some of the smaller pulmonary thrombi formed in situ. The clinical sequence of defaecation-induced syncope followed by acute PTE apparently resulted in this animal's sudden death.

Tricuspid insufficiency in this cat is likely to have developed secondary to pulmonary hypertension caused by smaller emboli (suggested by the enlarged pulmonary vasculature on radiographs and by the echocardiographic findings) and to have been worsened by the thrombus adherent to the valve apparatus. Thrombolytic therapy was contemplated, but postponed until the cat's haemorrhagic tendencies could be controlled. Despite anaemia, a plasma transfusion was chosen over fresh whole blood in treatment of suspected DIC to avoid adding even more volume to the circulation of a patient in right-sided heart failure. In retrospect, heparin therapy may have been beneficial. Structural myocardial disease may have been present before onset of other clinical signs and contributed to right-sided heart failure. The contribution of apparent multiple myocardial infarctions, identified at necropsy, to any clinical myocardial dysfunction in this cat is unknown. Based on limited antemortem echocardiography and on necropsy findings the cardiomyopathy was not typical of dilated, hypertrophic or restrictive disease and should therefore be placed in the category of feline unclassified cardiomyopathy. Gallop rhythms in cats are a cardinal sign of myocardial dysfunction, although could also have been present in this case due to severe anaemia or relative volume overload.

This report describes a case of neoplasia- and DIC- related pulmonary embolism in a cat, with sudden death apparently

associated with defaecation-induced syncope and subsequent PTE. Straining to defaecate may be a risk factor for sudden death in animals with underlying diseases associated with thromboembolism.

References

1. Norris CR, Griffey SM, Samii VF. Pulmonary thromboembolism in cats: 29 cases (1987-1997). *J Am Vet Med Assoc*, 1999;215:1650-1654.
2. Pouchelon J-L, Chetboul V, Devauchelle P et al. Diagnosis of pulmonary thromboembolism in a cat using echocardiography and pulmonary scintigraphy. *J Small Anim Pract*, 1997;38:306-310.
3. Fox LE. The paraneoplastic disorders. In:Bonagura J, editor. *Kirk's current veterinary therapy XII*. Saunders, Philadelphia, 1995:530-542.
4. Kapoor WN. Syncope and hypotension. In:Braunwald E, editor. *Heart disease: a textbook of cardiovascular medicine.* 5th edn. Saunders, Philadelphia, 1997:863-876.
5. Kollef MH, Schachter DT. Defecation syncope caused by pulmonary embolism. *Ann Intern Med*, 1990;113:86.
6. Kollef MH, Schachter DT. Acute pulmonary embolism triggered by the act of defecation. *Chest*, 1991; 99:373-376.
7. Kollef MH, Neelon-Kollef RA. Pulmonary embolism associated with the act of defecation. *Heart Lung*, 1991;20:451-454.

(Accepted for publication 24 January 2001)

BOOK REVIEW

***Rabbits: Health, Husbandry and Diseases**. Richardson VCG, Blackwell Science, Melbourne, 2000, 178 pages. Price $70.40. ISBN 0 632 05221 X.*

An enduring image of my time in private practice was the miserable demeanour so often exhibited by pet rabbits, most of whom spent their lives confined to a cramped hutch in some distant corner of the garden. It is therefore encouraging to read a book dedicated to the better care of pet rabbits. In this monograph, the emphasis is on practical issues and common sense. There are useful tips on handling, feeding, clinical examination, surgical procedures and administration of drugs. There is also a possibly unique chapter on behavioural problems. Unfortunately, given the book's quick reference format, there are some unexpected deficiencies: there is relatively little discussion of differential diagnoses, the amount of space devoted to some diseases is out of proportion to their practical importance, and it would have been useful to highlight certain anatomical peculiarities such as the thick-walled appendix and the round sac of lymphoid tissue at the ileo-caecal junction, both of which have the potential to be misinterpreted as gross lesions.

It is important to stress that this text should not be viewed as a source of 'scientific' information on rabbits. Discussion of disease diagnosis is based largely on clinical signs with sporadic mention of rather non-specific gross necropsy findings. The comments on clinical biochemistry are brief and generic in nature, and the modest amount of attention paid to laboratory confirmation of disease is not always helpful – in particular, the suggestion that myxomatosis and calicivirus disease can be confirmed by virus isolation is impractical and distracts from the value of far more accessible tests such as histopathology. There is also frequent reference to remedies based on vitamins, probiotics and various plants, the precise rationale for which is not clear. Having said that, the next time I see a rabbit with hair loss, I might be tempted to try some of the author's suggestions such as feeding groundsel, codliver oil or vitamin B1. If it works in rabbits…

Richardson's unsubstantiated claim that rabbits are now the third most popular pet is one I suspect has been made for other, admittedly less fecund, species. Nevertheless, rabbits don't bark, don't maul children and have bodily emanations that are far less offensive than those of their carnivorous counterparts. Practitioners with a city-based clientele will undoubtedly be seeing more of these creatures and as long as the limitations of this rough and ready guide are clearly recognised, it is not a bad starting place for those seeking a working knowledge of this species.

Malcolm France

Dr France is Director of Laboratory Animal Services at the University of Sydney