



Probable vasovagal reaction following cystocentesis in two cats

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Abstract

Case summary This case report describes an acute reaction, thought to be vagally mediated, in two cats immediately following cystocentesis. Both cats were being evaluated for feline idiopathic cystitis and developed bradycardia, hypersalivation, urination and weakness after a blind cystocentesis. Both cats recovered uneventfully with supportive care.

Relevance and novel information A vagally mediated response may occur in cats after cystocentesis, which is a common procedure performed by veterinary professionals in cats. This response may be very profound and dramatic. Affected cats will likely make an uneventful recovery. This vagally mediated response to cystocentesis, though reported by word of mouth among veterinarians, has not been described in the literature. This is the first documentation of its occurrence in cats.

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Introduction

Cystocentesis is a common medical procedure performed in veterinary hospitals to obtain urine samples for evaluation. It allows for obtaining a urine sample in a sterile fashion, which is preferred for urine culture evaluation.¹ Cystocentesis is also a practical way to obtain urine samples from animals that may not necessarily provide one on command or in a timely fashion when hospitalized, which is true in cats. Complications associated with cystocentesis, though uncommon, have been previously described, and include laceration of the aorta or uroabdomen and peritonitis.^{2–4} Cystocentesis can be a stressful procedure for cats as it may involve restraint in lateral or dorsal recumbency. The procedure may also be transiently painful. This case report describes two cats with suspected vagal reactions after routine cystocentesis was performed.

Case 1

A 4.17 kg 6-year-old male castrated domestic shorthair cat was presented for evaluation with a chief complaint of inappropriate urination. The owner reported pollakiuria and stranguria that had been going on for about 24 h. The cat had also vomited three times since urinary signs began. There was no prior significant medical history. To the owner's knowledge, cystocentesis had not been previously performed on the cat.

On presentation, the cat was quiet and responsive. Its heart rate was 180 beats per minute and its temperature was 100.8°F. Physical examination revealed moderate periodontal disease and the cat was estimated to be about 5% dehydrated. The urinary bladder was medium sized (about 4 cm) and the cat was not assessed to be painful on abdominal palpation. The rest of the physical examination was unremarkable.

The cat was placed in right lateral recumbency with minimal restraint and a blind cystocentesis was performed with a 1 inch 22 G needle. The first attempt, by a veterinary student, was unsuccessful and a second attempt was made by a veterinarian. After 2 ml of normal-appearing urine was obtained via cystocentesis, the cat vocalized and began voiding urine in a normal stream. The needle was withdrawn upon observing the urine stream. The cat immediately became weak, exhibited hypersalivation and became tachypneic (respiratory rate of 72 breaths per minute), which rapidly progressed to

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open-mouth breathing. The heart rate was noted to be approximately 120 beats per minute. Peripheral pulses were normal, and the mucous membranes remained pink. The cat was moderately obtunded but was still responsive. Spots of hematuria were noted on the towel. An indirect blood pressure was obtained and was normal (PetMap; CardioCommand), with an average systolic reading of 137 mmHg. Flow-by oxygen was initiated at 5 l/min, while an abdominal focused assessment sonography for trauma (FAST) scan was performed. There was no free fluid noted and a small urinary bladder was visualized. Thoracic FAST scan did not reveal evidence of pleural effusion. Supportive therapy consisted of oxygen support and observation. The bradycardia resolved to a heart rate of 180 beats per minute about 30 mins after the cystocentesis was performed. Within the same time period, the open-mouth breathing, tachypnea, and hypersalivation resolved, and the cat's demeanor returned to normal.

A complete blood count (CBC) and serum chemistry evaluation obtained prior to the cystocentesis were within normal limits. Urinalysis (from the cystocentesis sample) was within normal limits. The cat was diagnosed with feline idiopathic cystitis (FIC) and treated with 250 ml subcutaneous fluids (Plasma-Lyte A; Baxter Healthcare), buprenorphine (Buprenex, 0.01 mg/kg sublingually q8h; Hospira) and robenocoxib (Onsior, 1.4 mg/kg PO q24h; Novartis Animal Health). The owner elected not to pursue any additional diagnostics to evaluate the reasons for the previous vomiting. The cat was monitored in the hospital for 5 h after the event, and was assessed to be normal. Follow-up evaluation 1 month later revealed no abnormalities, and the owner reported resolution of the inappropriate urination.

Case 2

A 3.84 kg 18-year-old female spayed domestic shorthair cat was presented for a recheck evaluation of hematuria that had been assessed and treated at our institution 4 months prior. At that time, physical examination revealed dental disease and a grade 3/6 parasternal systolic murmur. CBC, serum chemistry and urinalysis were performed, and there were no abnormalities noted. A urine sample obtained via cystocentesis was negative for bacterial growth on culture. The owner declined abdominal imaging and elected for conservative treatment and monitoring. The cat was treated supportively for a presumed diagnosis of feline idiopathic cystitis with buprenorphine (Buprenex, 0.01 mg/kg sublingually q8–12h) and recommendations to increase water consumption.

At the time of the recheck examination, the owner reported that the cat had been doing well at home and the hematuria had resolved. Physical examination revealed scabs around the neck, missing upper molars

and premolars, as well as a grade 3/6 parasternal systolic murmur. There was a large (~7 cm), soft and non-painful urinary bladder on abdominal palpation.

The cat was restrained in lateral recumbency and a blind cystocentesis was performed using a 1 inch 22 G needle and normal-colored urine was collected. No complications occurred during the procedure. However, immediately following the cystocentesis, the cat vomited and defecated. The patient became tachypneic, which progressed to open-mouth breathing, and voided urine with evidence of gross hematuria. Owing to the presence of the heart murmur and potential for heart failure, furosemide (3.26 mg/kg) was administered intramuscularly and the cat was placed in an oxygen cage with a fraction of inspired oxygen of 40%. The open-mouth breathing resolved and the respiratory rate came down to 32 breaths per minute after 1 h. Thoracic radiographs and echocardiogram were declined by the owner. A urinalysis performed on the urine sample obtained via cystocentesis revealed no abnormalities other than trace amounts of protein diagnosed via urine dipstick. The patient was discharged on the same day after being observed in the hospital for about 6 h. Communications with the client 1 month after discharged revealed that the cat was doing well at home.

Discussion

Adverse reactions following cystocentesis have been anecdotally reported in feline patients. Reported clinical signs include collapse, lethargy, vomiting, defecation, hypersalivation, tachypnea and bradycardia. To our knowledge, this is the first case report describing this reaction in two cats. In this case report, both cats experienced a significant adverse event immediately following cystocentesis, which was thought to be vagally mediated based on the clinical signs associated with both events (open-mouth breathing, hypersalivation, bradycardia and hematuria). Both cats experienced a vasovagal response following the cystocentesis, sometimes referred to as 'cystocentesis shock'.

The vasovagal response described in human patients is characterized by an inappropriate combination of bradycardia and arterial vasodilation, resulting in cardiovascular collapse, syncope and, in some cases, cardiac arrest.^{5,6} This results from the simultaneous enhancement of parasympathetic tone and withdrawal of sympathetic tone. The inappropriate bradycardia is due to the augmentation of vagal activity, while the arteriolar dilation is due to sudden cessation of sympathetic activity.⁶ The vasovagal reflex may develop as a result of relative or absolute loss of blood, but can also occur as a result of strong emotions, as can be seen with humans that experience a syncopal episode as a result of needle phobia during phlebotomy.⁶

Both the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) contribute to the

regulation of various organs, including the urinary bladder, stomach, adrenal glands, pancreas and heart.⁷ Most of the PNS output occurs through the vagus nerve (cranial nerve X), which supplies parasympathetic innervation to all the viscera of the thorax and abdomen.⁸ Specifically, the urinary bladder is innervated by somatic (pudendal nerve), sympathetic (hypogastric nerve) and parasympathetic (pelvic nerve) nerves. There is a complex interaction of the SNS and PNS in urinary tract function.^{9,10}

The mechanism by which cystocentesis triggered a vasovagal response in these cats is unknown. Although stimulation of parasympathetic nerves innervating the bladder of cats has been reported,^{11,12} there has been no documented case of a vasovagal response. However, stress and/or painful stimuli are known triggers of the vasovagal response in humans.¹³ Routine medical procedures can be stressful to cats, and the associated stress can produce periods of physiologic instability.⁷ As both cats had evidence of gross hematuria immediately following the cystocentesis (even through the urine collected via cystocentesis had no evidence of gross or microscopic hematuria), it is possible that the needle insertion into the urinary bladder caused enough pain and/or trauma to the bladder mucosa to elicit a strong vagal response. In addition, both of these cats also presented with clinical signs potentially consistent with FIC. It has been reported that cats with FIC have increased catecholamine concentrations compared with normal cats, suggesting that cats with FIC have increased sympathetic tone.¹⁴ However, it has also been suggested that continual agonist stimulation may result in desensitization and down-regulation of α -2 receptors,¹⁰ which may reduce sympathetic tone and increase parasympathetic tone. It is possible that the combination of an already inflamed urinary bladder and the introduction of a needle was enough to stimulate a strong vagal response. This vagal response might explain the relative bradycardia, hypersalivation, sudden collapse and gastrointestinal signs seen in these cats. It is possible that cats with FIC are at higher risk of developing a vasovagal response to cystocentesis.

Both cats also appeared to recover within 1 h of the adverse event, which is supportive of a transient vagal influence as opposed to an ongoing pathologic event. Both cats responded well to supportive care, which consisted of oxygen therapy and observation. A blood pressure measurement was obtained on cat 1, which revealed that the cat was normotensive. Additional therapy, including the administration of an anticholinergic agent such as atropine was deemed not necessary. While a blood pressure measurement was not obtained on cat 2, the cat was closely monitored in the intensive care unit and determined to have strong femoral pulses. Although

thoracic radiographs and echocardiogram were not performed to rule out congestive heart failure, the administration of furosemide was likely not necessary. No other pharmacologic intervention was necessary as the cat's condition improved rapidly. Both cats returned to normal about 30–45 mins after the initial event and they were discharged on the same day of presentation.

Conclusions

This case report documents the first report of probable vasovagal reaction in cats following cystocentesis. Conclusions are limited owing to the rare occurrence and retrospective nature of this case report. Practitioners should be aware of this possible complication of a 'routine' procedure. If a vagal response is observed, therapeutic considerations should include monitoring of the cat until clinical signs resolve, as well as provision of oxygen therapy. In extreme cases of severe bradycardia and/or hypotension, atropine and intravenous fluid therapy may be required. Cystocentesis should not be repeated in cats with a history of such a response. Additional research is warranted to elucidate the prevalence and pathophysiology of this syndrome.

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