EPISODIC SYNCOPE CAUSED BY VENTRICULAR FLUTTER IN A TIGER (PANTHERA TIGRIS)


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EPISODIC SYNCOPE CAUSED BY VENTRICULAR FLUTTER IN A TIGER (PANTHERA TIGRIS)


Abstract: A captive, 9-yr-old castrated male tiger (Panthera tigris) from an exotic cat sanctuary and rescue facility was observed to have three collapsing episodes within a 2-wk interval prior to being examined by veterinarians. No improvement in clinical signs was noted after empiric treatment with phenobarbital. During a more complete workup for epilepsy, ventricular flutter was observed on electrocardiogram (ECG). The arrhythmia resolved with a single intravenous bolus of lidocaine. Cardiac structure and function were unremarkable on echocardiogram and cardiac troponin I levels were within normal limits for domestic felids. No significant abnormalities were noted on abdominal ultrasound. Complete blood count and biochemistry panel were unremarkable, and heartworm antigen and Blastomyces urine antigen enzyme-linked immunosorbent assays were negative. Antiarrhythmic treatment with sotalol was initiated. On follow-up ECG performed 1 mo later, no significant arrhythmias were noted, and clinical signs have completely resolved.

Key words: Arrhythmia, collapse, Panthera tigris, syncope, tiger, ventricular flutter.

BRIEF COMMUNICATION

The term “collapse” refers to a loss of postural tone with or without progression to recumbency and with or without loss of consciousness. Causes for collapse can be broadly categorized as either syncopal or nonsyncopal. Syncope is defined as a sudden transient loss of consciousness and postural tone with spontaneous recovery that occurs as a result of hypoperfusion and lack of sufficient nutrient delivery (i.e., glucose) to the reticular activating system and cerebral hemispheres of the brain for at least 6–8 sec. Causes for syncpe include decreased cardiac output from loss of preload, mechanical outflow obstruction, and arrhythmias, loss of vascular resistance, often by neurocardiogenic reflex mechanisms, and focal or generalized decrease in cerebral perfusion caused by cerebrovascular disease. Causes for syncpe include decreased cardiac output from loss of preload, mechanical outflow obstruction, and arrhythmias, loss of vascular resistance, often by neurocardiogenic reflex mechanisms, and focal or generalized decrease in cerebral perfusion caused by cerebrovascular disease. Nonsyncopal causes for collapse occur without alteration to cerebral blood flow and include metabolic disturbances like hypoglycemia or electrolyte disorders, neurologic abnormalities like seizures or neuromuscular disease, and hypoxia.

It is important to attempt to differentiate syncopal from nonsyncopal causes of collapse; differentiation is often difficult due to the many possible etiologies, similar clinical presentations, and intermittent nature of the events. Also, seizures and syncope may coexist in the same patient, adding to the diagnostic complexity. During a cardiovascular-mediated syncopal event, an animal will usually collapse into lateral recumbency and may display multifocal myoclonus, opisthotonos, vocalization, and urination. However, it is uncommon to see persistent facial spasms, persistent tonic/clonic motion, defecation, postictal dementia, or neurologic deficits. “Convulsive syncopal episodes” resulting from severe hypotension or asystole are typically preceded by loss of muscle tone, whereas seizure activity is usually preceded by atypical limb or facial movement or staring spells prior to the loss of body tone.

The most common causes for syncope in veterinary patients are cardiogenic in nature. Rhythm disturbances secondary to inherent cardiac disease account for almost two-thirds of the cases of syncope seen in dogs and cats. The following brief communication is, to the authors’ knowledge, the first case of arrhythmic syncope documented in a tiger.

A captive, 9-yr-old castrated male tiger (Panthera tigris) from an exotic cat sanctuary and rescue facility was observed to have three collapsing episodes within a 2-wk interval prior to being examined by veterinarians. The events were suspected to be seizures based on the staff’s description of the episodes (collapse with extensor rigidity lasting approximately 7 to 8 sec with immediate recovery). After a second episode was observed, the tiger was empirically treated with phenobarbital (0.74 mg/kg p.o. b.i.d.; Qualitest...
Pharmaceuticals, Huntsville, Alabama 35811, USA). When a third episode occurred after antiarrhythmic treatment had been initiated, magnetic resonance imaging (MRI) was advised to look for a possible structural brain lesion. Prior to these incidents, the tiger had no previously reported medical issues and was in ideal body condition at the time of this brief communication (174 kg). There was no known toxin exposure; however, Blastomyces dermatitidis had been previously reported on the premises.

The tiger was premedicated (5 mg midazolam i.m.; Hospira Inc., Lake Forest, Illinois 60045, USA) and then immobilized (300 mg ketamine; Bioniche Teoranta, Inveria County, Galloway, Ireland, 1.5 mg dexmedetomidine; Pfizer Animal Health, New York, New York 10017, USA, and 5 mg midazolam i.m.). Adequate sedation for intubation was not seen within 20 min, so additional medication was administered (100 mg ketamine and 0.5 mg dexmedetomidine i.m.). The tiger was then maintained on 3% inhaled isoflurane (Abbott, Chicago, Illinois 60064, USA). During anesthetic preparation for MRI, paroxysms of a sinusoidal arrhythmia were noted on the electrocardiogram (ECG) monitor. During these paroxysms, the heart was difficult to auscultate, and femoral pulses were weak to absent. After verifying that all leads and equipment were properly attached and working so as to rule out artifact, a diagnostic six-lead ECG was obtained to better characterize the rhythm. The ECG showed a predominant sinus rhythm at a rate of approximately 80 bpm with frequent paroxysms of ventricular flutter at a rate of approximately 250 bpm (Fig. 1A). A single bolus of lidocaine was given (1.15 mg/kg i.v.; Hospira Inc.) and within 4 min, complete resolution of the arrhythmia was documented (Fig. 1B). Recurrence was not seen over the next 4 hr, while the tiger’s workup was completed.

Transthoracic echocardiography was performed to look for structural cardiac causes for the arrhythmia. Subjective right and left ventricular dilation with mild to moderate systolic dysfunction was noted. These changes were assumed to be anesthesia related and not significant enough to warrant treatment. No other abnormalities were observed on echocardiogram. Blood was submitted for cardiac troponin I levels to look for evidence of ongoing cardiomyocyte necrosis or strain but were within the normal range for domestic felids (0.14 ng/ml; reference range: 0.00–0.15 ng/ml). An antigen enzyme-linked immunosorbent assay (ELISA) for Dirofi-

laria immitis was negative but was weakly positive on antibody ELISA.

Complete blood count and serum biochemistry panels were unremarkable except for a mild azotemia (blood urea nitrogen: 38 mg/dL; creatinine: 1.8 mg/dL). Urinalysis showed mild to moderate proteinuria with adequate concentration (urine specific gravity: 1.052). No evidence of Blastomyces was detected on urine antigen enzyme immunoassay. Abdominal radiographs showed mildly irregular kidney margins suggestive of chronic renal disease but were otherwise unremarkable. An abdominal ultrasound confirmed that the kidneys were irregularly margined and hyperechoic with mild loss of corticomedullary distinction. The contents of the urinary bladder were markedly echogenic, consistent with proteinuria. The only other abnormality noted on abdominal ultrasound was mild thickening of the gall bladder wall.

While positioning the animal for radiographs, a small, 1-cm mass was discovered on the caudomedical aspect of the left forelimb. Aspirates of the mass were submitted for cytology. The samples were of low cellularity and contained predominantly individual spindle-shaped cells with minimal anisocytosis and anisokaryosis, low numbers of erythrocytes, and rare, poorly preserved mast cells. The findings were more suggestive of a benign, non-neoplastic, inflammatory lesion with fibroblasts; however, the low numbers of mast cells in the presence of spindle cells raised the index of suspicion for a mast cell tumor. It was decided that if the tiger needed to be anesthetized in the future, the mass would be removed and submitted for histopathology; the period of anesthesia that day had already been protracted and results from the fine-needle aspirate were not available until the tiger was already recovering from anesthesia.

In light of the diagnostic findings, it was suspected that the reported collapsing episodes were syncopal episodes from the ventricular flutter, rather than seizures. The tiger was placed on sotalol as an antiarrhythmic medication (360 mg p.o. b.i.d.; Qualitest Pharmaceuticals), and phenobarbital was discontinued. A surveillance camera was placed within the tiger’s enclosure, and no further syncope was observed over the following 2 mo. A month after the initial workup, a repeat ECG was acquired under sedation (1.5 mg dexmedetomidine and 10 mg midazolam i.m.). A physiologic sinus arrhythmia at a rate of approximately 40 bpm was observed during the time of sedation (Fig. 1C). No significant arrhyth-
Figure 1. Six-lead ECGs acquired from the tiger. A. Note the sinusoidal waveform consistent with paroxysmal ventricular flutter at a rate of 250 bpm. The ventricular flutter terminates with a pause followed by normal P, QRS, and T morphology, consistent with normal sinus rhythm at a rate of 80 bpm. Paper speed: 50 mm/sec; amplitude: 10 mm/mV. B. After treatment with lidocaine, note the normal sinus rhythm at a rate of 70 bpm. Paper speed: 10 mm/sec; amplitude: 10 mm/mV. C. Follow-up six-lead ECG 1 mo after initiating treatment with sotalol showing normal sinus arrhythmia with a rate of 40 bpm. Paper speed: 50 mm/sec; amplitude: 10 mm/mV.
mias were present. At the time of this writing, the tiger continues to do well and has remained asymptomatic on sotalol.

Ventricular flutter is a very rapid, monomorphic ventricular tachycardia in which the individual waves and segments fuse into a sinusoidal pattern. Sustained ventricular flutter (typically lasting greater than 30 sec) results in faintness, followed by loss of consciousness, seizures, apnea, and eventually, if persistent, death. Most patients become hypotensive from inadequate ventricular filling and subsequent poor cardiac output. The condition often degenerates into ventricular fibrillation causing immediate cardiac arrest. In people, sustained ventricular tachycardias rarely occur in patients without underlying structural disease, but if structural disease is absent, the prognosis is usually very good. The tiger in this case had paroxysmal ventricular flutter while anesthetized. Ventricular arrhythmias have been reported in a tiger immobilized with medetomidine and ketamine, and so the arrhythmia could possibly have been precipitated by anesthesia. In both cases, similar doses of ketamine were used (2.30 mg/kg in this case, compared with 2.35 mg/kg in the previously reported case); however, dexmedetomidine (11.5 μg/kg) was used in this case compared with medetomidine (30 μg/kg) in the previously reported case. The authors of the prior case reported that when a lower dose of medetomidine (18 μg/kg) was used for subsequent immobilizations on the same tiger, no ventricular arrhythmias were noted.

Dexmedetomidine is the dextro-enantiomer of the racemate medetomidine and is about twice as potent as medetomidine, with similar sedation and analgesic effects. It is considered a safer preparation than medetomidine because it does not contain the levo-enantiomer, levomedetomidine, which appears to intensify the bradycardic effects of medetomidine. Accounting for the greater potency of dexmedetomidine, the tiger in this case would have received an equivalent of 23 μg/kg medetomidine, which is midway between the high and low doses of medetomidine given to the previously reported tiger.

Although the ventricular flutter may have been coincidental (i.e., anesthesia induced) rather than the true cause of collapse, this seems highly unlikely given the recent history of episodic collapse occurring in the weeks prior to workup and the complete resolution of clinical signs after sotalol administration. Additionally, the tiger did not experience recurrence of any arrhythmias during sedation for the follow-up ECG. For these reasons, it is very likely that the tiger was experiencing syncopal episodes from paroxysmal ventricular flutter. To completely confirm or rule out that the arrhythmia was the cause of clinical signs, an ambulatory ECG recording device, such as a Holter monitor or implantable loop recorder, would have to have been in place and recording while an episode occurred. Such devices are not practical in nondomesticated animals, so empiric treatment is often the mainstay of therapy if a serious arrhythmia is documented in a collapsing animal. As no similar episodes were observed after initiating antiarrhythmic therapy in this case, no further diagnostics have been recommended at this time. Because no underlying structural causes for such a malignant ventricular arrhythmia were found, discontinuation of the sotalol with close monitoring will be considered in the future in event that the arrhythmia in this case was transient.

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LITERATURE CITED


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