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Case Report

Acute Occlusion of the Abdominal Aorta with Sudden Paraplegia in a Captive Mustached Tamarin (*Saguinus mystax*)

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A wild-caught, research-naïve, adult male mustached tamarin (*Saguinus mystax*) experienced sudden onset of bilateral hindlimb paresis. Physical examination established the presence of paralysis and the lack of femoral pulses and deep pain in both legs. There were no signs of external trauma and, due to a poor prognosis, euthanasia was elected. Necropsy findings included pleural effusion, partial pulmonary atelectasis and congestion, dilatatory cardiomyopathy, a renal hemorrhagic infarct, and a thromboembolus located at the trifurcation of the distal abdominal aorta. The clinical and histologic findings were indicative of an aortic–iliac thrombosis.

In domestic animals, aortic saddle thrombosis is a common occurrence in cats;[10,21,29,32] it has also been described in dogs,[5,12,20,37] horses,[23] and calves.[28,35] Aortic thromboembolism has been reported in approximately 50% of cats with hypertrophic cardiomyopathy and to a lesser degree in cats with dilated and restrictive cardiomyopathy.[4,32] In dogs, aortic occlusions appear to result from primary aortic thrombus due to underlying prothrombotic conditions caused by protein-losing nephropathy rather than cardiomyopathy.[5,12,34] Treatment with thrombolytic agents appears to have a better effect in dogs than in cats.[37] In horses, suggested causes of aortic–iliac thrombosis include systemic infections, *Strongylus vulgaris* infection, trauma to the back, and aortic endothelial damage due to turbulent blood flow and mechanical stress.[23] Current treatment includes the administration of sodium gluconate with or without fibrinolytic enzymes, anticoagulant therapy, and continued exercise to maintain collateral circulation;[23] more recently, ultrasound-guided balloon and surgical thrombectomy have achieved limited success.[16,27]

In NHP, paresis and paralysis due to aortic thromboembolism is uncommon and has been reported in 3 owl monkeys and in a mustached tamarin.[11,18] In addition, acute death due to aortic thrombi has been reported in capuchin monkeys infected with herpes simplex 2 virus.[19] To our knowledge, there are no other reports in NHP. The suggested cause of thromboembolism in NHP appears to be associated to cardiomyopathy, similar to what occurs in cats.[18] No attempted treatment has been described in the NHP reports.[11,18]

In humans, acute aortic saddle thrombosis is a rare but often fatal pathology with postoperative mortality extremely high even when blood perfusion is restored to the lower extremities by surgical intervention.[6,33,39] Diseases associated with the presentation of aortic saddle embolism in humans are coronary atherosclerotic heart disease, recent myocardial infarction, and preexisting peripheral vascular disease.[33] A cardiac source of embolus is the predominant cause for acute aortic saddle embolism.[6] The causes of death are associated with major organ ischemia resulting in infarction and renal failure secondary to massive myonecrosis.[39] In contrast to aortic saddle embolism, aortic–iliac occlusive disease is a subset of a broad spectrum of atherosclerotic cardiovascular diseases in humans that affect the infrarenal aorta, common iliac arteries, or both.[38]

Here we describe the clinical signs in our mustached tamarin that presented with acute bilateral paresis of the hindlimbs, briefly review the pathophysiology and prospective treatments, and discuss outcomes of aortic thromboembolism in animals and humans.

Case Report

During morning health observations, a wild-caught, adult, male mustached tamarin (*Saguinus mystax*) was noted to have sudden onset of bilateral hindlimb paresis. The tamarin was enrolled in an IACUC-approved research protocol but was research-naïve at the time of presentation. The animal's health history was unremarkable except for dental tartar and gingivitis, tuberculin test negative, and no signs of infectious or contagious disease during quarantine. The tamarin was housed and cared for according to the *Guide for the Care and Use of Laboratory Animals*,[17] the Animal Welfare Act,[2] and Animal Welfare Regulations[3] at the National Institute of Allergy and Infectious Diseases, NIH, an AAALAC-accredited animal facility. Husbandry procedures included feeding New World Primate Diet 5040 (Purina Mills, St Louis, MO), Marmoset Diet (ZuPreem, Mission, KS), and dietary supplements (bananas, apples, grapes, and marshmallows) and providing unrestricted access to water. The animal was housed in a stainless steel 6.0-ft^2 biocontainment cage (Primate Products, Miami, FL) with a PVC nesting box, nectar log, and other callitrichid environmental enrichment objects. Room conditions included a 12:12-h dark:light photoperiod and temperature of 24 ± 2 °C. The tamarin was pair-housed and routinely screened for intestinal pathogens by bacterial cultures and for parasites by wet mounts and fecal flotation.[13]

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Approximately 3 y after the tamarin's arrival at our institution, the animal was noted to have a decreased appetite for biscuits but continued to eat fruit (grapes) and canned marmoset diet (ZuPreem). Weight was monitored weekly and showed no significant change.

However, 17 d after the decrease in appetite, the tamarin was found recumbent. Physical examination revealed decreased musculature and lack of movement in the hindlimbs, with a flaccid tail, no paniculus response, no anal tone, no palpable femoral pulse, no deep pain response, and both rear legs cool to touch. Mucous membranes were pink, with a 2-s refill. The differential diagnoses included trauma to the lumbar section of the spine or intervertebral disc, a primary spinal cord disorder, neoplasia compressing the spinal cord, aortic thromboembolism, and aortic dissecting aneurysm. In light of the poor prognosis, the tamarin was euthanized by sodium pentobarbital overdose according to the *AVMA Guidelines on Euthanasia* and was submitted for postmortem examination.[1]

At necropsy, removal of the skin from the subcutis was difficult, suggesting dehydration. Subcutaneous and intraabdominal stores of adipose tissue were limited. No gross lesions were noted that affected the lower back vertebrae, hindlimb muscles, joints, or bone structures that would explain the clinical signs observed. A modest amount of serosanguinous fluid was present in the thoracic cavity. The lungs were congested and partially atelectatic. The heart was enlarged and had a rounded appearance. The liver was congested, and the left kidney had a large hemorrhagic infarct involving the cortex. A large, well-adhered blood clot was visible at the trifurcation of the distal abdominal aorta and extended toward the iliac arteries. The major organs and abdominal aorta were fixed in 10% neutral-buffered formalin, embedded in paraffin, cut at 4 µm, and stained with hematoxylin and eosin for histologic examination. For the abdominal aorta, 4 planes of depth were processed to evaluate the thromboembolus. In addition, selected slides were stained with Masson trichrome, Mallory phosphotungstic acid–hematoxylin, elastic Van Gieson, and Gram stains.

Microscopically, the myocardium presented with mild to moderate multifocal myofiber degeneration and loss, with interstitial fibrosis. The pulmonary arteries had mild multifocal subintimal thickening. The lung parenchyma was atelectatic and congested, with discreet focal areas containing intraalveolar accumulation of fine to dense granular, amphophilic deposits and foamy alveolar macrophages showing alveolar structure preservation. The left kidney had a focally extensive, severe hemorrhagic infarct (Figure 1). Both kidneys had multifocal, mild, interstitial lymphoplasmacytic infiltrates and globally diffuse, moderately expanded mesangial matrix and tubular proteinuria. Small and medium-sized renal arteries had multifocal, moderate proliferative arteriosclerosis (Figure 2). The liver showed acute, centrilobular diffuse, severe congestion with hepatocellular degeneration (attenuation), necrosis, and mild multifocal hepatocellular hemosiderosis. The abdominal aorta and iliac arteries had marked thrombosis characterized by focally extensive, severe, endothelial degeneration, subendothelial fibrin deposition, and hemorrhage (Figure 3 and 4). The final diagnoses were congestive heart failure, hindlimb paresis, cardiomyopathy, aortic and iliac artery thrombosis, hepatocellular vacuolation and necrosis, glomerulonephropathy, and pulmonary alveolar proteinosis.

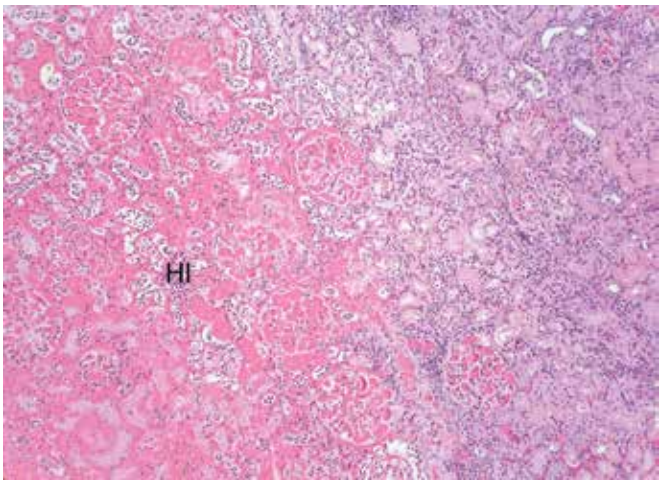

Figure 1. *Saguinus mystax*. Left kidney. Focally extensive, severe hemorrhagic infarct (HI) affecting the cortex. Hematoxylin and eosin stain; original magnification, 40×.

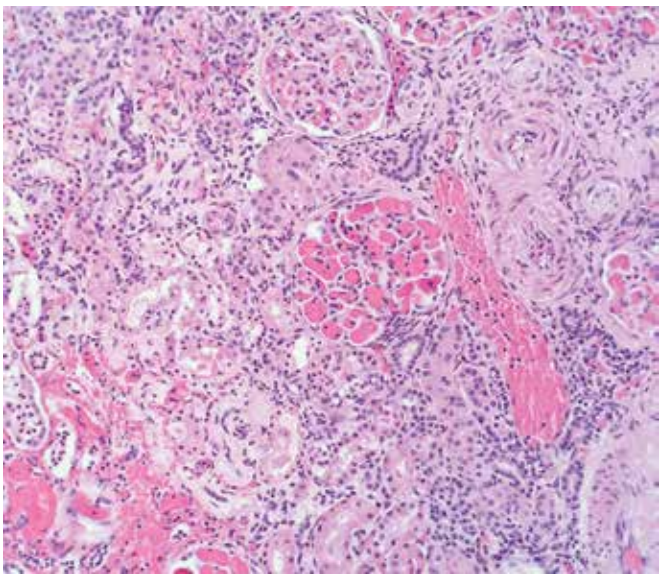

Figure 2. *Saguinus mystax*. Left kidney. Multifocal, mild, interstitial lymphoplasmacytic infiltrates and diffuse, moderate, expanded mesangial matrix and tubular proteinuria. Small and medium-sized renal arteries show multifocal moderate proliferative arteriosclerosis. Extensive areas of coagulative necrosis with hemorrhage at the interface with viable tissue. Hematoxylin and eosin stain; original magnification, 100×.

Discussion

The differential diagnoses for sudden bilateral paraplegia include thoracolumbar spine trauma that compromises the innervation of the lower extremities, either due to vertebral fracture or compression from a displaced intervertebral disk; neoplasia, either of the spinal cord or adjacent tissues that exerts a mechanical impairment of the nerves; vascular and inflammatory disorders of the spinal cord; hypokalemia and hyperkalemia (but these are not accompanied by the impaired sensation demonstrated in our case); and saddle thrombus or dissecting aneurysm of the abdominal aorta.[21,31] Because we found no other lesions that might explain the clinical signs, sudden occlusion of the abdominal aorta was most likely the cause for the bilateral posterior paresis noted in this mustached tamarin.

Multiple factors contributed to the deteriorating condition of this animal. The thrombus located at the trifurcation of the caudal aorta (where it divides into left and right iliac arteries and median sacral artery) resulted in the hindlimb paresis noted clinically. The flaccid tail and lack of anal tone suggest that the

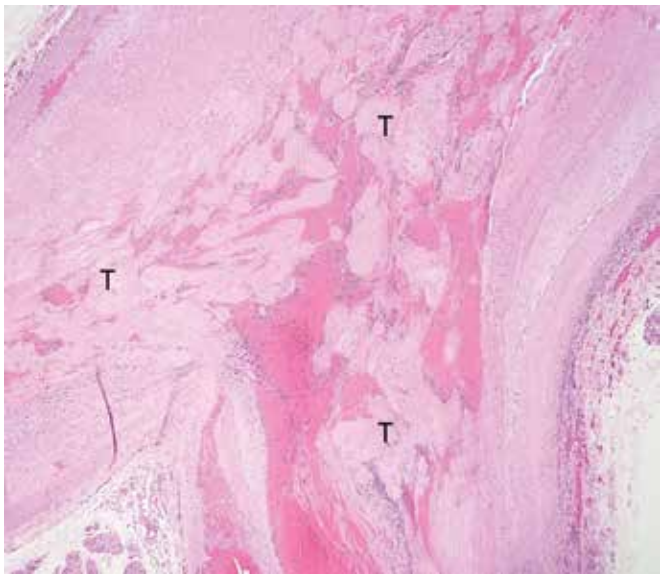

Figure 3. *Saguinus mystax*. Abdominal aorta and iliac arteries. A well-adhered thrombus (T) at the trifurcation of the distal abdominal aorta extends toward the iliac arteries. Hematoxylin and eosin stain; original magnification, 40×.

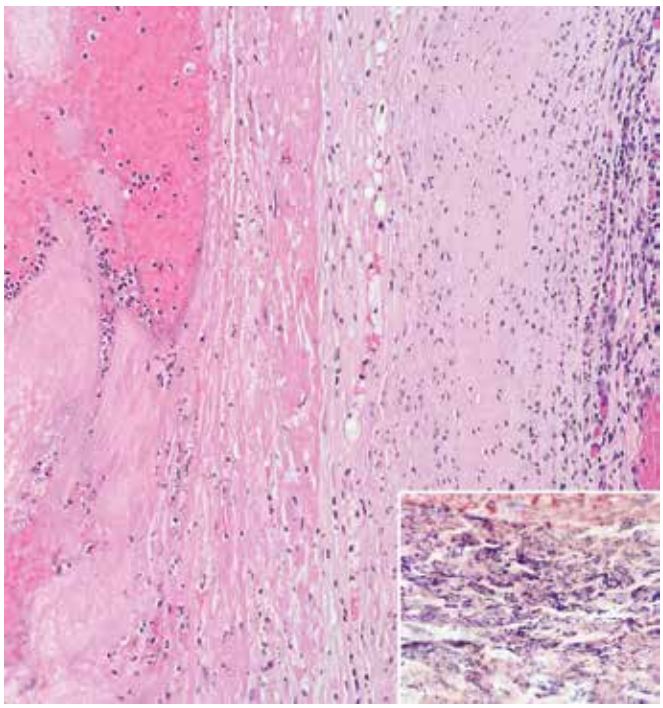

Figure 4. *Saguinus mystax*. Abdominal aorta. Marked thrombosis characterized by focally extensive, severe, endothelial degeneration, subendothelial fibrin deposition and hemorrhage. Hematoxylin and eosin stain; original magnification, 200×. Insert shows a section of the thrombus stained with Mallory phosphotungstic acid–hematoxylin, demonstrating extensive fibrin deposition (purple strands). Original magnification, 400×.

tamarin had lumbosacral spinal cord infarction as a result of the aortic occlusion or ischemic necrosis or atrophy of the tail and anal muscles due to occlusion of the median sacral artery. However, we were unable to confirm either hypothesis because neither the lumbosacral spinal cord nor median sacral artery was collected during necropsy for histologic examination. The hemorrhagic renal infarct likely developed after a section of the thrombus detached and blocked an arcuate artery, causing ischemic necrosis or thrombus development locally, given that this site is a point of blood turbulence and shear stress;[8] however, a thrombus or embolus was not noted histologically. Although we did not measure the tamarin's blood pressure, the pathologic changes in the microvasculature in the form of proliferative arteriolosclerosis, especially in the kidneys, are suggestive of hypertension.[30] The myocardial changes, pulmonary congestion, thoracic effusion, and hepatic congestion with hemosiderosis and hepatocyte vacuolation are changes consistent with congestive heart failure.[30] This animal also presented pulmonary alveolar proteinosis, a rare human disease characterized by accumulation of surfactant in alveoli without generation of an inflammatory response, a condition recently described in tamarins.[24]

Cardiomyopathy and renal disease are common in captive mustached tamarins, and a recent retrospective study demonstrated that a high percentage of mustached tamarins with cardiomyopathy also develop intracardiac thrombosis and aortic dissecting aneurysms.[13,14] Both conditions potentially can trigger aortic saddle embolism.[6,33] Left ventricular enlargement with or without aneurysm, depressed global systolic function, and a large thrombus at the left ventricular apex are all cardiac sources of embolism, and the trifurcation of the aorta is a common site at which large emboli lodge.[15,25] Often an intracardiac thrombus dislodged from the left ventricle migrates through the distal aorta until the diameter of the blood vessel is smaller than the thrombus, thus blocking the vascular lumen. However, our tamarin did not have an intracardiac thrombus, the development of the aortic saddle thrombus locally. The hemorrhagic renal infarct might have developed as a result of an embolus carried by the bloodstream; however, as mentioned earlier, no intracardiac thrombus was noted macro- or microscopically. Perhaps the renal infarct was caused by an embolus that generated from the renal artery, which is another location of disturbed blood flow.[8] Furthermore, high blood pressure can damage the vascular endothelium by shear stress (frictional force of the blood on the endothelial layer), resulting in the development of thrombi; the trifurcation of the caudal aorta, in particular, is a point subject to shear stress and blood flow turbulence, where vascular endothelium is at increased risk for damage, as well as are the renal arteries.[8] Recent studies show that disturbed blood flow and the associated low and reciprocating shear stress induce sustained activation of several atherogenic genes in endothelial cells.[8] These effects include vasoconstriction; high endothelial cell turnover; high macromolecular permeability and LDL uptake; increased expression of adhesion molecules, inflammatory, and chemokine genes; promotion of WBC adhesion and platelet aggregation; high oxidative stress and generation of reactive oxygen species; vascular smooth muscle cell activation; and fibronectin and fibrinogen deposition—all of these effects result in the promotion of atherosclerosis and thrombosis.[7,8]

In cats, thrombi typically form on the left side of the heart, with male predisposition.[21,32] Treatment of acute aortic saddle embolism in cats is mostly supportive—keep the affected limbs warm, and provide anticoagulant and antiplatelet therapy to help dissolve the embolus. Pain management includes the use of opioids; diuretics are provided when congestive heart failure is present; vasodilators (hydralazine, acepromazine) help collateral circulation in the hindlimbs; avoid the use of angiotensin-converting enzyme inhibitors (for example, enalapril, benazepril) in acute episodes due to potential coexisting renal infarctions; oxygen therapy is beneficial when anoxia or dyspnea is noted; however, fluid therapy using conservative

crystalloid therapy is recommended only when there are no signs of congestive heart failure.[21,22] Chronic treatment in cats is aimed at treatment of the underlying condition (most commonly heart disease); anticoagulant therapy using heparin–warfarin overlap may help dissolve the thrombus, although the efficacy and safety of warfarin therapy has not been sufficiently investigated in felines.[21,22] The suggested dose of low-molecular-weight heparin may not be efficacious in cats given that this dose is based on human data, with no controlled studies in cats, and antiplatelet therapy is still under investigation. Owing to multiple drug interactions for warfarin, care must be exercised when a feline patient is on any other medication regimen.[21] Complications are multiple and serious: gangrenous necrosis of hindlimbs, self-mutilation, visceral occlusions that lead to organ infarction and failure, cardiac arrhythmias, recurrent thromboembolic events, and death. Therapy with thrombolytics (streptokinase and recombinant tissue-plasminogen activator) can cause hemorrhagic complications and acute reperfusion syndromes (hyperkalemia, acidosis, death); diuretic therapy risks include blood volume contraction and azotemia; vasodilator therapy can cause hypovolemia, hypotension, and decompensation of renal function; and fluid therapy can result in volume overload and congestive heart failure.[21,22] Antiplatelet treatment with aspirin or clopidogrel is well tolerated, but its efficacy is unknown.[22] Constant monitoring is essential in the feline patient—physical, daily renal, electrolyte and coagulation profile and, if possible, continuous electrocardiogram.[22] Prognosis is always guarded; more favorable when only one limb is compromised, but there is a strong chance for thromboembolic episodes to recur. When occlusion of visceral arteries is present—clinically characterized by acute onset of abdominal pain, vomiting, and lethargy—the prognosis is grave.[21] Body temperature seems to be a positive correlate with time of the event, with a more favorable outcome when the temperature close to normal range.[21,22]

The pathophysiologic mechanism of aortic thromboembolism in dogs is different than in cats. In dogs, aortic occlusions appear to be due to a primary aortic thrombus as a result of underlying prothrombotic conditions caused by protein-losing nephropathy rather than cardiomyopathy.[5,12,34] Treatment with the thrombolytic agents streptokinase and recombinant tissue-plasminogen activator appears to have a better response in dogs than in cats.[36,37] However, published reports are sparse and, in some cases, contain conflicting results.[36] Reports on the use of tissue-plasminogen activator in dogs with thromboembolism have shown inconsistent results, with complete dissolution of the thrombus in one case, whereas another report indicated that treatment with tissue-plasminogen activator was ineffective.[5,9] A standard therapeutic approach using platelet inhibitors (aspirin and clopidogrel) and anticoagulants (heparin, low-molecular weight heparins, and warfarin) has not yet been established.[36] In addition, there are no reports of long-term management of the disease. However, survival times in dogs appear to be shorter for acute onset of clinical signs than with chronic presentation.[20,36] In humans, emergency surgical intervention to restore limb and organ perfusion is the treatment of choice in acute aortic thromboembolism.[39] Regardless, acute aortic occlusion involving both iliac arteries has a very poor prognosis in humans, with aortic–iliac arterial reconstruction the most effective treatment when the patient can tolerate laparotomy under general anesthesia.[39]

The aortic–iliac occlusion in our tamarin was severe and sufficiently extensive to block several visceral and parietal channels that provide collateral circulation and help maintain perfusion to the lower limbs. In humans, these collateral vessels include: the subclavian artery, which provides collateral circulation to the external iliac artery; the lower intercostal, subcostal, and lumbar arteries, which provide collateral circulation to the iliac internal artery; and branches of the superior mesenteric artery, which provide collateral circulation to the internal iliac artery.[38] Interestingly, men have a greater incidence of the disease, perhaps because collateral circulation is more efficient in women, given that the ovarian and uterine arteries also provide effective collateral circulation to the hindlimbs through anastomoses with the urogenital artery and, therefore, internal pudendal to deep femoral arteries.[26]

We know that captive mustached tamarins are prone to develop cardiomyopathy, intracardiac thrombosis, aortic aneurysms, and glomerulonephropathy—all of which are conditions that predispose to thromboembolism. In addition, this species might be predisposed to essential hypertension, as suggested by the characteristic renal arterial lesions, which also leads to disruption of vascular endothelial homeostasis and the activation of several atherogenic genes that result in atheroma and thrombosis. Taken together, and in light of similar preconditions in other species, these findings suggest that mustached tamarins are at greater risk of developing acute aortic thromboembolism than are other primates, including humans. Further study of these conditions in mustached tamarins may promote understanding of the disease in humans and domestic species and may prevent or help to decrease the frequency of this disease in captive mustached tamarin colonies. Acute aortic thromboembolism should be high on the list of differential diagnoses for tamarins presenting with posterior paraplegia. Additional research on the cardiovascular pathologies that affect *S. mystax* may improve treatment and prevention strategies that might be applied in other species, including humans.

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