

Tourniquet Injury: A Case Report and Review of the Literature

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ABSTRACT

This is the first report in veterinary medical literature of a dog suffering from tourniquet injury due to a circumferential foreign body of the distal limb. A 5-year-old male neutered Bichon Frise presented for evaluation of bilateral distal forelimb swelling, lameness, and licking/chewing of his forepaws. Upon closer inspection, a rubber band was found within the edematous skin and fur of each forelimb at the carpi. This report includes a discussion of a similar injury affecting human infants as well as a review of the human and veterinary literature of tourniquet application.

INTRODUCTION

There are many anecdotal reports of tourniquet injury in dogs and cats. Veterinarians have reported instances of animals suffering from tourniquet injury secondary to rope, wire, string, bandages, blankets, hay, and rubber bands. However, there is only one report of a circumferential foreign body in the veterinary literature.¹ In that case, a cat presented for respiratory distress and was later found to have been suffering from a rubber band around the neck. This is the first report in veterinary literature of a dog suffering from tourniquet injury due to a circumferential foreign body of the distal

limb. Circumferential foreign bodies causing tourniquet injury can cause local edema and can be difficult to identify within edematous tissue and fur. The purpose of this report is to increase awareness of this condition in dogs and cats. This report includes a discussion of a similar injury affecting human infants as well as a review of the human and veterinary literature of tourniquet application.

CASE REPORT

A 5-year-old male neutered Bichon Frise was presented for evaluation of bilateral distal forelimb swelling, lameness, and licking/chewing of his forepaws of 10 days duration. Previous treatment with an antibiotic (cephalexin 250 mg PO BID), an anti-inflammatory corticosteroid (prednisone 5 mg PO q24h), and an Elizabethan collar had not alleviated his clinical signs. A physical exam showed severe bilateral distal forelimb edema. There was cutaneous erythema and crusting on the palmar aspect of both forepaws. There was a circumferential lesion around both carpi with bilateral full-thickness palmar lacerations.

Upon closer inspection, a rubber band was found within the edematous skin and fur of each forelimb at the carpus (Figures 1–5). Both feet were warm, superficial nervous sensation was present, and digital pulses were palpable distal to each rubber band. A diagnosis of tourniquet injury was made.

Figure 1: Clipped forepaws showing the circumferential lesion and the distal edema and erythema.



Figure 2: Close-up of Figure 1.



Figure 3: Close-up of the palmar aspect of the forepaw showing the depth of injury and crusting.



Figure 4: Following foreign body removal. Note the relative size of the rubber bands in relation to standard thumb forceps.

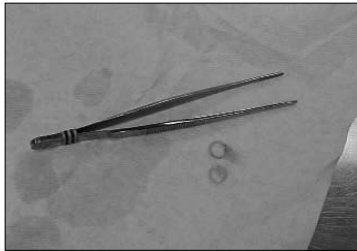


Figure 5: Following foreign body removal and flushing of the wounds.



Figure 6: Close up of Figure 5.



Sedation was achieved using intramuscular administration of 0.2 mg/kg hydromorphone and 0.02 mg/kg acepromazine. The rubber bands were removed with cold-sterilized suture scissors (Figures 6–8). The wounds were flushed and bilateral pressure wet-to-dry bandages were placed. A 25- μ g/hr fentanyl patch was also placed on the wounds. Given the presence of peripheral pulses and intact superficial nervous sensation, the prognosis for return to full function was good. However, the depth of circumferential skin lesions of each carpus was unknown, and the prognosis for healing without skin sloughing was guarded.

The patient was presented the following day with an intact blood supply, superficial nervous sensation, and normal skin color of the paws. There was no ligamentous instability. The bandages were replaced with dry wraps. The patient was presented again 2 days later with further progression towards healing. The bandage was not replaced at that time.

DISCUSSION

To the authors' knowledge, this is the first report of a tourniquet injury in the veterinary medical literature. However, a very similar injury occurring in human infants is well documented.²⁻⁴ The injury is termed hair-thread tourniquet syndrome, ischemic hair syndrome, toe tourniquet syndrome, or acquired constriction ring. The syndrome occurs most commonly in infants less than 6 months old and the etiology is human hair or thread that becomes encircled around one or more digits.²⁻⁴ Infants are presented to their physician for evaluation of irritability and/or inappetence. Physical examination shows edema and erythema and may also show serous drainage, crusting, and laceration of the superficial tissues of the affected digit. Close examination reveals the entrapped hair/thread, which is very difficult to identify given the local edema. Adjacent skin may also re-epithelialize over the constricting object thus further obscuring it.²

The constricting object acts as a tourniquet by blocking lymphatic drainage causing lymphedema, which reduces local blood supply and results in ischemic damage.³ If treatment is delayed, vascular compromise and necrosis may warrant amputation of the digit. Treatment is aimed at removing the hair or thread under general anesthesia. Making incisions along the medial and lateral aspect of the affected digit to relieve edema and to sever any remaining hairs/threads has been frequently described.^{2,3} The prognosis following treatment depends on tissue compromise at the time of diagnosis, but is generally excellent with normal development of the digit in most cases.²⁻⁴

Although foreign objects described as tourniquets can cause injury as described in this report, surgical tourniquets (Esmarch tourniquet, pneumatic tourniquet, blood pressure cuffs, etc.) have been used for centuries.^{5,6} The purpose of a tourniquet in surgery is to provide a bloodless field allowing the surgeon to view the anatomy, operate quickly, and reduce blood loss. The potential complications of tourniquet use in surgery have prompted several studies that evaluate the effects of tourniquet application on local tissues and blood flow, as well as metabolic changes that occur with prolonged tourniquet use. These changes are applicable to tourniquet injury as described above, because the physical processes of intra-operative tourniquet use and tourniquet injury are similar.

SKIN

Reports of hair-thread tourniquet syndrome in humans, as well as the report presented above, show that skin laceration may occur secondary to tourniquet injury. Reports of skin necrosis following improper bandage usage have also been reported.⁷ Conceivably, the degree and type of injury would be related to the width of the tourniquet, pressure of the tourniquet, and duration of application.

MUSCLE

Muscle damage may result from direct compression of the tissues beneath a tourniquet or ischemia of the tissues distal to a tourniquet. Muscle seems more severely damaged by ischemia versus compression. Rupture of muscle is an uncommon event with tourniquet injury and has been reported only once in the human literature.⁸ Both primary and secondary forms of ischemic myopathy have been described. Primary ischemia is immediate, related to local metabolic changes, and characterized by myofibril degeneration and cell necrosis within 2 to 3 hours after the onset of ischemia. Secondary ischemia is delayed, developing gradually following tourniquet release, and is characterized by microvascular congestion and muscle autolysis.⁸ This is significant when considering wound management with tourniquet injury in that necrosis may continue even after tourniquet removal in muscle tissue that initially appears viable. Several studies evaluating muscle tissue in ischemic conditions have been performed.^{6,8} Researchers have found that ischemia produces myocyte damage characterized by inflammation, necrosis, calcium ion channel disturbances, muscle weakness, and changes within the mitochondria of myocytes, including disorganized cristae, swollen mitochondria, and electron-dense material within the mitochondria.^{6,8} These effects vary in severity with the length of the ischemic episode and the pressure of tourniquet inflation. It has been theorized that an increase in muscle capillary permeability contributes to local edema in tourniquet syndromes.⁸

NERVES

Tourniquet paralysis syndrome has been described in humans. The syndrome is defined as an abnormal function of peripheral nerves distal to a tourniquet and is manifested by a loss of motor function, touch sensation, vibration, position sense, and the inability to discern light pressure.^{6,8} The abil-

ity to detect warmth, coldness, and pain is maintained, as is sympathetic function. The syndrome can last for days to months but the long-term prognosis for recovery is good.^{6,8,9} Tourniquet paralysis syndrome may occur in veterinary patients suffering from tourniquet injury, but it may be difficult or impossible to diagnose. Nerve paralysis, however, can be diagnosed easily in veterinary patients and is one of the most common complications following intra-operative tourniquet use.⁶ Nerve paralysis is defined as the loss or impairment of motor function and is more commonly known as palsy in the human medical literature.¹⁰ Nerve paralysis may be due to either direct compressive forces beneath a tourniquet or to ischemic conditions distal to a tourniquet. The effects of tourniquet application on nerve function have been under debate for decades.^{5,6,8,11} In vitro studies using sections of nerve, nerve conduction studies, and sensorimotor evaluations have supported ischemia as the major cause of nerve palsy.^{5,6,8} Electromyography, theoretical models using stress analyses, anecdotal experience, and studies evaluating microscopic changes within nerves under compression have supported compression as the major cause of nerve palsy.^{5,6,8} It is currently supported that nerve palsy following tourniquet application is due primarily to mechanical compression; however, it is accepted that ischemia may also contribute. The primary injury is thought to be displacement of the nodes of Ranvier, demyelination, and stretching of the paranodal myelin.^{8,11}

Studies in human medicine have shown that motor impairment is more common than sensory, and that large myelinated fibers (sciatic, femoral) are more prone to injury than smaller fibers.¹¹ These findings may help explain potential physical exam findings when evaluating a patient suffering from tourniquet injury. Studies have supported the intuitive hypothesis that tissue damage is related to the pressure of the tourniquet and the duration of application; higher tourniquet pressure and increased

duration of application lead to more tissue compromise and a greater degree of tourniquet injury.⁹

BLOOD FLOW

As previously mentioned, the function of a tourniquet intra-operatively is to reduce blood flow distal to the tourniquet site. The effects on blood flow distal to a tourniquet are very pronounced, and this has severe implications affecting an animal suffering from tourniquet injury. Blood flow distal to a tourniquet diminishes to less than 1% of normal, and venous return diminishes to less than 0.2% normal.⁶ Following tourniquet release, an increase in blood flow 10-times normal was consistently noted in humans.⁶ Following an ischemic episode of 2.5 hour-duration, perfusion returned to normal 1.75 hours later in a study using dogs.⁶

A study performed on rabbits demonstrated that blood flow to the skin was compromised at lower external pressures compared to muscle. Blood flow to skin and muscle was compromised when tourniquet pressures exceeded 30 mmHg and 50 mmHg, respectively.¹² Thus, pressure necrosis may occur to skin despite adequate peripheral pulses. This is most applicable as a precaution to the use of bandages and intra-operative tourniquets; however, this may also be relevant in wound management during treatment of tourniquet injury in that skin necrosis may have occurred or may occur despite adequate peripheral pulses.

Early reports of increased bleeding tendencies in dogs following tourniquet application prompted evaluation of coagulation function in primates during tourniquet application.⁶ In addition, a study performed in 1966 showed local clotting time in humans to be increased to 16 minutes (normal = 20–40 seconds) following tourniquet application of 1-hour duration.⁵ Tourniquet application reportedly activates the extrinsic, intrinsic, and fibrinolytic pathways. Compression of tissues causes local ischemia and hypoxia leading to acidosis and tissue damage. Tissue thromboplastin is

released that activates the extrinsic coagulation pathway, and damaged endothelium activates the intrinsic coagulation pathways, as well as the fibrinolytic pathway.⁶ Studies did not find any effect on platelets. The increased coagulation and fibrinolytic activity are characterized by reduced fibrinogen, antithrombin III, and plasminogen, and increased fibrin split products and fibrinopeptide A concentrations. Following tourniquet release, hemostasis is quickly achieved by ingress of clotting factors, dilution of fibrin split products, and removal of fibrin split products and fibrinopeptide A by venous drainage.⁶

METABOLIC CHANGES

Local metabolic changes in tissues distal to a tourniquet include hypoxia, hypercarbia, increased creatine phosphokinase, increased lactate, hypoglycemia, hypocalcemia, and hyperkalemia.⁶ The local pH may drop from 7.4 to 6.8, the partial pressure of oxygen (pO_2) may drop from 22 mmHg to near zero, and the partial pressure of carbon dioxide (pCO_2) may increase from 38 mmHg to 62 mmHg.⁶ Changes were relative to the duration and pressure of tourniquet application. There was a significant difference between 1 and 2 hours of application and also between tourniquet inflation pressures of 250 and 500 mmHg.⁶ Local hypocalcemia may contribute to impaired nerve function.⁶ Hyperkalemia has been shown to have vasodilatory properties in humans and rats, with increases in local muscle blood flow of 5- to 10-times normal following tourniquet release.⁶ It has been shown that capillary permeability increases when tissue pO_2 decreases to 10 mmHg and the volume within the tissues increases by 100%; thus, hypoxia may greatly contribute to local edema.⁶ Creatine phosphokinase did not increase when "breathing periods" of 10 minutes were allowed at 1-hour intervals.⁶ Breathing periods are used periodically during intra-operative tourniquet application and are periods of time during which tourniquet deflation is allowed between periods of

tourniquet inflation that may permit the tissues to recover briefly from the ischemic episode. There is controversy within human medicine regarding the efficacy of breathing periods in reducing tourniquet injury following intra-operative use.

Post-tourniquet syndrome, also known as Volkmann's contracture, has been described in the human medical literature.^{5,7} The syndrome is due to ischemic injury leading to metabolic derangements secondary to tourniquet application. This syndrome is characterized by changes in local pH, CO_2 , and O_2 that are manifested by edema, stiffness, pallor, weakness (without paralysis), and numbing sensations. This syndrome occurs following injury to a fascial compartment causing increased intercompartmental pressure, collapse of arterioles and venules, venous obstruction, and muscle damage, the end result of which is limb deformity (contracture). There is one report of Volkmann's contracture in a dog following a ballistic injury.⁷ However, the syndrome is rare in veterinary patients presumably due to the presence of collateral circulation and less defined osseo-fascial compartments in dogs.⁷

Depending on the location of the tourniquet (ie, amount of tissue affected) and the duration of application, one may need to be concerned about ischemia-reperfusion injury. A discussion of ischemia-reperfusion injury is beyond the scope of this report. However, it has been shown that following intra-operative tourniquet application for stifle surgery of 2-hour duration in dogs, there were increased local toxic oxygen products in the form of hydrogen peroxide (H_2O_2).¹³ In an animal with a tourniquet injury affecting relatively greater body mass (especially above the stifle or elbow), immediate removal of the tourniquet may not be warranted and pre-treatment for reperfusion injury may be indicated. Treatment of reperfusion injury is controversial as ischemia-reperfusion injury is a complex process. Treatment modalities that may be useful include neutrophil blockers (anti-ICAM-1

antibodies, anti- β 2 integrin antibodies, CD-18 blockers), allopurinol, deferoxamine, and dimethyl sulfoxide.¹⁴

CONCLUSION

The intra-operative use of tourniquets has prompted several studies to determine the local effects of tourniquet application on skin, muscle, nerves, blood flow, and the local metabolites. Because the physical processes of intra-operative tourniquet use and tourniquet injury are similar, these studies are relevant and reveal details about the process that may aid in treatment and prognosis; however, there are shortcomings to this comparison. Although the physical processes of intra-operative tourniquet application and tourniquet injury are similar, they are not identical. Studies discussed in this report used a variety of tourniquets often used intra-operatively, including Esmarch and pneumatic tourniquets.

However, an animal suffering tourniquet injury may have a variety of foreign objects causing the injury, including ropes, wire, chain link, bandages, or rubber bands. How these items may alter the local tissues and microenvironment is unknown. Moreover, in most studies discussed here, tourniquets were applied for a relatively short period of time (minutes to hours), whereas an animal may have been suffering from tourniquet injury for days to weeks prior to presentation. Additionally, the studies discussed in this report used a wide variety of species, including dogs, humans, rabbits, and primates. One can presume the physiology across species is similar. However, as discussed, Volkmann's contracture, which is very common in humans following tourniquet application, is seldom recognized in veterinary patients. There may be important differences in physiology that remain to be elucidated. The studies presented here also used a variety of tourniquets (Esmarch or pneumatic), a variety of occlusion times (minutes to hours), a variety of breathing periods, and a variety of inflation pressures. These variables make direct comparisons between results difficult.

Based on the studies discussed above, it is surprising that the dog in this report maintained deep blood flow and nervous function to both feet considering the foreign bodies had been present for 10 days.

Although the rubber bands appeared small and would be assumed to exert great pressure, they had elastic properties allowing them to stretch. Also, although the rubber bands had lacerated some superficial tissues, they may have relieved some local edema allowing preservation of deeper neurovascular structures. Upon presentation, a complete neurologic exam was difficult to interpret given the bilateral distal forelimb lameness and edema. The lameness was presumed to be due to pain and not neurologic compromise because the patient had demonstrated intact nervous sensation. Reperfusion injury was not a concern in this case due to the relatively small proportion of tissue involved compared to body mass.

Tourniquets have been used in human surgery for centuries and have been used to a lesser extent in veterinary surgery. However, there are risks associated with the use of tourniquets in medicine as demonstrated by Volkmann's contracture, nerve palsies, ischemic skin necrosis, injuries secondary to bandage application, and myocyte damage. Furthermore, as this report illustrates, severe injuries can occur secondary to foreign bodies acting as tourniquets. These injuries can arguably be much more severe than bandage injuries or intra-operative tourniquet use because the two variables affecting the degree of injury, pressure and duration of application, are not controlled, and their degree and extent may be unknown.

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