Acute masticatory muscle compartmental syndrome in a dog

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CASE DESCRIPTION
A 5.5-year-old sexually intact male Bull Terrier was referred for evaluation because of sudden facial swelling and an inability to close its mouth.

CLINICAL FINDINGS
Physical examination revealed bilaterally elevated nictitating membranes, an inability to adduct the mandible without assistance, and severe, diffuse, firm masticatory muscle swelling. Computed tomographic examination of the head revealed symmetric bilateral enlargement of the temporalis, masseter, and pterygoid muscles with heterogeneous contrast enhancement. Intracompartmental pressures in the left and right temporalis muscles as measured with an invasive arterial blood pressure transducer were 72 and 96 mm Hg, respectively.

TREATMENT AND OUTCOME
Emergent fasciotomy of the temporalis and masseter muscles was performed, followed by medical management with corticosteroids and analgesics. The diffuse facial swelling resolved within 1 week after surgery. Results of serologic testing for antibody against masticatory 2M muscle fibers were negative. Results of histologic examination of temporalis muscle specimens were consistent with mild to moderate multifocal neutrophilic and histiocytic myositis with myofiber degeneration and necrosis.

CLINICAL RELEVANCE
Acute compartmental syndrome should be considered as a differential diagnosis for dogs with a sudden onset of severe skeletal muscle swelling, signs of pain, and dysfunction. Findings for this dog with acute compartmental syndrome isolated to the masticatory muscles suggested that emergent fasciotomy followed by medical management may be an effective technique for treatment of this rare disease in dogs. (J Am Vet Med Assoc 2018;253:606–610)

ABBREVIATIONS
ACS  Acute compartment syndrome

A 5.5-year-old 23.0-kg (50.6-lb) sexually intact male Bull Terrier was taken to its primary care veterinarian for evaluation because of facial swelling, an inability to close its mouth, and bilateral nasal discharge. The owner reported that 2 days prior to evaluation, the dog had attacked a metal trap that contained a woodchuck. Per the description, the dog had no physical contact with the woodchuck, but no more detailed information was available about the incident. After the incident, the dog was suddenly unable to close his mouth and rapidly developed bilaterally elevated nictitating membranes and severe, diffuse facial swelling. Two years prior to this incident, the dog had similarly attacked a cage containing a woodchuck and developed mild facial swelling.

The primary care veterinarian had performed a CBC, serum biochemical analysis, and skull radiography. Radiography revealed no obvious fractures or abnormalities. Serum biochemical analysis revealed high activities of aspartate aminotransferase (1,256 U/L; reference range, 15 to 66 U/L), alanine aminotransferase (260 U/L; reference range, 12 to 118 U/L), and creatine kinase (80,969 U/L; reference range, 59 to 895 U/L). The CBC revealed neutrophilia (12,136 neutrophils/µL; reference range, 2,060 to 10,600 neutrophils/µL) but no other abnormalities. The dog was then sent home with tramadol hydrochloride (4 mg/kg [1.8 mg/lb], PO, q 8 h) and clindamycin (10 mg/kg [4.5 mg/lb], PO, q 12 h).

Two days later, the dog was brought to a referral hospital because of continued signs of inappetence with no clinical improvement at home. On initial physical examination, the dog appeared bright and alert. Abnormal findings included bilaterally elevated nictitating membranes, an inability to adduct the mandible without assistance, and a fractured left maxillary canine tooth. The masticatory muscles were severely swollen and firm, and signs of pain were elicited on gentle palpation (Figure 1). No superficial wounds or abrasions were identified. Results of neurologic examination confirmed masticatory muscle dysfunction and indicated decreased nasal sensation bilaterally, apparent ophthalmoplegia, and bilaterally miotic but responsive pupils. No other neurologic abnormalities were identified, and results of orthopedic evaluation were unremarkable. Serum electrolyte concentrations were measured, and all were within reference ranges.

The dog was premedicated with oxymorphone (0.1 mg/kg [0.045 mg/lb], IV) in preparation for CT examination of the head, and maropitant citrate (1 mg/kg [0.45 mg/lb], IV) and diphenhydramine (2 mg/kg [0.9 mg/lb], IM) were administered. Anesthe-
Sia was induced with propofol (3 mg/kg [1.36 mg/lb], IV) and midazolam (0.3 mg/kg [0.136 mg/lb], IV) and maintained with isoflurane in oxygen. Iohexol contrast medium (2 mL/kg) was administered IV, and CT of the head was performed with a 64-slice CT scanner, revealing profoundly, bilaterally enlarged temporalis, masseter, and pterygoid muscles. All 3 muscles had a heterogeneous contrast-enhancement pattern (Figure 2). The retropharyngeal lymph nodes appeared mildly enlarged. Both ocular globes were exophthalmic, with the posterior aspect of the left globe slightly deformed. No abnormality was detected in the nasal cavity; therefore, the previously observed nasal discharge was considered transient and likely clinically unimportant. Because compartment syndrome of the masticatory muscles with secondary associated (or consequential) ischemic neuropraxia was suspected, fasciotomy was recommended to the owner and subsequently pursued.

While still anesthetized, the dog was placed in sternal recumbency and the skin over each temporalis muscle was aseptically prepared. An 8-cm dorsal midline incision was made at the level of the temporalis muscle, and the subcutaneous tissues were dissected. Intracompartmental pressures were then measured prior to incision of the temporalis fascia. For this measurement, a standard 20-gauge hypodermic needle connected to a fluid-filled (saline [0.9% NaCl] solution) line, which was connected to an invasive arterial pressure transducer, was inserted approximately 1 cm deep into the left and then right temporalis muscles, revealing pressures of 72 and 96 mm Hg, respectively. Immediately afterward, a 4-cm, full-thickness incision of the temporalis fascia on the right side was made in a caudal to rostral direction (Figure 3). This process was then repeated for the left temporalis muscle fascia.

Figure 1—Photographs of a 5.5-year-old sexually intact male Bull Terrier referred for evaluation because of sudden facial swelling and an inability to close its mouth. A—On initial examination, the dog had bilaterally elevated nictitating membranes, an inability to adduct the mandible, and severely swollen masticatory muscles. Locations of the left temporalis muscle (asterisk) and left masseter muscle (arrow) are shown. B—By 24 hours after fasciotomy of the temporalis and masseter muscles, the nictitating membranes were no longer elevated and the masticatory muscle swelling was less severe. The dog continued to have a mildly dropped jaw.

Figure 2—Transverse contrast-enhanced CT image of the head of the dog in Figure 1. The temporalis (star), masseter (polygon), and pterygoid (black arrow) muscles appear bilaterally and symmetrically enlarged. The deep portion of the temporalis muscle appears hypodense with no contrast enhancement. An irregular cap-like contour of the contrast-enhanced portion of the temporalis muscle (white arrow) is superficial to the edematous deeper portion of the temporalis muscle. The pterygoid and masseter muscles have a heterogeneous enhancement pattern that is subtler than that of the temporalis muscle.

Figure 3—Intraoperative photographs of the dog in Figure 1 showing full-thickness incision of the temporalis fascia on the left side in a caudal to rostral direction (A) and bilateral fasciotomies of the temporalis muscles (B).

After bilateral fascial releases were performed and left open (Figure 3), temporalis muscle compartment pressures were reassessed bilaterally by use of the previously described technique, revealing pressures of 54 and 27 mm Hg in the left and right sides, respectively. A 0.5 X 0.5 X 1-cm section of each temporalis muscle was removed and submitted for histologic examination and complete muscle analysis, including examination of the muscle for underlying inflammation, necrosis, fibrosis, fiber loss, microorganisms, or specific cytoarchitectural abnormalities.
A venous blood sample was also collected for serologic testing for antibody against masticatory 2M muscle fibers. The fascia was left open, and the subcutaneous layer was closed with 3-0 polydioxanone in a simple continuous pattern. The dermis was closed with 3-0 poliglecaprone 25 in a continuous intradermal pattern.

The skin over the right masseter muscle was then incised in a dorsoventral direction. The fascia was incised in a horizontal direction along the muscle fibers and left open. The subcutaneous layer was closed with 3-0 polydioxanone in a simple continuous pattern. The dermis was closed with 3-0 poliglecaprone 25 in a continuous intradermal pattern. This procedure was then repeated for the left masseter muscle. No intracompartmental pressure measurements were obtained for the masseter muscles. Following surgery, an esophagostomy tube was placed. A single lateral thoracic radiograph was obtained, evaluation of which revealed appropriate tube placement. The dog was then allowed to recover from anesthesia, with no complications noted.

In the postoperative period, the dog received 1 dose of methylprednisolone acetate (30 mg/kg [13.6 mg/lb], IV) as well as methadone (0.3 mg/kg, IV, q 6 h) and crystalloid solution (60 mL/kg/d [27.3 mL/lb/d], IV). The following morning, the nictitating membranes were no longer elevated, extraocular muscle function and pupillary size were unremarkable, and the dog was observed to eat without any apparent dysphagia (Figure 1). The dog no longer appeared uncomfortable on palpation of the masticatory muscles, but a mildly dropped jaw persisted. It was discharged from the hospital that day with prednisone (0.65 mg/kg [0.3 mg/lb], PO, q 12 h) and codeine (1.3 mg/kg [0.6 mg/lb], PO, q 8 h).

The dog was returned to the referral hospital 1 week after surgery for a recheck examination. All 3 incisions appeared clean, dry, and intact. The previous severe, diffuse facial swelling had completely resolved, and the esophagostomy tube was removed. The owner reported that the dog was doing well at home, appearing comfortable and eating as usual. Results of serologic testing for antibody against 2M muscle fibers were negative (titer, < 1:100). Results of muscle profile analysis were consistent with mild myofiber atrophy and perimysial edema of undetermined origin. No inflammation, necrosis, fibrosis, fiber loss, microorganism, or specific cytoarchitectural abnormalities were identified. Histologic examination of temporalis muscle specimens revealed multifocal swollen eosinophilic myofibers, fragmentation of the sarcoplasm, and occasional myofiber infiltration by macrophages. Rare myofiber mineralization was noted as well as mild to moderate intervening edema with a mild neutrophil and macrophage infiltration and occasional plump fibroblasts. The microscopic interpretation was consistent with mild to moderate multifocal neutrophilic and histiocytic myositis with myofiber degeneration and necrosis (Figure 4).

At a recheck examination 2 weeks after surgery, the dog was still doing well. No recurrence of swelling, dysphagia, or signs of pain was evident. Results of physical and neurologic examination were unremarkable. The owner had been mistakenly administering prednisone at a higher dose than prescribed (1 mg/kg rather than 0.65 mg/kg), and tapering of prednisone administration over a 12-day period was initiated.

Twenty-six days after surgery, the dog was taken to its primary care veterinarian because of an incisional infection and partial dehiscence along the left masseter muscle. Treatment was initiated at that time with amoxicillin trihydrate–clavulanate potassium (21.7 mg/kg [9.9 mg/lb], PO, q 12 h). Twenty-nine days after surgery, the dog was re-admitted to the referral hospital for a recheck examination, which revealed improvement of the infected incision. Given the amount of time elapsed since surgery, the incisional dehiscence may have been consistent with secondary abscess formation, particularly given the severe muscle trauma. However, because the dog was evaluated elsewhere for the dehiscence, the possibility of secondary abscess formation was not confirmed. Recommendations were made to continue antimicrobial administration and return the dog in 2 weeks for another recheck examination and antibody test, but the owner declined further diagnostic testing. No additional follow-up was pursued by the owner.

Discussion

Acute compartment syndrome refers to an increase in intracompartmental pressure within a closed osteofascial compartment, resulting in a decrease in perfusion pressure. These changes lead to hypoperfusion and neuromuscular hypoxemia with irreversible necrosis of tissues as well as contracture and potential tissue infection. Several forms of ACS...
have been described in human and veterinary medicine, including a form involving the extremities and abdominal and thoracic cavities. The dog of the present report had ACS of the skeletal muscle.

In human medicine, reported causes of skeletal muscle compartment syndrome include trauma (eg, fractures), thermal injuries, iatrogenic causes (eg, positioning during surgery or bandaging), nephrotic syndrome, rhabdomyolysis, bleeding disorders, and infection. Fractures are the most common cause of ACS of the extremities in humans, accounting for approximately 69% to 75% of cases. However, soft tissue injury without fracture has been associated with ACS of the extremities in as many as 23% of cases.

As in human medicine, trauma is the most common cause of ACS in veterinary medicine. Additional reported causes include hemangiosarcoma, other expansile tumors, postanesthetic myopathy, and hemorrhage. In all reported instances, the episodes of ACS involved the skeletal muscle of an extremity. To the authors' knowledge, no reports exist of ACS isolated to the masticatory muscles in the veterinary or human literature.

The recommended treatment for ACS of the extremities in both human and veterinary medicine is surgical decompression typically via fasciotomy. Most commonly, an intracompartamental pressure of 30 mm Hg is the cutoff used in human medicine to justify surgical intervention, regardless of the affected muscle. This practice is based on the belief that an intracompartamental pressure > 30 mm Hg is insufficient to maintain capillary flow within the affected muscle, risking potential muscle damage, which is based on findings in dogs. Compartmental pressures in healthy humans are typically < 10 to 12 mm Hg and in healthy dogs range from –4 to 0 mm Hg. In the dog of the present report, intracompartamental pressures of the left and right temporalis muscles were 72 and 96 mm Hg, respectively. Therefore, ACS was suspected and emergent fasciotomy pursued. Other authors recommend that surgical intervention be pursued if the intracompartamental pressure is within 30 mm Hg of the patient's diastolic arterial blood pressure. Presurgical attempts at arterial catheter placement in the dog of the present report were unsuccessful, so no such comparison could be made. Still, surgical intervention was considered not only because of the immensely high intracompartamental pressure, but also because the severe muscle swelling was causing neurologic dysfunction.

After fasciotomy of both temporalis muscles was performed, intracompartamental pressures decreased to 54 and 27 mm Hg for the left and right temporalis muscles, respectively. This procedure was then repeated for masseter muscles given the physical examination and CT findings. Ideally, pressure measurements would have been obtained for the masseter muscles as well. In human medicine, delayed primary closure is often recommended following fasciotomy. Vacuum-assisted closure may also help assist with recovery and future closure. Because of financial constraints and the lack of veterinary evidence to support those practices, primary closure was pursued. The dog had clinical improvement within 24 hours after surgery, appearing more comfortable and better able to chew food, and no longer had ophthalmoplegia or miotic pupils.

The cause of ACS in the dog of the present report remains unknown. Traumatic causes, rhabdomyolysis, and acute masticatory muscle myositis were considered at the initial referral evaluation. In any of these conditions, excessive contraction of the masticatory muscles combined with a potentially high body temperature may have contributed to ACS. Trauma was considered given the patient's clinical history. However, on physical examination, no superficial trauma was identified. Furthermore, the inflammation of the masticatory muscles was symmetric, making trauma less likely.

Given the degree of muscle swelling, we considered that the dog might have masticatory myositis. A similar event involving a metal trap had occurred with the dog 2 years previously that resulted in mild facial swelling. That previous episode reportedly resolved with medical management. Following the second, more severe episode, myofiber mineralization was noted on histologic examination, which may have indicated a recurrent problem. However, it is more common for dogs with masticatory myositis to have trismus rather than the inability to close their mouth. Regardless, a serum sample was obtained and submitted for testing for antibody against 2M muscle fibers. The test, which has high sensitivity and specificity for detection of masticatory myositis, yielded negative results. Our recommendation to repeat the antibody test, which was made to rule out the possibility that the negative results were attributable to antibody suppression by previous corticosteroid treatment, was declined by the owner.

Exertional rhabdomyolysis is another disease with a sudden onset, involving marked breakdown of striated muscle and a substantial increase in serum creatine phosphokinase activity. In veterinary medicine, racehorses and sled dogs are most commonly affected. In human medicine, exertional rhabdomyolysis reportedly causes ACS, but no such association has been reported in veterinary medicine. Clinical features of exertional rhabdomyolysis include muscle soreness, pain, myoglobinuria, and high serum aspartate aminotransferase and creatine phosphokinase activities. The dog of the present report had no signs of muscle soreness and no increase in these enzyme activities. Exertional rhabdomyolysis can lead to acute kidney injury; however, the dog had no evidence of azotemia or corresponding changes in serum electrolyte concentrations. Although no urinalysis was performed while the dog was hospitalized, no gross pigmenturia was noticed, also making exertional rhabdomyolysis unlikely.

Reports of ACS in the veterinary literature have been limited to cases involving the thoracic cavity, abdomen, and extremities; however, findings for the
dog of the present report suggested that ACS can also affect the muscles of mastication and therefore should be considered in patients with a sudden onset of severe skeletal muscle swelling, signs of pain, and dysfunction. Surgical decompression via fasciotomy provided a safe and effective treatment option.

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Footnotes

a. Aquilion 64, Toshiba Corp, Tustin, Calif.
b. Deltran, Utah Medical Products Inc, Midvale, Utah.
c. PDS, Ethicon, Johnson & Johnson, Somerville, NJ.
d. Monocryl, Ethicon, Johnson & Johnson, Somerville, NJ.
e. Esophagostomy feeding tube, MILA International Inc, Florence, Ky.
g. Plasmalyte, Zoetis Inc, Florham Park, NJ.
h. Clavamox, Zoetis Inc, Florham Park, NJ.

References