The NERVE of this bull, a truly jaw-dropping case

Cranial Nerve Deficits (VII&VIII) due to Trauma

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Introduction

There have been numerous causes of facial paralysis and vestibular disease that have been described in domestic animals.[10] The facial nerve possesses a complex anatomical course, therefore facial nerve dysfunction can be due to congenital, traumatic, inflammatory, infectious, and neoplastic etiologies.[13] In cattle, meningoencephalitis caused by *Listeria monocytogenes*, brain abscess, traumatic damage from head butting or being kicked in the face, otitis media/interna, and neoplasia's such as meningiomas and acoustic neuromas, may also cause vestibular signs and facial paralysis. [5,10] Diagnostic tools include physical examination, blood work, diagnostic imaging, and bacterial analysis and culture. Treatment options include antibiotics, steroids, non-steroidal anti-inflammatory drugs, and even more novelty treatments in food animals such as laser therapy. The following case report details the clinical signs, diagnostic approach, pathophysiology, treatment, and follow up considerations of cranial nerve deficits in cattle.

History and Presentation

F302A is an approximately 2-year-old Wagyu bull that presented to Mississippi State University College of Veterinary Medicine (MSU-CVM) Food Animal Service on November 11, 2020. F302A had a history of fighting with another bull 2-weeks prior, where head-butting was observed and F302A was knocked down. After the fight, he had a swollen left eye, so his owners called his referring veterinarian to have him examined. His rDVM gave him a steroid injection. F302A's inflammation persisted, and he started having problems utilizing his jaw, which led to him losing approximately 150 pounds. His owners were concerned with possible lumpy jaw, so they decided to bring him to MSU-CVM.

Upon presentation, F302A was quiet, alert and responsive. He was slightly ataxic upon entering the clinic. He weighed approximately 1,195 lbs. and was underweight with a body condition score of 3/9, with 4-6 being ideal. His vital parameters were within normal limits with a respiratory rate of 24 breaths per minute (range 10-30 breaths per minute), heart rate of 72 beats per minute (range 55-80 beats per minutes), and a temperature of 102.4° Fahrenheit (range 100°-102.5° Fahrenheit). Cardiopulmonary auscultation revealed no crackles, wheezes, or irregular heart sounds. Abdominal auscultation revealed normal rumen contractions, but upon rectal palpation it was noted he had decreased rumen fill. Unilateral cranial nerve deficits were observed on his left side on initial examination of the head. F302A had a drooped left ear and drooped left muzzle. His left eye was partially closed due to his upper eyelid being drooped. He had an abnormal accumulation of feed material in the left jaw and hypersalivation was observed. He had a left-sided head tilt, an absent menace response, and absent palpebral response on the medial and lateral canthus of his left eye. He had no sensory response to stimuli on his left nostril, and he had decreased tone when his tongue was pulled to the left. He had an exaggerated mandible deviation to his left side when ruminating. When offered water and food, noticeable amounts of water and food fell out. On otic examination, there were copious amounts of purulent debris present in his left ear. There were no other irregularities noted and the remainder of the physical exam was within normal limits.

Diagnostic Approach and Differential Diagnoses

Important tools to better diagnose the causes of cranial nerve deficits in cattle include, but are not limited to, a thorough history of the patient, a physical exam in which cranial nerve function is assessed, hematological analysis, cerebrospinal fluid analysis and culture, and

bacterial culture of aural discharges. Diagnostic imaging also plays an important role in the evaluation of facial nerve disorders, especially when traumatic in origin. Radiographs are useful to examine dense tissues and bone. Computed tomography (CT) is useful when identifying bony abnormalities of the facial nerve's bony canal where it is located, and magnetic resonance imaging (MRI) is useful when identifying any soft tissue abnormalities surrounding or involving the facial nerve.[13] Our top differential diagnoses in this case were traumatic injury to the head, listeriosis, caused by *Listeria monocytogenes,* and otitis media/interna. Lumpy jaw, brain abscessation, and neoplasia were also differentials on our list, but they were lower priority due to lack of clinical signs or inability to diagnose pre-mortem.

Clinical signs of facial nerve paralysis can vary depending on the cause, severity, location and chronicity of the lesion.[15] Clinically, facial nerve paralysis in cattle will manifest with a droopy ear, ptosis, and atonic lips. Saliva can occasionally be seen drip out of the affected side of the mouth. Signs associated with dysfunction of the vestibular system in cattle include a head tilt, a loss of balance, and circling. When the lesion is unilateral, cattle will lean towards the lesion; if the animal is recumbent, it will lie with the lesion side down.[12] Clinical findings associated with otitis media/interna include epiphora, purulent discharge from the external ear canal, head tilt, and paresis or paralysis of the pinna. [6] Findings associated with listeriosis include encephalitis, which is characterized by bending of the head to one side, walking in circles, ear droop, tongue protruding from the mouth, and abundant nasal secretion and drooling. Irritability, impaired locomotion and increasing emaciation are also common findings with listeriosis. [17]

Generally, a definitive diagnosis of listeriosis is achieved by bacterial culture, where the organism is usually isolated from sterile tissue or a body fluid sample such as CSF or blood, by

histopathology, and sometimes by serology.[17] Otitis media/interna, in adult cattle, is poorly documented and is a lot more prevalent in calves. In general, diagnosis can be achieved by hematological analysis, radiographing the tympanic bullae, bacterial culture of aural discharge and CSF analysis.[6] Radiographs, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound are very useful in achieving definitive diagnosis of trauma to bone and soft tissue.

Due to F302A being a production animal and financial limitations, radiographs were the only imaging modality utilized in F302A's case. Radiographic results revealed heterogeneous soft tissue swelling and osteomyelitis of the left cornual process with associated frontal sinusitis. F302A's large animal profile and complete blood count both contained no significant abnormalities indicative of inflammation or present infection, and the CSF sample collected from the lumbo-sacral space revealed no infectious agents, inflammation or evidence of neoplasia present. Unfortunately, no aural discharge sample was submitted for culture, so we were unable to verify what type of organisms were present. Based on our diagnostic findings and given that blood and CSF analysis weren't indicative of infection and that his radiographs revealed soft tissue swelling and localized osteomyelitis, F302A got a presumptive diagnosis of cranial nerve deficits due to trauma. Given the fact that in most cases definitive diagnosis of listeriosis cannot be accomplished in a live animal, instead it requires a post-mortem pathologic and histopathologic examination and/or culture on organs collected post-mortem [17], it was decided to also keep listeriosis on F302A's presumptive diagnosis and treat accordingly.

Pathophysiology

Facial nerve paralysis mechanism is dependent on the cause.[14] Fracture of the petrous temporal bone, presence of *Listeria monocytogenes* infection, extended or prolonged recumbency causing compression of the facial nerve over the mandible [3], and ear infections are the main causes of facial nerve paralysis. The facial nerve is a mixed nerve with special visceral efferent, general visceral efferent, special visceral afferent and general somatic afferent functions.[7] A thin myelin sheath surrounds the nerve in the intracranial portion, where it is subject to demyelinization.[7] The facial nerve runs through a narrow bony canal, so any inflammation or growth of the nerve will cause compression, causing ischaemic changes. Any skeletal abnormality or trauma can disrupt the relationship between the facial nerve and its bony canal, leading to paralysis.[16]

Listeria monocytogenes is a gram-positive bacterium capable of causing both human and animal disease. In cattle, turning or twisting of the head to one side, dullness, walking in circles and head pressing against solid objects are common clinical signs observed with the neural form of listeriosis.[8] It is ubiquitous in nature, and it may be endemic in certain regions. It is a facultative intracellular pathogen that invades macrophages and tissue cells of the infected hosts where it is able to proliferate.[11] Once the bacterium has internalized into the host cells, it is able to escape from the phagosomal compartment and is then able to enter the cytoplasm.[11] It then spreads from cell to cell, and in infected hosts, the bacteria can cross the intestinal wall at Payer's patches and invade the mesenteric lymph nodes and blood.[11] Since the liver is the main target organ, the bacteria multiply inside hepatocytes.[11] Through a series of recruitment of cells, hepatocyte lysis occurs, causing a bacterial release.[11] This bacterial release causes a prolonged septicemia, thus exposing the brain to infection.[11] Otitis media/interna is a common disease in

cattle. Although reports in adult cattle exist, it is most often seen in young animals from 1 week to 18 months of age.[2] There has been no gender predisposition found, but inclement weather has been found to increase its incidence.[2] Reportedly, beef calves are most often affected, but this condition seems to be on the rise with dairy calves.[2,8] The most common route of infection is through the Eustachian tube.[2] Pathogens are also able to reach the middle ear by hematogenous spread, or migration from the external ear.[2] One of the main etiological agents found in otitis media/interna is *Mycoplasma bovis*.[2,6] It is usually found either alone, or in association with other bacteria.[2,6] It is common for purulent aural discharge to appear into the external acoustic meatus.[2] The exudate filling the tympanic bulla increase its pressure, often causing the tympanic membrane to rupture.[2]

Treatment and Management

Treatment is generally warranted for traumatic facial nerve paralysis, listeriosis, and otitis media/interna. Facial trauma is usually treated with steroids or non-steroidal anti-inflammatory drugs, helping resolve inflammation. Listeriosis usually progresses rapidly; in encephalitic cases, death can usually occur in a timeframe as early as 2 days up to 2 weeks of the first onset of clinical signs.[17] Therefore, rapid diagnosis and treatment initiation immediately after onset of clinical signs are imperative for successfully treating listeriosis. Oxytetracycline and penicillin G have been used successfully to treat listeriosis.[17] Otitis media/interna occurs either sporadically or as an outbreak, causing important economic losses.[2] To prevent any economic loss, diagnosis should be made during the early stages of the disease, allowing for effective treatment. Antimicrobial therapy is the treatment of choice.

On presentation, F302A received a dose of sodium iodide intravenously, which was intended to treat his possible lumpy jaw. His initial treatment also consisted of antibiotics and steroid therapy. He received a 14-day course of antibiotics to treat our presumptive diagnosis of listeriosis and otitis media/interna. The antibiotics chosen were Penicillin at 44,000 IU/kg subcutaneously every day and Nuflor at 40 mg/kg subcutaneously every 4 days. To help alleviate inflammation, he received 3 doses of dexamethasone at 0.1mg/kg intramuscularly every other day and received 2 final tapered doses every other day at .05mg/kg. His left ear was flushed with sterile saline daily until purulent debris was no longer present. The first 3 days of his hospitalization, F302A was unable to consume any hay and only consumed minimal amounts of grain and water. After 4-5 days of his stay, his cranial nerve deficits began to improve. He began to consume larger amounts of hay, grain, and water, but they were still quantities well below maintenance requirements. Although his facial swelling and cranial nerve deficits were improving, F302A's jaw deviation worsened, so a new therapeutic approach was implemented to his treatment plan. Nerve and tissue regeneration can be a lengthy process; therefore, massage and heat are often useful therapies to help denervated muscles maintain their integrity and function.[15] Laser therapy, also known as "photobiomodulation therapy" (PMBT), is a type of light therapy that provides beneficial therapeutic outcomes including but not limited to the promotion of wound healing, alleviation of pain or inflammation, and nerve and tissue regeneration.[1] Seven days after initial presentation, F302A started receiving laser therapy of his masticatory muscles and jaw. The main muscles targeted were the masseter, temporal, lateral and medial pterygoid, digastricus and sternohyoideus. A dose of 10 J/cm^2 was administered. Administration with an on-contact laser probe with a circular massage motion and slight pressure is ideal and were utilized for thorough penetration. The effect of PBM is

cumulative. After each session, the patient's clinical condition should be improving.[7] The amount of time per laser therapy session was based on his response, but he averaged approximately 10 minutes per session, once a day. Significant improvement was noticed as sessions went on, and it was noted during treatment that he would often lean into the probe, indicative sign of relief. As sessions went on, he also became easier to put in the squeeze chute, as if he was anticipating the laser therapy and was eager to have it performed. Seven days after initiating laser therapy and discontinuing steroid administration, it was also decided to give F302A one-weeks' worth of non-steroidal anti-inflammatory drugs to help with any remaining inflammation. F302A received 1 mg/kg of oral Meloxicam for 4 days and then received a tapering dose of 0.5 mg/kg for 3 days.

Case Outcome

After 24 days in the hospital, F302A was discharged. At the time of his discharge, F302A was moving his jaw normally while ruminating and the exaggerated deviation towards the left had subsided. Maintenance amounts for a bull his size are approximately 27-29 lbs. of dry matter intake, and approximately 15-19 gallons of water per day. He was able to consume maintenance amounts of hay, grain and water. At the time of his discharge, he had regained approximately 75% of his cranial nerve function, but it is unknown if he will ever regain full nerve function. It was advised to keep him in a small pasture/paddock where he would be away from other bulls and where he could be closely monitored for decreased feed intake or worsening of his neurological signs. Nerve injuries can take up to 6 months to fully heal, so it was advised to get in contact with us if he started having abnormal jaw movements or difficulty eating again, in which case a maintenance protocol of laser therapy could be started.

Discussion

During his stay at MSU-CVM, F302A showed substantial improvement from his presenting state. While a definitive diagnosis could not be determined, traumatic injury, listeriosis and otitis media/interna are all possible causes consistent with the clinical signs showed by F302A. He was treated for all 3 differentials and showed substantial improvements. As therapy of listeriosis in ruminants is often difficult and unsuccessful, and as mentioned before, it is an often rapidly progressing disease, it was determined that listeriosis was unlikely, as he presented to us 2-3 weeks after initial onset of clinical signs. However, Listeriosis could not be ruled out and presumptive treatment must be administered. This meant he received a 2-week course of antibiotics and steroids to treat for listeriosis and otitis media/interna. Our next step was then to treat for traumatic injury with laser therapy and NSAID administration. 10 days after laser therapy began, F302A's jaw deviation ceased completely, and the cranial nerve deficits improved by 75%. It is unknown whether the NSAIDs or laser therapy or a combination of both helped improve his condition, but it is worth noting that laser therapy is a fairly underutilized resource in large animal medicine, especially cattle. The broad systemic effects of PBMT make it an ideal therapy to use to promote wound healing, resolve inflammation, and treat pain.[4] It also plays a key role in tissue and nerve regeneration, which NSAIDs can't do. Another great advantage that laser therapy has compared to NSAIDs is that laser therapy carries no withdrawal period, an extremely valuable tool in animals meant for human consumption. Lastly, laser therapy is priced reasonably, and in our current case, each session cost $20, making it fairly accessible for the producer looking for an alternative treatment to NSAID use. F302A's case taught us to have a flexible treatment mindset and gave us some valuable insight on a treatment option that is often underutilized in bovine medicine.

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