Unrelenting Forces Meet Geriatric Elbows

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Introduction:

Pressure sores, also known as bed sores or decubitus ulcers, are commonly seen in geriatric patients, patients with poor body condition, or those with splints or casts.[1,2,3] They are a result of ischemic assault over part of the body that is subject to prolonged mechanical forces such as compression (pressure), shear, or friction.[2] Pressure sores may form on any part of the body, but are most commonly seen over bony prominences such as the hips, elbows, and stifle. Pressure sores result in pain and act as a portal for bacteria to freely enter the body if not treated appropriately or, ideally, prevented. Treatment methods and prevention methods start the same: relieve pressure from the affected site. This can be done with thick padded bedding, the use of slings or wheelchairs, challenging the patient with activity, changing patient position, and removing any iatrogenic causes.[3,4] Further treatment involves wound management, surgical closure, and appropriate antimicrobial therapy. A case is described in detail below identifying how pressure sores can change from a small setback to a disease entity of their own. Treatment and prevention options are discussed in detail both in relation to this case as well as to provide an overarching view of medical and surgical management of pressure sores.

Clinical Report:

A 12-year-old male neutered yellow Labrador Retriever was referred to the Mississippi State University College of Veterinary Medicine (MSU-CVM) on September 21, 2017 after suffering from a pressure sore over his right elbow for several months. At the beginning of June 2017, the patient presented to his RDVM for a routine appointment at which time his arthritis medication was changed to Galliprant and tramadol with no other changes to his diet or lifestyle recommended. After this appointment, the patient developed severe vomiting and diarrhea. He was hospitalized at the RDVM where pressure sores developed over both elbows, with a more

severe sore forming over his right elbow. After discharge from his RDVM, the sore over the patient's right elbow worsened. Closure was attempted and a Penrose drain was placed on June 29, 2017, but the wound opened again after suture removal on July 10, 2017. The pressure sore was treated with antibiotic ointment, frequent bandage changes, and padding via a DogLeggs device. Bloodwork performed at this time revealed an elevated white blood cell count and the patient was referred to the MSU-CVM Internal Medicine department. The patient had been medicated with prednisone from June to August 2017 and treated with enrofloxacin, marbofloxacin, and ampicillin for suspected wound infection with no improvement seen. At the time of referral, the patient was only receiving Gabapentin, Rutin, folate, Denamarin, and prednisolone-acetate ophthalmic drops.

On initial physical examination, the patient was bright, alert, and responsive with a temperature of 101.3 degrees Fahrenheit, a heart rate of 92 beats per minute, and a respiratory rate of 40 breaths per minute. His mucous membranes were pink and moist with a capillary refill time of less than 2 seconds. Cardiovascular auscultation revealed no murmurs or arrhythmias and bronchovesicular auscultation did not reveal any crackles or wheezes. The patient's body condition score was an 8 out of 9. He had a non-healing wound on the right elbow and a thickened band of tissue on his dorsal neck (suspected to be secondary to wearing his DogLeggs sling). Further examination revealed mild muscle atrophy and pitting edema of the right forelimb and an erythematous pressure sore on his left elbow. The patient also had a freely moveable, firm mass on the dorsal aspect of his caudal rear trunk cranial to his right hip, as well as some reddening in his inguinal region. Oral examination revealed moderate dental tartar and gingivitis. Ocular examination revealed bilateral nuclear sclerosis (normal aging change) and a mild amount of dried discharge in the medial canthus of the right eye. The rest of his physical

examination was within normal limits. Orthopedic examination revealed decreased flexion and extension of most joints and pain on hip extension bilaterally. The patient was admitted into the intensive care unit and was maintained on gabapentin and tramadol for pain control, and he was provided a padded kennel. Complete blood count (CBC), neurochemistry profile, and urinalysis samples were collected on presentation. Results included a slight lymphopenia with all other parameters on the CBC within normal limits while neurochemistry results showed an ALP increased 8 times that of normal with no other significant findings. Urinalysis showed 1+ protein, 1+ SSA, and 10-25 white blood cells per high powered field. The patient's pressure sore over the right elbow was clipped, cleaned, and lavaged on presentation before a culturette swab was submitted for analysis. An absorbent bandage was placed before the patient's subsequent transfer to the surgical department. Abdominal radiographs and ultrasound were elected for by the owner to rule out any liver abnormalities before surgery. Abdominal radiographs showed rounded liver margins and a slightly enlarged liver. Abdominal ultrasound revealed rounded liver lobe margins, choleliths, and nephroliths with one small, irregularly marginated region of the right liver that was aspirated for cytology. Cytology results revealed only mild lipid and glycogen accumulation with adequate cellularity, and no atypical cell populations.

Overall, the patient was declared healthy for surgery and surgical options were discussed with his owner as medical management had not been successful. An elbow flap fold was the option selected and surgery was performed the next day. The right axilla was examined to determine the best possible donation site before the wound on the caudal surface of the elbow was flushed with sterile saline and the edges of the wound were freshened with Metzenbaum scissors. The donor site for the elbow fold flap was marked on the skin in the axillary region with a sterile skin

marker. The wound was scraped to remove any remaining debris and the outlined skin flap was incised before the flap was undermined from the underlying tissues. The elbow fold flap was then rotated on its base to cover the exposed wound. A 7mm Jackson-Pratt (JP) drain was then placed underneath the flap and a suction bulb was attached. The donor site was then held together using 2 towel clamps while the JP drain was secured to the skin with a fingertrap suture. The flap covering the elbow and the donor site was sutured using a buried, simple interrupted subcuticular pattern. The skin was closed with medical grade skin staples and the surgical site was covered using sterile Telfa pads and Sure Site adhesive dressings.

While recovering in the intensive care unit after surgery, the patient was maintained on gabapentin and Tylenol 4 for pain control and trazodone for anxiety. While awaiting culture and sensitivity results, the patient was also given 200mg cefpodoxime once daily for three days. Culture and sensitivity results returned revealing a *Pseudomonas aeruginosa* infection sensitive to amikacin, gentamicin, imipenem, and ticarcillin. An update was given to the owner and permission was given to proceed with appropriate antibiotic therapy. As the bacteria was determined to be most susceptible to amikacin, 15mg/kg (2.1mLs) amikacin injections were given subcutaneously daily for 9 days and an intravenous catheter was maintained in an effort to avoid renal toxicity by providing maintenance fluid therapy and urinalyses were collected every two to three days to monitor for renal tubular cast formation, an indication of antibiotic nephrotoxicity. The patient's drain was monitored every 4-6 hours until fluid production from the site had decreased to less than 1.0mL/kg/day and the drain was pulled 10 days post-operatively when fluid production reached 0.6mL/kg/day. For padding and increased protection of the area, thick bedding was maintained in the patient's kennel and a padded bandage was made using pipe insulation. The bandage consisted of a dual layered antebrachial pad to elevate

his incision site and reduce pressure. A piece of tissue approximately 2cm in diameter at the tip of the surgical site necrosed 6 days post-operatively and was sharply debrided while the patient was under heavy sedation. This surgical site was then sutured with buried, simple interrupted subcuticular sutures and the skin was closed with medical grade skin staples. There were no other complications while in hospital.

Discussion:

Pressure sores are a result of continuous pressure (compression), shear forces, or frictional forces acting on the skin over a period of two or more hours' time that will eventually affect deeper tissues such as muscle and bone.[2] These sores take the form of skin ulcerations that form after such prolonged mechanical forces collapse capillary beds. This results in local ischemia which causes cell death at the affected site.[1] Compression of capillaries not only removes the oxygen supply from tissues, it also releases platelets and platelet products such as thromboxane. A study performed in 1989 looking at the amount of thromboxane present in pressure sores revealed that thromboxane may exacerbate ischemic insults as a result of its platelet aggregation and vasoconstrictive properties.[5] Thromboxane was elevated in the center of early sores and around the edges of later stage sores. These locations correlate with the areas of more intense tissue damage in early and late stage pressure sores.[5]

As pressure sore development is multifactorial, there may often be a problem microscopically before it is recognized clinically. The stages of histopathologic pressure sore development are as follows: blanching erythema, non-blanching erythema, decubitus dermatitis, early ulcer, healing ulcer, chronic ulcer, and black eschar/gangrene.[1] The first two stages deal with dilated capillaries from red blood cell engorgement, ultimately leading to hemorrhage. They can be the most difficult stages to identify on physical examination as there are few macroscopically visible

changes. The decubitus dermatitis stage shows either diffuse or focal eosinophilia with either epidermal atrophy and subepidermal blister or a normal looking epidermis with a subepidermal blister.[1] An early ulcer will look the same as the decubitus dermatitis stage except that there is no epidermis present. It includes all of the previously mentioned stages with the addition of mononuclear and acute inflammatory cell penetration.[1] A healing ulcer will show classical signs of healing, including granulation tissue and neovascularization, whereas a chronic ulcer will display a fibrotic dermis, acute inflammatory cell infiltration, and is typically encrusted in red blood cells.[1] The black eschar is full thickness skin necrosis where the skin is hard, black in color, and lacks sensation. The surrounding tissue is crusted and the epidermis is entirely absent. There may also be necrosis of inflammatory and red blood cells.[1]

Of the several clinical classification systems for pressure sores in human medicine, the National Pressure Ulcer Advisory Panel system is the most commonly accepted.[1] It consists of four stages that describe the depth of a visible ulcer. A stage one ulcer shows intact skin with signs of impending ulceration. It encompasses the first two histopathological stages and clinical signs include skin that is warm to the touch and skin thickening over the affected site.[1] Stage two ulcers are shallow. They may appear more like an abrasion or blister. Both stage one and stage two ulcers are easily reversible with medical management and have few side effects on overall health and well-being of the patient.[1] Stage three ulcers consist of full thickness skin loss with damage to the subcutaneous tissues, but show no damage to the underlying fascia. They may be necrotic and odiferous on presentation.[1] At this point, a pressure sore is a more obvious open wound and must be managed as such. Stage four ulcers are the same as stage three ulcers, except that the damage has penetrated through to the underlying fascia, muscle, or bone.[1] The patient discussed above presented with a stage three ulcer.

Risk factors allowing pressure sore development are varied. They are most commonly seen in geriatric or ill patients as these are the populations most likely to be still for two or more hours at a time. Other risk factors include poor body condition, diabetes mellitus, vascular disease, paralysis, and wearing a cast or splint.[1,2,3] Pressure sores can develop anywhere, but are more commonly seen over bony prominences where there is increased pressure inside and outside of the body.

Once a pressure sore, or any opening into the body, develops, bacterial infection is quick to follow. This, along with continued mechanical forces on the site, results in delayed or even non-healing pressure sores. Ideally, the diagnosis of infection is simple and is based on the classic clinical signs of infection: redness, heat, pain, swelling, and loss of function. Typically, a red border is seen around sore and the area is warm/hot-to-the-touch and may be tender-to-the-touch.[6] Systemically, there would be leukocytosis or even an elevated temperature. However, inapparent infections may also occur. Inapparent infections show little to no classic clinical signs of infection and can be difficult to discover.[6] Inability of the sore to heal, dehiscence after surgical closure, and healing of the sore after anti-microbial therapy are all suggestive of an inapparent infection.[6] Bacterial culture is strongly recommended to determine the best treatment modality. Samples should be taken from the surface of the sore as well as from deep in the sore to get a good representation of all bacteria present. If available, a sample of any purulent debris should be collected. Samples for culture may be a culturette swab, punch biopsy, or fine-needle aspirate.[6]

Some treatment modalities are also useful as prevention modalities and will be discussed in more detail below. These include providing thick padded bedding, changing position, and removing pressure from bony prominences via the use of slings, activity, or carts.[3] This focuses on the

most important aspect, which is to not develop more sores and to not further develop the current sore. This method of treatment is often all that is needed for a stage one or stage two pressure sores as described above.[7] Other treatments start with managing the sore as a wound. Wound management is the same as what is done for any open wound presented to a veterinary hospital: clip, clean, copious lavage, culture, cover, and potentially close.[7] Clipping the area around the sore removes fur that could track bacteria and other debris in, as well as defining the extent of the sore. Sterile lube should be placed in the sore before clipping to protect it from further contamination. Cleaning the surrounding skin is done with a cleansing agent that is active in organic debris, such as diluted chlorhexidine.[7] Large pieces of debris are removed at this time. Lavage is important to remove smaller debris and contaminant bacteria that may affect culture results. Lavage is performed with warm sterile solutions such as saline, balanced electrolyte solutions, 1% povidone iodine, or 0.05% chlorhexidine solution.[7] Tap water may be used in emergency situations to decrease gross contamination, but is not recommended as it can delay healing. Lavage must commence for a minimum of 2-3 minutes at 7-8psi. This psi can be obtained using a fluid bag cuff or with a 35mL or 60mL syringe capped with an 18-gauge needle.[7] Culture takes place next, as previously described, with samples collected from superficial and deep portions of the sore. At this time, debridement occurs.

Devitalized tissue may be removed surgically, where the exposed tissue is removed in layers until apparently healthy tissues are revealed or en bloc where the entire sore is excised from the surrounding healthy tissue. Debridement may also be achieved chemically, with compounds such as Granulex V that break down necrotic tissue and bacterial biofilms, or mechanically where tissues and foreign material are removed with the primary bandage layer after placing a dry-to-dry or wet-to-dry bandage.[7] Once debridement is over, the sore is then covered and

possibly closed. Options for cover include many types of bandages all of which allow for cleanliness, edema and hemorrhage reduction, comfort, absorption of wound secretions, and an acidic environment. This acidic environment increases oxygen availability to the wound tissues and absorbs ammonia released by bacteria.[7]

Antibiotics and treatments to encourage wound healing are also applied to the sore. Antibiotics can be topical or systemic, depending on likelihood of infection and bacteremia. Antibiotics may be added to lavage solutions such as with penicillin, ampicillin, and the cephalosporins or may be utilized as ointments.[7] Topical antibiotics are preferred over antiseptics for multiple reasons. Topical antibiotics are effective while in organic material, are selectively toxic to bacteria only, and provide combined efficacy if systemic antibiotics are chosen. [7] Specific topical therapies include triple antibiotic ointment containing bacitracin, neomycin, and polymyxin, silver sulfadiazine cream, nitrofurazone cream, and cefazolin. These are chosen based on the type of wound and clinician preference. Wound-healing enhancers are beneficial in encouraging the wound to heal. Again, these are chosen based on wound type and clinician preference, but there is a lack of information on which are most beneficial in the veterinary species.[7] These enhancers include gels containing acemannan to promote cytokine production and growth factors, D-glucose polysaccharide as a bacteriostatic and antibacterial, and tripeptide-copper complex ointments to attract mast cells and macrophages that encourage angiogenesis, cytokine production, and growth factor production.[3] Honey, aloe vera, and sugar may also provide assistance with wound healing and antibiotic therapy.[7] Systemic antibiotics are used based on culture and sensitivity testing. If a systemic infection is suspected, it is best medical practice to start treatment with a broad-spectrum antibiotic while waiting for culture and sensitivity results.

Once culture and sensitivity results are received, then the patient may be started on an antibiotic the bacteria is most susceptible to.

Closure is performed surgically with primary, delayed primary, or secondary closure occurring if the wound is not allowed to heal by second intention. Primary closure occurs when the wound is closed immediately upon presentation after initial wound management. Delayed primary closure occurs one to three days later.[7] Originally, the patient described had a sore which was closed with secondary closure, or closure after the formation of granulation tissue, as it was failing to heal via second intention. This was most likely done by freshening the granulation bed and sore edges, cleaning the sore, and suturing it closed. Other methods used to close wounds are medical grade skin staples and tissue adhesives, but these were not opted for in the patient's case. A bandage was then placed and changed as needed. Unfortunately, if wounds are infected, as in the patient's case, closure of the wound may fail. Post-operative complications include inflammation, edema, seroma or hematoma formation, necrosis, failure to heal, and dehiscence.[7] Failure to heal was seen with our patient after secondary closure of his sore, so a more advanced surgical procedure was attempted.

The next stage of pressure sore treatment when simple closure of the sore fails or is unachievable, is surgical closure via a skin flap. A skin flap allows blood flow to be maintained in the transposed skin. In this case, an elbow fold flap, a specific type of transposition flap, was used. This flap of epidermis and dermis was elevated from the underlying tissue, then rotated to cover the pressure sore. These flaps maintain their blood supply at the attachment point, also known as the base or pedicle, of the flap.[7] The size and length of these flaps will vary with body conformation and the margin of the defect to be closed.[7] Before the incision is made, granulation tissue is excised and wound edges are freshened. The amount of skin that can be transposed is

determined by tenting the skin to estimate how much flap can be harvested while still being able to close the donor site. A measurement is also made from the proposed pivot point of the flap to its far corner. This is compared to a corresponding measurement to the far corner of the wound to ensure the proposed flap will cover the wound and the donor site. A general outline of the incision is made with a sterile skin marker before incising.[7] The flap is elevated off the triceps muscles and transposed into place. The skin edges are then apposed with buried subcuticular sutures and medical grade skin staples or skin sutures are added for additional security.[7] Complications from flap procedures are similar to those of simple closure. Inflammation, edema, seroma or hematoma formation, necrosis, failure to heal, and dehiscence are all seen. Due to the dead space created, and because taking sutures are not used, drain placement is recommended to help prevent seroma formation. In this case, a Jackson-Pratt drain was placed and secured using a fingertrap suture pattern. Drains may be removed once discharge becomes serosanguineous and is ideally one-quarter (or less) than the original amount produced or less than 1.0mL/kg/day.[7] However, these ideals may not be achieved, and drains may be removed once fluid production has reached a stable plateau for several days. Drains should be covered with bandages at all times as they are conduits for infection into the body.[7] As our patient developed necrosis at the tip of the flap site over his elbow, the necrosed tissue was sharply debrided and simple closure of the wound was performed with buried subcuticular sutures and medical grade skin staples while the patient was under heavy sedation.

Pressure sore prevention is all about relieving compression, shear forces, and frictional forces. For recumbent patients, soft padding such as thick egg-crate foam that will support their entire body covered in a non-slip surface is recommended.[3] Changing position in the animal every 2 hours from left lateral to sternal to right lateral will also help prevent prolonged compression.[3]

Encouraging the patient to rise and walk multiple times will also eliminate these prolonged forces, and use of slings for 2-4 hours has been found to be beneficial.[1,3] Sheepskin overlays are the recommended non-slip covering. They not only provide a non-slip surface to prevent shearing and frictional forces, but they also wick away moisture to keep the skin dry and clean which assists in avoiding bacterial infection.[3,4] Maintaining adequate nutrition and ideal body mass will further assist in these endeavors.[1,3]

Case Outcome:

At the time of discharge, the patient was doing well and his renal values were excellent with no sign of renal tubular casts. At his recheck for staple removal the patient's incision was healing wonderfully and there was no evidence of tissue necrosis. The patient's owner has been adjusting to the patient's recommended weight loss diet and is keeping the patient well-padded at home with a DogLeggs sling and an orthopedic mattress. The patient is currently seeing the MSU-CVM Community Veterinary Services department for his routine care, where he was recently diagnosed with hypothyroidism. The patient has been doing well at home as of his most recent visit and is now a body condition score of 7 out of 9, having lost 12 pounds since his initial presentation.

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