
REPTILES – BONES – CLINICAL CONSIDERATIONS

Douglas Mader, MS, DVM, Dipl ABVP (C/F, R/A), Dipl ECZM (Herpetology)

Marathon Veterinary Hospital, 5001 Overseas Hwy, Marathon, FL 33050 USA

ABSTRACT

Introduction

Conditions affecting the bones of reptiles are commonplace. Whether it is iatrogenic or traumatic, or a combination of both, orthopedic pathology is a frequent reason for a visit to the veterinarian. Understanding the genesis of the pathology helps manage the condition and ensure rapid and complete resolution.

Metabolic Bone Diseases

Metabolic bone diseases (MBD) are common in captive reptiles. MBD is not actually a single disease entity, but rather a term used to describe a collection of medical disorders affecting the integrity and function of bones. There are many different metabolic bone diseases that affect both animals and people.

Historically, in the reptilian literature, any pathology affecting the bones of reptiles has been haphazardly called MBD. It is imperative that the old, incorrect nomenclature be dropped from the literature and vocabulary of reptile practitioners.

The following is a brief overview of several well documented MBD's in reptiles.

Nutritional Secondary Hyperparathyroidism (NSHP)

Metabolic bone disease of nutritional origin (nMBD), which is the most common type of MBD that affects captive herpetofauna, is a consequence of dietary and husbandry mismanagement. Several factors combine to cause a prolonged deficiency of calcium and/or vitamin D, an imbalance of the calcium to phosphorus ratio in the diet, lack of exposure to direct, unfiltered natural sunlight or combinations thereof.

Nutritional Secondary Hyperparathyroidism (NSHP) is the technical name for the MBD of nutritional origin that is commonly seen in captive herpetofauna. With NSHP there is an excessive production of parathyroid hormone (PTH) from the parathyroid gland in response to hypocalcemia. Calcium is then resorbed from the bones and transferred to the extracellular fluid. This results in an increase in serum calcium (but not necessarily hypercalcemia), but the consequence is a weakening of the bones. If this occurs in a young, growing animal it is called rickets, and if it occurs in an adult it is referred to as osteomalacia.



External symptoms of the chronic form of the disease correlate with the insidious physiologic changes occurring within the animal's body. The body compensates for deficiency in serum calcium by mobilizing calcium from the bones. The bones most commonly affected are the long bones (legs or ribs), the mandible (lower jaw), the flat bones of the skull, and lastly, the bones of the spine (vertebrae).

As the condition progresses, calcium salts in the bone are replaced by softer fibrous connective tissue (fibrous osteodystrophy). This results in pliable bones. Presumably, tension exerted on the jaw by muscles of the tongue deforms the lower jaw, resulting in the common "smile" appearance.

Differentials must include RSHP, HO, osteomyelitis (bacterial and fungal) and disuse osteomalacia.

Renal Secondary Hyperparathyroidism (RSHP)

Hyperphosphatemia is the hallmark of renal secondary hyperparathyroidism, a consequence of chronic renal disease (rMBD). The hyperphosphatemia is associated with reduced calcitriol levels, soft tissue calcification, renal osteodystrophy and hypocalcemia.

Phosphorus is absorbed from the gastrointestinal tract and eliminated via the kidneys. Excretion of phosphorus is a sum of glomerular filtration and tubular resorption. In renal failure, decreasing filtration rate leads to phosphorus retention and hyperphosphatemia.

Calcitriol, the most active form of vitamin D in mammals, is formed by renal hydroxylation of 25-hydroxycholecalciferol. This hydroxylation reaction is promoted by parathyroid hormone (PTH). The elevated phosphates have a negative effect on the hydroxylase activity in renal tubular cells. In turn, since elevated calcitriol normally has a negative feedback effect on PTH production, this decreased calcitriol formation, which results from the hyperphosphatemia, promotes RSHP, and osteodystrophy.

Phosphate retention also decreases extracellular calcium due to the mass law equation. In addition, the decreased production of calcitriol further limits absorption of calcium from the intestinal tract. These changes result in low normal or low serum calcium levels.

Differentials must include NSHP, HO and osteomyelitis (bacterial and fungal).

Hypertrophic Osteopathy (HO)

Although not common, HO has been reported in lizards. In mammals HO, formerly called Hypertrophic Pulmonary Osteoarthropathy, or Hypertrophic Osteoarthropathy, is characterized by lameness, painful limbs and reluctance to move. Pulmonary pathology has been associated with this condition in greater than 90 percent of the cases.

Radiographic signs consist of extensive periosteal proliferation beginning in the distal long bones, progressing proximally (digits proximal to the humerus or femur). The pathogenesis is unknown

but theories include chronic anoxia, toxins, and complicated neurologic pathways involving the vagus nerve.

In mammals, once HO has been diagnosed the condition is usually terminal. If an identifiable thoracic mass is found resection may result in temporary resolution of the clinical signs, which may take several months to regress.

Differentials must include NSHP, RSHP, gout, tumoral calcinosis (pseudogout), and osteomyelitis (bacterial and fungal).

Osteopetrosis (OP)

This is a rare hereditary disease in humans. Two forms, one an autosomal recessive and the second an autosomal dominant, cause excessive thickening of the bones. The bones become radiographically dense, eventually obliterating the marrow cavity. The cause is not known, but is believed to be an inability to resorb bone in a normal fashion. Since the marrow cavity is destroyed, the patients become anemic. Nerve foramina in the skull become diminished which leads to blindness and hearing impairments. The bones become brittle and fracture easily.

Cases of osteopetrosis in reptiles in this author's practice (unpublished), made by a radiologic diagnosis only, have had similar clinical presentations.

In birds, similar signs are seen secondary to avian leukosis virus infection. A parallel situation in reptile patients has not been identified but has been suggested in earlier literature.

Differentials must include NSHP, RSHP, HO, osteomyelitis (bacterial and fungal), excessive dietary supplementation of vitamin D and/or calcium and inappropriate nutrition.

Paget's Disease (PD)

A PD like condition has been reported numerous times in the reptilian literature. In humans, this condition, also known as osteitis deformans, results from repeated cycles of bone resorption and deposition. The bone eventually becomes dense and brittle. Pain and pathologic fractures are common.

In people, many cases are asymptomatic. The diagnosis is made through radiographs, physical examination and laboratory testing. The cause is not known, but, research at the NIH suggests the possibility of a slow virus being involved. Genetic influence may also play a role.

The name "Paget's Disease" has been used to describe the lesions seen in reptiles for many years. Current opinion and recent studies suggest that this may be an inappropriate term because it is based on the mosaic appearance of the bone changes in humans. In reptiles, this mosaic pattern of bone growth may be a normal feature. Additionally, many snakes with this disorder have active inflammatory changes from which bacteria have been isolated, such as *Salmonella*, *Klebsiella*, *Morganella* and *Providencia*, and bacterial osteomyelitis may be a more appropriate term.



Differentials must include gout, fibrous osteodystrophy, trauma, tumors (usually solitary) and tumoral calcinosis. Lesions so noted in reptile patients should be properly evaluated rather than just assumed to be Paget's Disease.

Fractures: Causes and Practical Therapy

Fractures in captive reptiles are common, usually being secondary to one of the MBDs previously discussed. Primary nutritional deficiencies such as Metabolic Bone Disease of Nutritional origin (nMBD) is by far the most common. Specifically, pathologic fractures frequently occur as a result of Nutritional Secondary Hyperparathyroidism (NSHP), which is a general lack of dietary calcium, excessive phosphorus or deficiency in exposure to ultraviolet light/vitamin D3. Even traumatic fractures, which under normal conditions with healthy bones would not occur, are more likely due to the generalized osteopenia associated with NSHP.

Extremity fractures are rarely compound or comminuted. As a result, most fractures are readily treated with external coaptation. In addition, since most fractures are often associated with demineralization and softening of the bones, internal fixation is usually not indicated. In the unlikely event of a traumatic fracture involving normal bone, internal fixation can be utilized.

Regardless of the etiology, nutrition and diet should be thoroughly evaluated in all fracture cases. Before attempting any repair calcium homeostasis should be established. The medical management in these cases is equally as important as the surgical attention.

Overview

Frye states that most fractures occur as a result of low impact forces, thus making the incidence of comminuted fractures uncommon. In addition, due to their relatively inelastic skin, open or compound fractures are infrequent.

Little information is available on fracture healing in reptiles. No controlled studies have been conducted. Most of the information that is known comes from anecdotal reports relating treatment successes/failures in cases of NSHP. It is generally accepted that reptilian bone heals slower than either mammalian or avian bone, requiring from 2-18 mo to completely heal.

When planning fracture repair in reptiles, general principles of orthopedic management apply. Proper alignment, rigid stabilization, minimal disruption of soft tissue and conservation of the blood supply is paramount. The forces acting on the fracture (bending, rotation, compression and shear) must be evaluated and neutralized to promote rapid healing. In general, the more forces that must be neutralized by the type of fixation, the higher the incidence of complications and failures.

Additional considerations when deciding upon type of fracture repair include the patient's functional requirements (pet lizard in a terrarium vs. a Komodo dragon being returned to the wild), cost limitations set forth by the client, the cost and availability of the required materials and the experience of the veterinarian.

Most long bone fractures will heal in time with nothing more than strict cage rest. Although there may be some severe malunions, these complications do not seem to affect captive reptiles in an adverse manner.

The size of the patient and its nutritional state may have a direct impact on the type of fixation required. Large, heavy bodied lizards and turtles may require internal fixation, whereas small, delicate lizards may do well with a light splint.

The general condition of the patient often plays a major factor in the selection of fixation methods. In many of these NSHP animals it is physically impossible to utilize any type of internal fixator, as the bones just are not physically strong enough for the implant to gain purchase.

As in anything in veterinary medicine, the dollar is often the deciding factor in final determination of fixation technique. Internal fixation carries a higher price tag due to the cost of the materials, the time necessary for application and the training of the surgeon. Although internal fixation may be the best for the patient, it is not always an option.

External Coaptation: Minimal Intervention

External coaptation involves the use of splints, slings, casts and any other technique needed to immobilize a fracture. This is by far the most commonly utilized technique in reptilian fracture repair. In general, the best splints/casts are those that are lightweight and comfortable for the patient. If the patient's activity is restricted lightweight splints/casts are effective.

When treating pathologic fractures secondary to nutritional disease external fixation is the treatment of choice. NSHP is the most common disease presenting to reptilian veterinarians, and most frequently seen in the Green iguana (*Iguana iguana*).

Bone is a dynamic organ, undergoing constant remodeling. During prolonged hypocalcemia/hypovitaminosis D, the mineralization process lags behind the formation of organic bone matrix, resulting in the formation of hypomineralized bone. When this occurs in young, growing animals it is called rickets, and in adults, it is known as osteomalacia. Pathologic fractures occur when the calcium content decreases to approximately one-third of its baseline. Aside from pathologic fractures of the long bones and appendicular skeleton, soft, swollen mandibles and long bones (fibrous osteodystrophy), stunted growth, deformed heads and abnormalities in ambulation are common.

These bones are too soft to provide support to the implants used in internal fixation techniques. IM pins, cerclage wires and bone screws all penetrate, crush and pull out when used in these wax-like bones. An IM pin may be utilized for alignment in long bone fractures, but when used, it should be in conjunction with external coaptation.

Once the calcium homeostasis is corrected the healing progresses rapidly, with a bony callous forming in about three to four weeks. Correcting management and husbandry deficiencies and providing proper dietary and supplemental calcium is needed. In addition, treating the patient with



synthetic salmon derived calcitonin helps speed recovery by inhibiting the actions of parathyroid hormone, blocking the actions of the osteoclasts, stimulating the osteoblasts and providing bone analgesia. 50 IU/kg of calcitonin, IM in the triceps, administered q 1 week for two treatments is the recommended dosage. It is important that the patient is eucalcemic prior to the administration of the calcitonin.

There have been numerous methods reported in the literature for external coaptation in reptiles. There is no one right way. Whatever technique works best in your practice situation is the best method to use. The most important thing to remember is that the best splints/casts are the lightest and most comfortable to the patient.

When applying external coaptation remember that the patient is most likely in pain. Anesthesia or sedation is recommended for patients that struggle or if extensive manipulation of the fracture(s) is required.

The initial padding around the limb can be performed with many different types of bandage material (Specialist Cast Padding, Johnson & Johnson, New Brunswick, NJ; Conform, Kendall Co., Boston, MA). Make sure that the padding is cut to the appropriate width to prevent bunching of the padding around the joints.

Tape stirrups should be incorporated into the padding when applying the splint/cast to prevent slippage. It is not uncommon for the splint/cast to slide down the leg after the cast padding compresses.

This padded limb can now be reinforced by adding aluminum rods, tongue depressors and light weight casting material. It is important to conform the shape of the splint/cast to the natural angles of the limb. This will prevent the development of fracture disease, or periarticular fibrosis, in the immobilized joints.

There are several types of veterinary Thermoplastic such as X-lite (Allard USA, Rockaway, NJ) and Orthoplast (Johnson & Johnson, New Brunswick, NJ) are rigid at room temperature, but malleable when heated in a water bath. The Veterinary Thermoplastic is easy to apply when heated and cools to make a rigid splint. It comes in different sizes and thicknesses, making it convenient for different size patients.

Splints/casts can be easily applied to any of the long bones in lizards. When applying splints/casts it is important to follow general principles of fracture stabilization. The joints both proximal and distal to the fracture should be immobilized.

For both humeral and femoral fractures a modified Spica-type splint must be used. The splint should incorporate the distal joint, and then have a portion that crosses over the body. For the femur, the band should cross cranial to the vent so that it does not interfere with elimination. In humeral fractures, the band can cross diagonally across the chest, passing between and under the front legs.

Chelonians can also be splinted, but modifications in technique are required. It is usually not possible to apply a splint to a proximal long bone (humerus/femur). These bones can be reduced (with sedation/anesthesia as needed) and then taped into the leg opening in the shell. I recommend covering the limb with cast padding to add stability to the "set limb" before taping over the opening. I also recommend taking a radiograph of the leg folded up within the shell to make sure that fracture alignment is appropriate.

Splints/casts do not provide rigid fracture fixation. As a consequence, fracture healing is not as rapid as it would be with a plate or external fixation device. However, the bone will heal.

I recommend re-checking the fit of any splint/cast within one week of the initial application. You should always check for slippage, swelling of the distal extremities and pressure sores. Splints/casts are usually left on for a minimum of four, and usually six to eight weeks. Follow-up radiographs should be taken at four weeks, and again when the cast is removed.

Internal Fixation

Internal fixation is warranted for long bone fractures in reptiles where external coaptation is not a practical option. Large, heavy, active and otherwise healthy reptiles all do well with internal fixation. Internal fixation techniques utilized in mammals and approaches to the long bones are similar to those employed in reptiles.

Steinmann pins, Kirschner wires, spinal needles and stylets can all be used as IM pins in reptiles. In addition, these devices can all be used as parts for External Skeletal Fixation (ESF). ESF can be used in a variety of fracture types in reptiles of all sizes.

When using these delicate implants as a part of the ESF, the external connecting bar and clamps are replaced by a methylmethacrylate polymer. This is inexpensive, easy to use and light.

Pin loosening is a common problem with ESF. Whenever possible it is recommended to use threaded pins. The threads should be applied to the outside of the pin, not cut into it.

Bone plating can be utilized, but in general requires a larger patient. Cuttable plates (DePuy Synthes Vet, PA) with 1.5 mm diameter screws can be applied to bones as small as 3 mm diameter. Finger plates are also applicable in certain situations.

In general, plates do not need to be removed. IM pins and ESF should be removed when there is radiographic evidence of bone healing. In some cases a fibrous union may be all that is needed to ensure eventual healing, thus allowing the removal of loose pins as needed.

Salvage Procedures: Amputation

When there is severe tissue trauma, loss of blood supply or granulomatous infection in the limb, fracture repair may not be a viable option. Amputation of either the fore- or hind limbs is a viable option in reptiles, as they do quite well with three limbs. Amputation of digits or limbs can be accomplished with excellent cosmetic and functional results.



Digits should be amputated at their base. The plantar/palmar flaps should be longer than the dorsal flaps so that the incision is sutured above the substrate. This keeps the incision clean.

When amputating limbs it is best to remove the entire appendage. Disarticulation at either the scapulohumeral or coxofemoral joints is recommended. Limb muscles are transected distally and then elevated proximally. The joint is exposed and the limb removed. The muscle bellies are then sutured over the joint space to provide soft-tissue padding. Nerves can be transected with a scalpel and injected with bupivacaine to provide local analgesia post-operatively.

In chelonians after a limb amputation it may be necessary to provide some sort of prosthesis. A block of wood, a plastic skid or a furniture roller can be glued to the plastron to aid in locomotion.

ACKNOWLEDGMENTS

Special thanks to Mike Garner for his assistance with this manuscript.

