DOWNLOAD PDF METABOLIC BONE DISORDERS

Chapter 1 : Osteoporosis & Metabolic Bone Disease | Cleveland Clinic

Metabolic Bone Disorders Metabolic bone diseases are disorders of bone strength, usually caused by abnormalities of minerals (such as calcium or phosphorus), vitamin D, bone mass or bone structure. The most common metabolic bone disorder is osteoporosis.

Start oral P supplements as soon as its feasible. Bone mass density; Ca: Tubular reabsorption of phosphate. Serum biomarkers As a normal serum Ca level can still be maintained to the detriment of Ca loss from the bone, it should not be used to screen infants at risk. Furthermore, serum Ca may also be affected by unrelated conditions such as hypophosphataemia[16 , 21]. Serum P concentration is correlated with BMD, is highly specific but is not sensitive enough to identify infants with ostepenia. While serum P concentration adequately reflects P levels in the bone, serum Ca concentration remains maintained at the cost of Ca content in the skeleton. Serum alkaline phosphatase ALP is a marker of bony turnover. The diagnosis of MBD in the preterm infant is usually suggested by the presence of low serum P levels in association with elevated serum ALP levels[1]. A serum ALP level exceeding five times the upper limit of the normal range in adults is also associated with an increased risk of rickets[25]. Because it is located on osteoblast surfaces, bone-specific ALP is a more specific biomarker of bone turnover, useful to confirm OOP, when high levels of total serum ALP are found[28 , 29]. Despite its limitations and, despite the absence of a clear cut-off diagnostic level, serum ALP measurement is frequently used to screen high risk infants for MBD. It is a readily available measurement in most laboratories and serial serum levels provide a trend very useful for follow up. Using it in conjunction with serum P levels as a screening tool significantly increases the sensitivity of identifying infants at risk of MBD. Serum osteocalcin OC , a non-collagenous protein of the bony matrix, is also a biomarker of osteoblastic activity. It is synthesized by osteoblasts and is partly regulated by 1,dihydroxyvitamin D levels. Its serum concentrations are elevated whenever bone turnover is increased, making it a possible useful tool to diagnose OOP[1]. However, despite its specificity, there is no correlation between serum OC levels and BMC in the first four months of age[30]. Urinary biomarkers Urinary Ca and P excretion have also been used as biomarkers of postnatal skeletal mineralization. Urinary excretion of Ca exceeding 1. Infants born between 23 and 25 wk of gestation have a significantly lower renal P excretion threshold than other preterm neonates, resulting in elevated urinary P excretion even when serum P levels are low[31]. Similarly urinary Ca or P to creatinine ratios may also be useful as biomarkers for OOP; normal reference ranges in preterm infants have already been established for these ratios[33 , 34]. However these urinary ratio results need to be carefully interpreted as they are highly dependent on the dietary intake resulting in uncertainty if the standard reference range and are also affected by the administration of drug such as furosemide or theophylline[35]. It correlates well with fracture risk and, in both term and preterm infants, it can be used to estimate BMC[37]. In addition, the establishment of robust, reliable neonatal, ethnic and gender specific normograms is urgently needed. Barriers to the routine use of DEXA as a screening tool for OOP include its high cost, its limited availability, the dimensions of the equipment used, the lengthy time required for imaging, as well as its sensitivity to movement artifact. Quantitative Ultra sound QUS , with already established reference values for both preterm and term infants, is a new inexpensive and portable modality of investigating OOP[38 - 40]. This simple, non-invasive and inexpensive bedside method measures the broadband ultrasound speed attenuation, and is usually performed on the tibia. Although the measurements it provides correlate well with bone density and structure, the association is a poor with the thickness of the bony cortex[41]. Although ultrasound reference values are available for term and preterm infants, there is limited information showing its usefulness. The prevalence and also the severity of OOP can be reduced by early nutritional intervention. Supplementing milk with both Ca and P is more effective: Ca and P retention rates similar to those observed in utero are attained with high-mineral preterm milk formulae or with fortified human milk[45]. To prevent OOP, serum Ca concentration should be maintained between 2. Parenteral nutrition preparations providing 1. Ca and P delivery by parenteral nutrition are affected not only by their respective concentrations in the intravenous solution, but also by the ratio of their concentrations. The supply of these minerals to infants is limited by the

poor solubility of both Ca and P in parenteral nutrition solution, resulting in an increase in the risk of OOP when enteral feeding is not possible for an extended period. Further research is required to improve Ca and P delivery with parenteral nutrition. Vigilance is required during parenteral nutrition as the increase in parenteral mineral delivery may result in metabolic acidosis and hypercalciuria[52]. If needed, parenteral P delivery can also be enhanced by using special preparations of organic P. Because of the crucial role of mechanical forces on the development of the skeleton, daily exercises such as gentle compression and movements of the limbs are recommended in infants at risk of OOP if greater increase in body weight, forearm bone length, bone area and BMC are to be achieved[53 - 55]. If serum P levels fail to increase and if serum ALP levels keep on rising, ergocalciferol or alphacalcidol therapy should be then considered. The American Academy of Pediatrics recommends that all breast-fed, partially breast-fed and non-breast-fed infants consuming less than mL of vitamin D fortified milk daily should be supplemented daily with a minimum of IU vitamin D[57]. In addition, daily passive exercises should be encouraged and the medications in use should be regularly reviewed with discontinuation of diuretics and steroids when appropriate. Complications such as rickets and pathological fractures may be the first manifestation of the condition. To detect the early asymptomatic phases of impaired bone mineralization and allow early intervention, all neonates at high risk of MBD appropriate biochemical markers of insufficient intake minerals and of abnormal bone turnover should be regularly monitored. DEXA is being increasingly used for assessing BMD in neonates, but more studies are still needed before it can be used as a useful clinical tool. Prevention and early diagnosis of MBD are key to the successful management of this condition and oral P supplements should be started as soon as is feasible. Prospective studies of cohorts of preterm infants with OOP are needed with close long-term follow up for later outcomes. More research into urinary Ca and P to creatinine ratios is needed before they can reliably replace direct measurement of BMC. As the poor solubility of Ca and P in parenteral nutrition solution hampers the adequacy of their supply to the growing newborn, further research in this area is required to increase their delivery. The authors have no commercial, personal, political, intellectual, or religious conflict-of-interest to report in relation to this work. This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. January 20, First decision: March 6, Article in press: August 7, P- Reviewer: Gong XM L- Editor: Jiao XK References 1. 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Chapter 2 : Metabolic Bone Disease | UW Orthopaedics and Sports Medicine, Seattle

Metabolic bone disease is an umbrella term referring to abnormalities of bones caused by a broad spectrum of disorders. Most commonly these disorders are caused by abnormalities of minerals such as calcium, phosphorus, magnesium or vitamin D leading to dramatic clinical disorders that are commonly reversible once the underlying defect has been treated.

Lumps or hard bumps on the limbs or along the spine Softening of the shell in turtles Constipation How is metabolic bone disease diagnosed? The diagnosis of metabolic bone disease is based on the findings of the physical exam, and the diet and husbandry history of the animal. Radiographs x-rays may help assess the severity of the disease, determine if there are any fractures present, and follow the progress of treatment. How is metabolic bone disease treated? The diet and husbandry conditions that led to the development of metabolic bone disease must be corrected. Adequate and balanced levels of calcium, phosphorus, and protein must be provided. For herbivores, know that lettuce and fruits are generally low in calcium, so concentrate on giving your herp dark green vegetables that are low in oxalates. Usually a calcium supplement will also be necessary. For herps primarily eating insects, feed the insects with calcium-rich supplements prior to feeding them to your herp. In addition, they can be dusted with a calcium supplement. For carnivorous herps, remember that a meat only diet is extremely low in calcium. If possible, offer whole prey items that include the bones. Pinky rats and mice also do not contain sufficient calcium since their bones have not matured. Thus, calcium supplementation would be necessary. The reptile should be exposed to 12 hours of UVB light each day. Natural sunlight is best, but the UVB does not penetrate plastic or glass, so mesh screening is required between the light and the animal. Do NOT expose a reptile to direct sunlight while the herp is in a glass or plastic cage, as overheating can quickly develop. If exposure to sunlight is not an option, fluorescent lighting will work. The light should be positioned no less than 18 inches from the animal 12 inches is optimal. Bulbs should be replaced every 6 months. Again, make sure the light is not filtered through plastic or glass. Research the needs of your herp and talk to your veterinarian regarding the optimum cycle. The proper temperature gradient should be maintained. Again, research what is the optimal temperature gradient for your herp and discuss it with your veterinarian. Provide room for adequate exercise. If your herp has difficulty moving or your veterinarian says your herp has or is susceptible to fractures, you will need to restrict exercise at first. You may need to remove any branches your herp would attempt to climb. If climbing is restricted, remember your herp will not be as close to the basking light, so be sure to measure the temperature of the basking area to be sure it falls within the optimal gradient. In very mild cases, the above changes may be sufficient. In more severe cases, oral or injectable calcium supplements such as calcium glubionate NeoCalglucon , calcium lactate Calphosan or calcium gluconate are indicated. In addition, after the serum calcium level is normal, calcitonin may be prescribed. For severe cases, it may be necessary to tube feed the animal until he regains strength. How is metabolic bone disease prevented? Metabolic bone disease is very easily prevented by providing the correct diet, adequate exercise, and exposure to UVB light.

Chapter 3 : Metabolic Bone Disorders | Johns Hopkins Bayview Medical Center

Metabolic bone disease, any of several diseases that cause various abnormalities or deformities of bone. Examples of metabolic bone diseases include osteoporosis, rickets, osteomalacia, osteogenesis imperfecta, marble bone disease (osteopetrosis), Paget disease of bone, and fibrous dysplasia.

This is an article intended for use by licensed wildlife rehabilitators and others working on behalf of the opossum. If you have found an opossum and are trying to seek dietary advice then please go ahead and read this article. It will help you to understand why it is critical you turn the opossum over to a trained rehabilitator and not try to raise it yourself. Too many opossums have died or suffered painful, crippling disorders as a result of well-meaning but misinformed people attempting to raise the opossum themselves. See photo of opossum fed a diet consisting of only hot dogs and the resulting facial deformities. Unfortunately, this opossum responded to initial treatment and proper diet but later succumbed due to a complication of his advanced disease. It is sometimes seen by wildlife rehabilitators receiving an opossum from a well-meaning but misinformed finder. Often, a finder will not know what to feed the opossum and will offer an incorrect, grossly imbalanced diet. Once the finder notices that something is wrong with the opossum it can be too late unless the rehabilitator takes immediate action. The rehabilitator should always obtain a detailed diet history and suspect MBD if the opossum was fed a calcium deficient diet, an all-meat diet, or a commercial diet supplemented with meat off the bone, or if the diet history is unknown. Top of Page What causes the disease? MBD is a complex disease with many etiologies. However, it is primarily due to poor husbandry, feeding an improper diet. Since the rehabilitator will most likely encounter the dietary form of MBD, only the nutritional-induced MBD will be discussed. The most common dietary errors are not enough calcium in the diet, too much phosphorous, too much protein and too many fruits. The opossum must have a certain amount of calcium and phosphorous in the diet with a specific calcium to phosphorous ratio Ca: The desired ratio for the opossum is 1. If the opossum does not receive an adequate diet then dietary-induced disorders such as nutritional MBD may result. At present, there is no commercially available diet formulated for the opossum. Do not feed the opossum meat without bones. When an opossum eats a prey, it consumes the entire animal, bones and all. Top of Page Commercial cat and dog food alone are not adequate diets for opossums but will provide a balanced dietary staple and help prevent diet-related disorders. A commercially prepared diet can more accurately guarantee that the opossum will receive a certain level of nutrients and on a consistent basis. Opossums permitted to select and choose favored foods will not put together a balanced diet. In general, homemade diets are not recommended if a reputable commercially available product is available to serve as a dietary staple. If you do plan to use a homemade diet then please evaluate the diet for completeness. It is important not to over-supplement with any food, nutrient, vitamin, etc. Do not let this happen to the opossums in your care. Take the time to educate yourself about proper opossum nutrition. There are many opossum diets offered on the web and through different organizations. Some are acceptable diets and some are not. The OSUS diet has been proven to be a nutritious, balanced diet by a number of wildlife rehabilitators successfully raising and releasing opossums for over 25 years. If you have had success with a different diet and wish to use it then you may continue to do so. You are asking for trouble and an imbalance may result. If you find a diet that works for you then follow it in its entirety. Top of Page What happens? As stated above, MBD is a complex disease and far too complex to describe here in detail. Basically, a low calcium diet can result in a decrease in the calcium in the blood. The parathyroid gland secretes parathyroid hormone PTH in response to a decrease in plasma calcium concentrations. When there is a calcium deficiency, high concentrations of circulating PTH hyperparathyroidism result. PTH prevents plasma calcium from falling too low by increasing conservation of calcium by the kidney, enhancing the absorption of calcium from the intestine, and by stimulating calcium mobilization from the bone. This results in a release of calcium into the blood to maintain the blood calcium level but also weakens the bone as minerals are lost thus subjecting the bones to increase risk of fractures. A sustained hyperparathyroidism can result in reduced appetite, interstitial nephritis, and can be toxic to the cardiac and skeletal muscles as well as the central and peripheral nervous systems. What are the

signs? When first picking up opossums, obtain a detailed history from the finder. Knowledge of the dietary history will help you to recognize and correct MBD earlier. The rehabilitator can easily miss early signs. In the early stages days to weeks watch for a loss of grip in the hands or feet, tremors, twitching, decreased activity, sleeping more, uncoordinated gait, and a decreased appetite. You may notice as the disease advances that the opossum may have difficulty walking and climbing. It may take short, mincing steps, often with a hunched back. It may crawl or drag its limbs instead of walking. The limbs may appear chubby. Other skeletal deformities may be obvious. The bones at this stage are demineralizing and are extremely fragile. Jerky movements and tremors may become more pronounced. Pain will be present. Top of Page As the disease progresses further, the opossum may be unable to eat and drink. Changes in the skull occur resulting in an inability to use the mouth. The tongue may appear longer and protrude from the side of its mouth. Other organ systems may become affected, as the opossum gets closer to death. The rate of development of the disease is dependent upon the severity of the calcium deficiency and the age and health status of the opossum. How is MBD treated? If MBD is caught early enough then it can be treated and may be reversible. The diet must be corrected and supportive care offered. Feed a high quality, balanced diet with sufficient calcium and a proper Ca: Do not feed meat without the bone. Calcium is best obtained from the diet. Feed calcium-rich foods such as low or non-fat yogurt, kale, cabbage, collard greens, bok choy, etc. Calcium supplementation may be recommended in certain circumstances, especially advanced cases. Caution must be taken to avoid over supplementation. Too much calcium can also be bad. Consult your veterinarian, experienced rehabilitator, or OSUS for advice and dietary recommendations. In moderate to advanced cases make sure the opossum is not able to fall and hurt itself. Top of Page Make sure the opossum receives some exposure to sunlight for Vitamin D. They are nocturnal animals and receive Vitamin D in their diets but they also receive some exposure to sunlight while sleeping under woodpiles, dense shrubs, etc. Vitamin D is important for calcium metabolism. Make sure the opossum does not receive direct sunlight because it may suffer from the heat and may become sun burned. The opossum did not develop MBD overnight and it will not be corrected overnight. Recovery will take at least as long as the time it took for the disease to cause the damage. Recovery may or may not be complete depending on a number of factors including duration of the disease, diet, overall health, age, parasite load, and any other complicating factors. Many opossums caught and treated early enough can be releasable. Others treated at more advanced states may recover sufficient motion but not sufficient enough to be released. These animals can be placed in wildlife centers for educational purposes, if of the proper temperament. Severe cases may require euthanasia, in the best interest of the opossum. This is meant for opossums that are to be re-released into the environment, non-releasables diet must be adjusted after symptoms are reversed.

Chapter 4 : Bone disease - Metabolic bone disease | theinnatdunvilla.com

This is a short video on five bone diseases caused by altered bone metabolism. I created this presentation with Google Slides. Image were created or taken from Wikimedia Commons.

Metabolic bone disease The normal function of bone requires an adequate supply of amino acids the building blocks for proteins for the synthesis of collagen , the chief component of the organic matrix; of calcium and phosphate for mineralization of the organic matrix; and of other organic compounds and mineral elements. Also, growth, repair, and remodeling of the bone tissue require a precisely regulated supply of hormones, vitamins, and enzymes. Skeletal disease, when it is due to inadequacies in the supply or action of the above essentials, associated with abnormalities outside the skeleton , is termed metabolic; in such cases the entire skeleton is affected. Examples of such abnormalities are dietary deficiency and gastrointestinal, liver, kidney, and hormonal diseases. In addition, osteoporosis age-related loss of bone with tendency to fractures is traditionally included among the metabolic conditions even though its cause is not known. Changes in bone tissue due to metabolic abnormalities are classified with regard to the amount and composition of the bone tissue. When the amount of bone is lower or higher than normal, the conditions are termed, respectively, osteopenia and osteosclerosis. These terms do not imply any specific disease but simply describe the amount of bone present. Osteopenia is common both locally and generally throughout the skeleton. Localized osteopenia is evident in X-rays of tumours or infections of bone, in osteonecrosis death of bony tissue , in fracture , and in conditions of diminished mechanical demand. Osteopenia may thus be associated both with atrophy from disuse and with active remodeling of bone; it occurs when bone resorption occurs faster than bone formation. Generalized osteopenia occurs in osteomalacia , osteoporosis, and osteogenesis imperfecta. Osteosclerosis occurs locally in osteoarthritis , osteonecrosis, and osteomyelitis ; it represents an attempt at structural strengthening by thickening of bony trabeculae, but its X-ray appearance may be confused with that of dead bone, retaining its density while adjacent normal bone has become osteopenic. Widespread but hardly ever truly generalized, osteosclerosis occurs in osteopetrosis marble bone disease and in Paget disease. Except in the latter condition, however, osteopenia and osteosclerosis are not associated with detectable biochemical abnormalities. These diseases are characterized below. When the normal composition of bone tissue is altered by deficient mineralization of the organic matrix, the condition is called rickets in children and osteomalacia in adults. The mineralization deficiency is in part due to a lower than normal calciumâ€”phosphate ion product in the body fluids. In rickets the bones become tender, soft, and deformed; X-rays show characteristic abnormalities at the growth zones, especially evident at the wrist , knee , and ankle joints. In osteomalacia, bone tenderness and pain accompany the slow development of the spontaneous, often symmetric fractures characteristically present in the osteomalacic pelvis and thighbones. The X-ray appearance of osteomalacia is rather normal until visible fracture has developed. Biochemical abnormalities usually present in rickets and osteomalacia are increased blood concentration of the enzyme alkaline phosphatase , believed to be important for bone formation or resorption, and decreased blood concentrations of calcium or phosphate or both; the calcium concentration may fall to levels so low that muscle and nerve function is impaired tetany. Microscopic examination of the bone tissue reveals the deficient mineralization of the organic matrix. The entire skeleton is affected in both rickets and osteomalacia, although abnormalities are more evident in growth centres in children and in areas of maximal mechanical load in adults. Insufficient protein, caloric, and vitamin intake interferes with bone formation during growth and remodeling, directly because of an inadequate supply for matrix formation and indirectly because of a deficient production of crucial hormones and enzymes. The effect is stunted growth in the young and osteopenia in adults. Deficient intake of calcium or phosphate or both, unassociated with vitamin D deficiency, causes a compensatory action of parathyroid hormone whereby the mineral is mobilized from the skeleton with eventual development of osteopenia. Deficient calcium intake combined with excessive phosphate intake causes osteopenia, fractures, and loss of teeth in dogs, cats, and other animals by excessive compensatory parathyroid hormone action. Insufficient intake of vitamin D is one of many ways in which rickets may develop. The condition, once universally prevalent, is now rare in

countries that ensure adequate supply of vitamin D in fortified milk and healthy living habits, including adequate exposure to sunshine. Malabsorption of calcium and vitamin D causes a mixture of osteopenia and osteomalacia and requires high intake of calcium and vitamin D. Parathyroid hormone is concerned with the maintenance of calcium concentration at the cell membranes. It functions by increasing the passage of calcium through the lining of the intestine, by increasing the resorption of bone tissue, and by increasing the reabsorption of calcium in the renal tubuli. Overactive parathyroid hormone causes osteopenia by excessive resorption of bone; in extreme cases, spontaneous fractures may occur. Excessive secretion of parathyroid hormone may be due to a tumour of the parathyroid glands, may be secondary to dietary deficiency or malabsorption of calcium and vitamin D, or may be due to renal osteodystrophy see below. Adrenal corticosteroid hormone is associated with skeletal abnormalities, osteopenia, and osteonecrosis. Osteopenia develops because increased levels of corticosteroids, caused by disease e. Osteonecrosis is associated with even short-term intake of large doses of high corticosteroid medication. The effects of kidney disease on bone reflect the role of the kidney in maintaining calcium and phosphate balance, mediated by parathyroid hormone. The two main units of the kidney, the tubules and the glomerulus , are associated with two groups of bone diseases: In addition, kidney transplantation is associated with overactivity of the parathyroid glands and osteonecrosis. Reabsorption of phosphate by the kidney tubules is deficient in a hereditary disorder known as familial hypophosphatemia ; the phosphate leak causes low concentration of blood phosphate and, in turn, deficient mineralization of bone tissue, rickets, and osteomalacia. Familial hypophosphatemia is the most common cause of rickets in Europe and the United States. The basic deficiency is treated with high oral doses of phosphate. Advanced forms of the disease result in stunted growth and skeletal deformity, often necessitating repeated surgeries. More-complex tubular reabsorption defects are the cause of bicarbonate, amino acid , and glucose losses; the resulting disease is so severe that the bony abnormalities usually become less important. Renal glomerular disease with high levels of urea in the bloodâ€" uremia â€"is associated with renal osteodystrophy. This condition leads to severe rickets or osteomalacia associated with compensatory secondary hyperparathyroidism. In children, stunted growth may be the first symptom that leads to detection of the kidney disease; the skeletal abnormality cannot be ascribed solely to an abnormal mineral balance but is probably also due to an adverse effect of uremia on protein metabolism. Growth may resume after successful kidney transplantation, and gross deformity of the extremities may be corrected surgically. Chronic uremia in adults, even when treated by use of the artificial kidney, causes osteoporosis and deposition of calcium apatite in arterial walls and tendon sheaths, probably associated with hyperparathyroidism. Kidney transplantation is occasionally followed by hyperparathyroidism and osteonecrosis. The overactivity of the parathyroids is ascribed to the fact that, prior to correction of the kidney disease, the glands have had to function at an abnormally high level for such a long time that the mechanisms for shutting them off have become deficient. Osteonecrosis after kidney transplantation is at least partly due to the high doses of corticosteroid medication used to prevent rejection of the transplant. Osteonecrosis of the hip or knee joints may cause residual disability after successful kidney transplantation. Generalized osteopenia without evidence of osteomalacia is termed osteoporosis. It may be secondary to metabolic abnormalities discussed above or may be without known cause. Osteoporosis from unknown cause is by far the most common bone disease; it probably occurs in all elderly individuals and may sometimes become evident as early as age 30 or The spine is particularly affected. It is generally believed that the commonly occurring fractures in old ageâ€"namely, those of the hip, knee, and wristâ€"are due to osteoporosis. Unlike vertebral fractures in osteoporosis, fractures of the limbs hardly ever occur without a distinct accident, and they are never preceded by bone pain or tenderness. The diminished quantity of bone tissue, the characteristic feature of osteoporosis, is clearly implicated in the diminished resistance of the bones to fracture, but there may also be a change in the quality of the bone tissue. In women, osteoporosis is caused by a change in the hormonal pattern, and hormone replacement therapy is sometimes used. Weight-bearing and resistance exercises are advocated both as prevention and as therapy in osteoporosis. Paget disease , increasingly common after middle age , is characterized by widespread areas of osteosclerosis; the cause is unknown.

Chapter 5 : Metabolic Bone Disease (MBD) | Opossum Society of the United States

This technique allows the physicians to evaluate the degree of bone loss, to diagnose osteoporosis and the risk for future fracture. Appointments. To make an appointment with a physician in the Center for Osteoporosis and Metabolic Bone Disease, call

Osteoporosis Hypoparathyroidism Hypoparathyroidism is a condition caused by an underproduction of parathyroid hormone PTH by the parathyroid glands. These glands, located in the neck, are responsible for managing the levels of calcium, vitamin D and phosphorus in the body. If the parathyroid glands do not secrete a sufficient quantity of PTH, the amount of calcium and phosphorus in the body become unbalanced. It can cause symptoms including cataracts, dry hair, abdominal discomfort, scaly skin, muscle cramps and seizures. To diagnose hypoparathyroidism, your doctor will perform blood and urine tests to measure the levels of various minerals and PTH. Treatment for hypoparathyroidism consists of taking daily supplements of calcium carbonate and vitamin D. Regular follow-up appointments are essential to ensure the correct levels are steadily maintained. Osteomalacia Osteomalacia, also known as rickets, is a softening of the bones that may occur as a result of a vitamin D deficiency. This condition often leads to a dull aching pain, muscle weakness and an increased risk of fractures, especially within the ribs, spine and legs. If this condition is suspected, your doctor may perform diagnostic testing such as X-rays or a bone mineral density test to evaluate the health of the bones and determine the underlying cause of symptoms. Osteopenia Osteopenia is a condition characterized by low bone mineral density, but not as low as that for osteoporosis. A bone mineral density test estimates the strength of your bones by measuring the density of minerals like calcium. If too many minerals are lost, bones become more porous, light and considerably weaker. Osteopenia can be a result of the natural aging process, eating disorders, vitamin absorption problems or medications. There are no symptoms associated with osteopenia, but those with the condition have a higher risk of bone fractures. It is diagnosed through bone mineral density testing. Treatment for osteopenia often consists of making lifestyle changes to maintain existing bone mass, such as increasing calcium intake through diet or supplements, and exercising to strengthen the bones. Osteoporosis Osteoporosis is a disease characterized by increasing bone loss which can lead to fractures, height loss and a hump-backed appearance. One in two women, and one in five men, over the age of 65 will suffer at least one bone fracture due to osteoporosis. A diagnosis of osteoporosis is made after a complete medical history, physical examination and laboratory tests, including X-rays and bone densitometry. Other possible causes of bone loss must be ruled out as well. Lost bone cannot be replaced, but the treatment team will work with you to prevent further weakening. The treatment plan may include exercise, diet changes, hormone therapy with estrogen ERT or anti-estrogens SERMs , or bone-preserving medications such as Calcitonin or Alendronate. We naturally produce vitamin D when exposed to sunlight. But if sun exposure is low and you do not consume foods rich in vitamin D, your levels will become deficient. Vitamin D is important because it works together with calcium to strengthen the bones. A vitamin D deficiency can cause such symptoms as bone pain and muscle weakness, but for many people the symptoms are subtle. This condition has been shown to increase the risk of cardiovascular ailments, cognitive problems and cancer. Vitamin D deficiency is diagnosed through a blood test.

Chapter 6 : Metabolic bone disease - Wikipedia

Metabolic Bone Disorders - This is a collective term used for a number of diseases which affect the strength of the bones of the body and make them weak. Some of the diseases which can come under the umbrella of Metabolic Bone Disorders are Osteoporosis, Osteomalacia, Paget's Disease etc.

Chapter 7 : Metabolic Bone Disorders: Treatment, Causes, Symptoms

The sections cover disorders of the upper and lower limb and spine, including anatomy, trauma, and degenerative and

acquired disorders, and the basic science of the musculoskeletal system, metabolic bone disease, rheumatologic diseases, musculoskeletal tumors, the sequelae of trauma, and congenital deformities.

Chapter 8 : Metabolic bone disease in the preterm infant: Current state and future directions

Metabolic Bone Disease Reptiles that eat primarily insects or plants are at risk for developing metabolic bone disease, which is caused by an imbalance in the levels of calcium, phosphorous, and vitamin D in their bodies.

Chapter 9 : Metabolic bone disease | pathology | theinnatdunvilla.com

Paget's disease is a chronic bone disorder that affects bone metabolism and causes bones to become fragile and unable to regenerate correctly. New bone is produced at a faster rate and is often softer and weaker than normal, increasing a patient's risk for pain and fractures.