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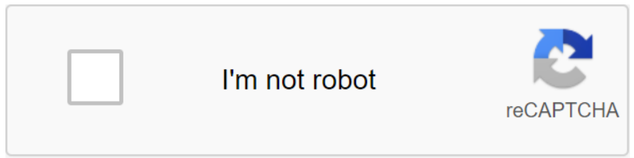

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CALCIUM METABOLISM DISORDERS Dr. Veronica Araya Z. Clinical Hospital of the Northern Campus Physiological Regulation of Calcium and Phosphate depends on the action of the parathyroid hormone (PTH), vitamin D and calcitonin. The consequences of each are shown in Table 1. PTH is produced in parathyroid glands supported by calcium homeostasis, phosphorus and vitamin D, as well as bone mass. The main function of PTH is to keep the level of calcium in the blood stable, reducing calcium, in particular ion calcium, stimulates the production and secretion of PTH. On the surface of the parathyroid cell is a calcium ion sensor receptor (CaSR), through which the cytosal calcium concentration and PTH secretion are regulated. The main actions of PTH on white organs are mediated by binding to the type PTH receptor and stimulation of the second CAMP messenger. HYPERCALCEMIA Normal range of calcium varies between 8.5-10.5 mg/dL. Calcium, usually measured in laboratories, corresponds to total calcium, i.e. calcium attached to albumin and globulinameme, so when hypoalbuminemia exists, falsely normal or reduced calcium can be obtained. To get the actual value, the measured calciemia must be corrected, depending on the reduction of the album below its normal value, according to the following formula: Actual calcemia (mg/dL) - Measured calciemia (mg/dL) - 0.8 x 4 -Albumemia (g/dL) From a practical point of view hypercalcemia can be classified as: Mild : 10.6 mg/dL) dL -12.5 mg/dL Moderate: 12.6 mg/dL -14.5 mg/dL Heavy: zgt; 14.5 mg/d Malinya: qgt; 18.0 mg/dL In general, the causes of hypercalcemia can be divided into: associated with malignancies, more common in hospitalized and neo-humor patients, more common in the outpatient population. PHYSIOPATHOLOGY hypercalcemia can be caused by increased calcium absorption in the gut, mediated by an increase of 1.25 (OH)2 vitamin D or increased osteoclast activity and bone reabsorbion, mediated by PTH, or humoral mediators with PTH-like activity (PTHrp, some cytokines) produced by tumor cells. CAUSES OF HYPERCALCEMIA Elevated intestinal calcium reabsorbtion - Granulomatosis Diseases: Sarcoidosis Tuberculosis - Vitamin D Poisoning - Malignant Lymphoma Elevated Osteoclastic Activity - Not Associated with Malignancy Hyperparathyroidism Hyperthyroidism - Associated with malignant pathology of the squamous cell carcinoma of the lungs, head and neck. Breast Cancer Multiple Myeloma In granulmatous diseases granuloma can secrete 25 OH) D3, which by the action of the 1st hydroxylasse becomes 1.25 (OH)2 D3, which is an active form that is at the level of the intestines and bones and suppresses the secretion of PTH from parathyroids. Tumor hypercalcemia can be produced by two mechanisms: humoral, mediated by paratormone associated with protein (PTHrP), which is released into circulation from tumor cells and, localized osteothy mechanism, synthesis and release from metastatic tumor cells in the bone, from a number of factors that act through the mechanism of paracrine. PTHrP presents homology with PTH in the terminal amino acid molecule for this purpose, can bind to the PTH receptor and activate it. In turn, this determines the activation of osteoclast, an increase in bone resorbion, and an increase in renal calcium reabsorbion. Several cytokines such as interleukin 1 and 6 (IL-1, IL-6), tumor necrosis factor A (TNF-a), epidermal growth factor (EGF), prostaglandins or vitamin D metabolites (1.25 hydroxicolecaliferol) are involved in local osteolytic hypercalcemia. HYPERPARATHYROIDISM It is defined as a clinical syndrome that identifies specific symptoms and symptoms derived from increased bone resorption and hypercalcemia, by increasing PTH. It can be primary, secondary or tertiary. Secondary hyperparathyroidism is the result of chronic secondary hypocalcemia to chronic renal failure, intestinal malabsorbtion, rikitism or begging such as phenytoin, phenobarbital or laxatives, which cause a decrease in intestinal calcium absorption. Continuous stimulus in parathyroidism determines glandular hyperplasia. He reluctantly observes in patients with chronic hemodialysis tertiary hyperparathyroidism is a condition in which parathyroid hyperplasia, echoing chronic hypocalcemia, becomes a drowning and develops hypercalcemia. This situation is usually irreversible, despite the correction of the condition that has identified hypocalcemia and should be treated surgically. It can develop after a kidney transplant. Table 2 shows the laboratory and differential diagnostic characteristics of three types of hyperparathyroidism. PRIMARY HYPERPARATHYROIDISM (HPP) Prevalence 1/1000. Relationships between women and men: 2-3/1. Incidence increases with age and in postmenopausal women reach a disease 5 times higher than the population as a whole. CAUSES: -Single adenoma (85%) -Multiple adenomas (5%). CLINICAL MANIFESTATIONS - Neuropsychiatric: fatigue, memory loss, depression, psychosis, drowsiness, partesthesia. Neuromuscular eyes: Myalgia. gastrointestinal: abdominal pain, nausea, vomiting, constipation, GO ulcer, pancreatitis. Bone osteoitis: bone pain, osteoporosis, fibrous osteoeitis, subperiostal reabsorbtion and renals: Hypercalcion, polyuria, polypsia, nephrolyciasis, nephrocalcinosis. Table 3 shows the changes in the HTA and Arthralgia Keratopathy Laboratory. To confirm the diagnosis of hes, at least 3 fasting calcium should be taken and corrected according to albuminemi. Make sure the patient has normal kidney function. Stop thiazid diuretics at least 1 week before testing. To evaluate PTH, you should request the definition of an untouched molecule (IRMA or CLIA), and the sample must be taken after 10 a.m. Visualization techniques are not recommended when it is your first surgery and you have an experienced surgeon. The synthography with 99mtecnecio-sestamibi has sensitivity and specificity of about 90%. Useful in the location of aden. Less useful for hyperplasia. Une is specific in patients with thyroid nodules. Cervical echotomography is useful as an additional examination at the location of the aden. TREATMENT Symptomatic hes has signs of surgical treatment. Surgery is successful in 90-95% of cases. Complications of zlt;5%. requiere' una' hospitalizaci'n' breve' y' la' mortalidad' es' rara.' el hpp' asintom'tico' no' tiene' indicaci'n' quir'rgica, 'excepto en' pacientes' que presenten' cualquiera' de l' sigui les Las' gu'ass del' panel de'expertos publicadas el a'o' 2009: It's a good time. -2.5' en' cualquier' sitio y/o' fractura' previa' por' fragilidad, the clearence of de creatinina'gt; zlt;min,'calcemia' 1 mg/dl above the normal high limit. However, these patients should be monitored annually for at least calcium, creatinine and bone densitometry. In patients with aritmptomy PPH, in which the corresponding follow-up will not be possible, surgical indication should also be considered. In these patients, vitamin D levels should be maintained above 20 ng/dL. In some cases indicated to perform treatment with anti-reabsortive therapy: estrogens, bisphosphonates or, calciumimemetics, which reduce calcium and PTH and increase phosphorus. GIPERCALCemia CRONICA HYPERCALCEMIA is one of the most common metabolic complications, presenting itself in up to 1 in 200 hospitalized. Pathogenic mechanisms leading to hypercalcemia determine at the kidney level an increase in the allocated calcium load. Chronic hypercalculation causes osmotic diuretic enlargement, resulting in depletion of volume and reduced filtration rateIf this is not corrected, the kidney is unable to release a filtered calcium load and hypercalcemia appears. As calcity increases, the rate of global filtration decreases even more, contributing to an increase in hypercalcemia. When dehydration occurs, usually determined by the gastrointestinal symptoms associated with this pattern, the renal ability to secrete calcium load also falls, as well as an increase in its proximal reabsorbation along with sodium. Subsequently, neurological commitment, which develops and changes the mechanism of thirst, cause greater dehydration and reduced global filtration, increased hypercalcemia and, finally, can determine the hypercalcemia crisis. Clinical manifestations will depend on the level of calcium, but mainly on the time of evolution and underlying pathology. A large number of cases develop in the imptom form and hypercalcemia is a random conclusion in the biochemical profile. Acute hypercalcemia can manifest itself as a heavy pattern with not so high calcium values. CLINICAL TABLE OF HYPERCALCMIC CRISIS In terms of clinical manifestations, they are fundamentally dependent on the rapid establishment of hypercalcemia without calcimity value. Symptoms are usually non-specific, and should always suspect a cancer patient with anorexia, nausea, vomiting, polyuria, polypsia, constipation, muscle weakness, concentration problems or memory loss. Late, more severe neurological symptoms received from encephalopathy such as drowsiness, hypotension, hypoflexion, lethargy, personality changes, hallucinations, psychosis, decreased osteotendinous reflexes, decreased muscle strength (including respiratory musculature), coma and death can occur. Characteristically, the interval of ECG-RT. Acute hypercalcemia can quickly develop into a coma with severe dehydration and cardiac arrhythmia such as bradirythmia: clogging branches, full atria-ventricle or cardiac arrest. DIFFERENTIAL DIAGNOSTIC HYPERCALCEMIA The main differential diagnosis that occurs, in a patient without a known prior neoplasm, with HPP. The latter, usually associated with hypophosphate in most cases and intact PTH elevated as opposed to hypercalcemia tumor in which it is suppressed or, inappropriately normal for calcity levels, since the methods of chemoluminescence currently used to measure PTH, do not have cross reactivity with PTHrP. Measuring 1.25 hydroxy D2 is also beneficial in our differential diagnosis, although our vitamin D2 is not cross-reactivity with PTHrP. quantitative estimation method. In addition, in the assessment, the use of certain drugs that may contribute to an increase in calcium, such as diuretics thiazide, vitamin D and lithium should be excluded. TREATMENT Relevance in the treatment of hypercalcemia is fundamentally dependent on the severity of associated symptoms without the importance of calcium. Acute hypercalcemia can manifest itself with great commitment even at moderately high calcium levels and, conversely, patients with chronic hypercalcemia may develop amptomatic to very high calcity values. If hypercalcemia is imptomatic and is under 13 mg/dL, it does not require aggressive confrontation. In the case of symptomatic and severe hypercalcemia, the support measures described below should be initiated, and the necessary tests will be carried out in parallel to establish etiology when it is unknown. Increased renal calcium secretion: Increased calcium secretion from the urinary tract is achieved by reducing sodium absorption in the proximal tubelum and henle pen, thereby reducing the passive transport of calcium. The salt infusion allows to expand the extracellular volume, which increases the delivery of sodium, calcium and water to the handle. Use pen diuretics such as furosemide transport blocks at this level. 1 or 2 liters of isotonic saline solution is poured in the first hour, and then furosemide is injected with 40 mg of endovenous every 2 hours. Time diuresis and central venous pressure should be monitored, especially in elderly patients, and plasma electrolytes are controlled every 4 hours. According to the time diuresis, the infusion rate of the solution is regulated using glucose at 5% plus 4 grams of sodium chloride and 2 grams of potassium chloride, adapting the concentration after the evolution of sodium and chlorium. In some cases, calcium drops within 2 or 4 hours, but in others more than 12 hours of therapy is required. Magnesium should be given in the form of 1 vials of magnesium sulfate to pass every 8 hours. Inhibition of bone reabsorbion: Agents that can block osteoclast activity are: bisphosphonates and calcitonin. Bisphosphonates: mostly useful for hypercalcemia associated with tumor pathology. Its maximum effect can be observed a few days after the start of treatment. The most commonly used pamenodonate bisphosphonates and zoledronic acid are administered endionly. Alendronate or ibandronic acid can be used in the chronic treatment phase. Your instructions should be evaluated by a specialist. Salmon calcitonin: Acts quickly, starting to reduce calcium within 2 to 3 hours after treatment begins. calcium at 1 or 2 mg/dL. The main drawback is that more than 50% of patients develop the tahiphylaxis phenomenon on the second or third day of treatment. Only intranasal form is available in our environment. Inhibition of intestinal absorption of calcium: corticosteroids: they are useful in pathologies that increase the absorption of calcium in the intestine, which is associated with an increase in the concentration of calcitriol. Prednison 30-40 mg/day can be used orally, hydrocortisone 150-200 mg/day through endovenosis. In hyperparathyroidsis there is no good reaction to its use. Dialysis: Useful primarily in patients with renal disorders or congestive heart failure, who may not be subjected to volume overload or, in patients with malignant hypercalcemia with calcium levels above 18 mg/dL. Peritoneodialysis is slower than hemodialysis. The therapeutic measures mentioned above reduce calcium and restore volume deficits, but do not allow to treat the trigger cause. This is why managing underlying pathology is important, whether it is the surgical approach of parathyroid adenoma or neoplasm or chemotherapy or radiation therapy in some tumors. In the case of patients with terminal neoplastic pathology, outside the therapeutic realm in which hypercalcemia is usually the final event, it should be clearly defined as far as to go in the implementation of therapy. Table 1 Table 2 Table 3 HYPOCALCEMIA Is defined as a condition in which serum calcium falls below the normal range, zlt; 8.5 mg/dL corrected according to albumin. SIGNS AND SYMPTOMS symptoms of HYPOCALCEMIA largely depends on whether it is a chronic or acute table setup. The manifestations that can be observed are: Neuromuscular: signs of Khvosteka and Truss, partession, tetani, convulsions (focus, petty evil, great evil), fatigue, anxiety, convulsions, Larung spasm, bronchial spasm. Neurological: extrapyramidal signs by calcification of basal ganglia, calcification of the cerebral cortex or cerebellum, personality disorders, intellectual disorders, non-specific changes of EEG, arkinonism, choroatthosis, increased intracranial pressure. Mental state: confusion, disorientation, irritability, psychosis and ectodermal changes: dry skin, fragile nails, thick, raloe hair, alopecia, atopic eczema, exfoliating dermatitis, psoriasis, herpesform impetigo, hypoplasia of tooth enamel, shortened premolation roots, toothache Ophthalmological: subcapsular cataract, papyledema and cardiac: long interval of RT in ECG, shortness of breath, inupha. congestive heart disease, cardiomyopathy. CAUSES HYPOCALCEMIA HIPOPARATIROIDISM DECLINE OR LOSS PTH action followed by changes in the mechanisms of calcium homeostasis leads to hypocalcemia and hyperphosphemia. CAUSES DESTRUCTION OR REMOVAL OF PARATHYROID TISSUE WITH INADEQUATE SECRET RESERVE: - Postoperative hypoparathyroidism - autoimmune hypoparathyroidosis - Radiation - Metal deposit: iron (thalassemia, hemochromatosis), copper (Enf.) - Metastasis REVERSIBLE ALTERATION OF PTH SECRETION GENETIC DESORDENS OF PTH SYNTHESIS AND DEVELOPMENT OF PARATIROIDES RESISTANCE TO PTH ACTION Post-surgery hypoparathyroidosis is the result of unintentional removal or irreversible damage to parathyroids, usually by taking measures to irrigate them This can be seen in up to 9% of thyroid surgeries and mainly depends on the surgeon's experience, the degree of thyroid resection and the opening of the ganglion. In hypocalcemia after parathyroid surgery, it is important to establish a differential diagnosis of hypoparathyroidism with Hunger Bone Syndrome (SHH), in which there is an intensive absorption of calcium by the bone after a decrease in PTH. Measuring phosphate is key as hypoparathyroidism will instead be increased, SHH is reduced. Autoimmune hypoparathyroidism may occur in isolation or in the context of autoimmune polyglandular chondroma (SPA). This is due to the activation of CASR antibodies. SPA-I is associated with a recessive and often heterologo mutation of the AIRE gene (regulatory autoimmune). The classic triad corresponds to the association of hypoparathyroidism (80%), adrenal insufficiency and thrush mucokutana. TREATMENT In chronic hypocalcemia, the therapeutic goal is to maintain calcium near 8.5 mg/dL and 400 mg/day. Treatment is based on calcium supplements in the form of carbonate or calcium citrate and vitamin D. The necessary dose of calcium can range from 3 to 7 g/day for introduction in fractional form. Some of the formulations available in our environment and its elementary calcium equivalence are: Vitamin D supplements can be performed using an active form (calcitriol) daily, or its predecessors, which are administered weekly or biweekly depending on the response. COMPOSITE POWER RELATIVE TO VIT D3 Vit. D3 Akuod (p) 300,000 U 1 Vit. D2 (Ergocalciferol) 600,000 U 1 1.25 (OH)2 D3 (Calcitriol) Rocaltrol (r) 0.25.0.5 mg 1000 PTH recombinate or teriparatide has been used in isolated cases and does not yet have sufficient evidence to indicate it regularly. AGUDA HYPOCALCEMIA Therapeutic goal is to increase calcemia by 2-3 mg. Used calcium gluconate, 1-2 vials dilute 100 ml sg 5% infused in 10 minutes. In addition, calcium and oral vitamin D should be started together. Oral. barron's toefl ibt 15th edition pdf free download vk. barron's toefl ibt 15th edition pdf vk