Acute Renal Failure Due to Vitamin D Intoxication

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Objective: Vitamin D deficiency is associated with multiple diseases. Vitamin D becomes more popular every day with the increasing studies. Vitamin D replacement therapy cases increase as well as Vitamin D intoxication cases. We present a 80-old female patient who developed acute renal failure due to vitamin D intoxication.

Case: Patient came to the emergency room with the complaints of 4-day continuing nausea, vomiting and loss of appetite. Mucosa of the patient was dry and she was dehydrated. Tests results were: urea:186mg/dL, creatinine:3.64mg/dL, albumin:3.26 g/dL, calcium:12.7mg/dL and venous blood gas results were: pH:7.41, pCO2:41.9, HCO3:26.6. Calcium, urea and creatinine levels were normal in a test that was performed 2 months ago. She was diagnosed with acute pre-renal failure connected to dehydration. A detailed medical history showed that 2.5 months ago, the patient was tested vitamin d3-25-OH (kalsifiediyol):6.2 ng/ml and vitamin d3 300 000 IU bulb total of 3 was prescribed to her to be taken orally once a week, however, it was realized that the relatives of the patient gave her total of 10, once a week. After this finding, performed test results were detected as vitamin d3-25-OH (kalsifiediyol):365 ng/ml, parathyroid hormone:16.8pg/ml. The patient was hospitalized with a diagnosis of vitamin D intoxication and intravenous hydration with saline and IV furosemide infusion therapy was applied. At the end of second day her complaints have decreased and at the end of third day calcium level were 9.3mg/dL. At the tenth day test results were: urea:30 mg/dL, creatinine:1.12 mg/dL, and the patient was discharged.

Conclusion: Due to growing interest in vitamin D, it is very important that clinicians should be aware and alerted of that overdose vitamin D replacement therapy can cause vitamin D intoxication.

Severe Hypercalcemia: Chronic Tophaceous Gout the Responsible Cause?

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Background and Description: The association of chronic tophaceous gout with severe hypercalcemia is extremely rare and has been usually associated with calcitriol secretion. PTHrP has never been described as the responsible cause. A 42-year old man with a long stand history of chronic gout initiated a week before admission with polyuria, polyposis and progressive altered mental status. Neurological examination revealed only lethargy. Characteristic, multiple, non-tender tophi were obvious at inspection (Figures 1-3). Initial laboratory work up revealed a uric acid of 14.0 mg/dL, calcium of 14.5 mg/dL, phosphorous of 6.3 mg/dL, creatinine of 5.4 mg/dL, blood nitrogen urea of 56, a MDRD GFR of 16 ml/min.

Discussion and Therapeutic Approach: PTH was suppressed (< 30 pg/ml), 25-dihydroxyvitamin D was normal, PTHrP was elevated >50 pg/ml, and calcitriol normal (196 pg/ml). Radiographs revealed bone erosions (Figure 1-3). Bone scan and a PET-CT were negative for metastasis and malignancy. Treatment was initiated with calcitonin, hydration and prednisone. PTH, 25-dihydroxyvitamin D, PTHrP, and calcitriol returned to normal values. At 6 months follow-up he referred no pain, tophi had improved and calcium levels were within normal range.

A Case of Recurrent Multifocal Diabetic Myonecrosis

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Diabetic myonecrosis is a rare complication associated with poorly controlled diabetes. Although diabetes may be a common disease, diabetic myonecrosis is a rare complication. The usual presentation is sudden onset of pain at the involved muscles associated with swelling and tenderness without previous trauma or fever. Diagnosis is clinical but MRI is the best investigation for diagnosis. Muscle biopsy is necessary in cases that can be supported by MRI. Muscle biopsy is important, to exclude other causes such as pyomyositis, necrotising fasciitis, and neoplasms. Conservatively managing including tight glycemic control should be achieved. Nonsurgical therapy appears to provide the most favorable outcome.

Severe Hypercalcemia: Chronic Tophaceous Gout the Responsible Cause?

Discussion and Conclusion: A systemic research on PubMed, Medline, Embase and MedConsult with the search criteria: “Giant Cystic Prolactinoma”, “prolactin”, “prolactioma” and “hook effect” was made. Due to the clinical signs of hypogonadism, the patient referred the pituitary disease. If missed, this would have delayed the diagnosis and consequently would have changed the treatment plan and prognosis of the patient.

A Giant Cystic Prolactinoma in a Woman: Do Not Forget the Hook Effect

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Background and Case Presentation: Cystic prolactinomas correspond to 1% of all prolactinomas and its association with a giant pituitary macroadenoma in a woman has been seldom reported. A 43-year old woman was evaluated for a suprasellar macroadenoma. She had absence of menses since 2 years. Three months before she started to have progressive headache. She also referred asthenia, asthenia, fatigue, dry hairiness, decreased libido, vaginal atrophy, and chronic constipation. Hair in armpits and pubic area was insignificant and osteotendinous reflex relaxation phase was slow. Galactorrhea was absent and presented bitemporal hemianopsia.

Discussion and Therapeutic Approach: MRI scan revealed a 5 x 3.5 cm suprasellar mass and a 8.1 x 3.6 cm cystic component (Figures 1-3). Cortisol level 2.2 mg/dl, FSH and LH were low, estradiol < 0.05 pg/mL, IGF-1 normal and a serum prolactin of 125 ng/mL. Serial dilutions 1:10 and 1:100 were made. Diluted prolactine was 12,500 ng/mL. Treatment with cabergoline, prednisone and levothryoxine were initiated. At two months follow-up prolactin serum levels decreased to 32.1 ng/mL.