osteolytic metastases, or tumor production of calcitriol. In cases of hypercalcemia due to excess PTH secretion, primary parathyroid etiologies are typically considered while ectopic PTH-secreting tumors are rare. PTH staining of biopsy specimens and total body sestamibi scan may prove useful in the early detection and treatment of these tumors, but HCM offers a poor prognosis with mean survival of 2 to 3 months and in-hospital mortality of 6.8%. Currently, there are only three cases in the reported literature of ectopic PTH-induced hypercalcemia related to ovarian cancer. To our knowledge, this is the fourth reported case.
Conclusion: Ectopic PTH-secreting tumors carry a poor prognosis and should be considered in cancer patients presenting with PTH-associated hypercalcemia. Biopsy staining for PTH and total body sestamibi scan may assist in the early detection of these tumors, but current treatment strategies offer suboptimal outcomes.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Tumor Induced Osteomalacia - Long Term Medical Treatment When Surgery Is Risky

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Introduction: Tumor induced osteomalacia (TIO) is a rare paraneoplastic syndrome characterized by bone pain, muscle weakness and fractures caused by production of a phosphaturic factor by rare mesenchymal bone or soft tissue tumors that causes isolated renal phosphate loss and osteomalacia. Low phosphate (iP), high bone alkaline phosphatase (BAP), and normal or low 125 dihydroxy D, are salient biochemical findings. Fibroblast derived growth factor (FGF 23) may be high or inappropriately normal. Gallium-68 Dotatate (DoT) imaging has become the imaging method of choice. Long term medical management may be required when removal of the tumor is risky or not feasable. **Case Report:** 65 yr old woman with carcinoid tumor of the right lung and a bony lesion in the T3 vertebral body diagnosed with TIO. She was initially screened for osteoporosis after traumatic rib fractures. Bone scan and SPECT-CT revealed numerous foci of increased uptake. She had elevated alkaline phosphatase 186 IU/L (0–140) and PTH 83 pg/ml (15–65) with a low phosphate 1.5 mg/dl (2.5–4.5), along with normal FGF23 level 102 RU/ml Ref Range < + 180 RU/ml. DoT and PET CT imaging for TIO evaluation showed a foci of increased uptake right lower lobe of her lung, and "osseous metastatic disease" in the right scapula, vertebral body, iliac, and pubic ramus. Sclerosis of T3 vertebral body was noted in the area of intense Gallium Dotatate uptake. Transbronchial excision of the lesion showed a well differentiated neuroendocrine carcinoma. Chromogranin A and 24 hour urine for 5HIAA were normal. She responded well to medical therapy with oral phosphate, calcium and calcitriol. Follow up, DoT and FDG PET showed persistent intense uptake in the sclerotic lesion on T3 vertebral body, while the rest of the hot spots resolved. Sclerotic T3 lesion is likely the primary lesion that is responsible for the TIO. Neuroendocrine tumor of the lung may be a mere association. Biopsy of the T3 lesion was not feasible and excision was considered risky to the patient.
Discussion: Our case illustrates that awareness is the key to early diagnosis of TIO. FGF 23 in some TIO cases may be inappropriately normal in commercial assays and even in research labs. Measurement of fibronectin 1 (FN1) and FGF receptor 1 fusion gene which is noted in up to 60% of tumors are not commercially available. While DoT and PET CT are imaging modality of choice, CT and MRI may be useful to define the anatomy of the lesion. Long term medical management may be necessary when removal of primary lesion is not possible or risky. Most tumors are benign while some may prove to be malignant.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Tumor Induced Osteomalacia in Patients With Metastatic Prostate Cancer: Case Report and Literature Review

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Background: Tumor induced osteomalacia (TIO), driven by elevated levels of fibroblast growth factor (FGF23), has been associated with progressive aggressive prostate cancer. FGF23, expressed by osteocytes, plays an important role in tumorigenesis through cell proliferation, chemotaxis, and angiogenesis. FGF23 regulates phosphate homeostasis through feedback loops between the kidney and bone. We present a case report of a patient with TIO and advanced prostate cancer (PC) and review previously published data.
Clinical Case: A 63-year old male with castrate-resistant PC with bone metastases presented with symptoms of profound fatigue, memory loss, and brain fog. Laboratory studies demonstrated severe hypophosphatemia (phosphorus 0.8 mg/dL, normal 2.5–4.5 mg/dL), mild hypocalcemia, secondary hyperparathyroidism, normal 25 hydroxy-vitamin D and 1,25-dihydroxy vitamin D. After treatment with IV and oral replacement, phosphorus improved to 2.2 mg/dL. Urinary phosphorous was elevated at 2105 mg/24 hours (normal 400–1300 mg/24 hours) and FGF23 545 RU/mL (normal < 180 RU/mL). His PC treatment regimen extended over 10 years with multiple systemic, radiation, and targeted ablation therapies. He was also on denosumab every 3 months for metastatic bone disease, but it was discontinued due to hypophosphatemia. He is now treated with calcium carbonate-vitamin D 1000 mg-250 IU twice daily, calcitriol 1.5 mcg/daily, Phospha Neutral 250mg 2 tablets four times daily, and abiraterone acetate. His hypophosphatemia, hypocalcemia and secondary hyperparathyroidism has resolved with this regimen. To our knowledge there are 17 cases of TIO in metastatic PC are reported to date. Majority of the patients have similar clinical presentation. Treatment of metastatic PC, as well as calcitriol, phosphorus replacement, octreotide, and cinacalcet were described.