Abstract citation ID: luae218.024

P-21 POSSIBLY ECTOPIC PTH SECRETION IN A WOMAN WITH METASTATIC BREAST CANCER

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Introduction: Hypercalcemia is a common complication in malignancies, with various causes such as tumor secretion of parathyroid hormone-related peptide (PTHrP), osteolytic metastases or tumor-induced calcitriol production. Ectopic production of parathyroid hormone (PTH) is a rare cause of hypercalcemia. This report presents a rare case of metastatic breast cancer possibly associated with ectopic PTH secretion. **Clinical Case:** A 66-year-old female with a history of invasive ductal carcinoma and metastases of bone and liver was referred to our endocrinology clinic due to persistent hypercalcemia. She had been presented with atypical-pathological femur fracture 2 years ago and metastatic breast cancer had been detected. Her primary cancer treatment included conventional chemotherapy and immunotherapy over the past 2 years. She had been already treated with zoledronic acid from the beginining of the chemotherapy for her bone metastases. During her follow up hypercalcemia had been detected at the 30 th month of the therapy; her calcium was around 9-10 mg/dl and increased to 12 mg/dl abruptly while shewas on zoledronic acid. Laboratory tests showed elevated PTH levels accompanying hypercalcemia. Neck ultrasound and parathyroid scintigraphy were negative for any parathyroid pathology. A biopsy of the liver metastasis was performed during the transarterial chemoembolization procedure to make an immunohistochemical staining for PTH but it was negative. The patient was given cinacalcet with changing dosages according to the calcium levels besides zoledronic acid. Under these medications both calcium and PTH levels continued to rise;during a short period which she could not take cinacalcet because of side effects, her calcium was measured 17.5mg/dl,and didn't come to normal levels despite glucocorticoid and furosemide treatment which she subsequently required hemodialysis. Zoledronic acid was replaced with denosumab. Although calcium levels were controlled with denosumab and low dosage cinacalcet PTH levels remained elevated, suggesting ongoing ectopic PTH secretion. With no identifiable parathyroid hormone source and persistent high PTH levels despite normalized calcium, the diagnosis of ectopic PTH production was assumed. Besides the high levels of PTH we couldn't visualize a lesion secreting PTH on the contrary of direct relationship between the volume of parathyroid lesions and PTH levels. **Conclusion:** This case represents a rare complication of metastatic breast cancer possibly associated with ectopic PTH production causing hypercalcemia. Despite extensive imaging,no parathyroid adenoma or alternative explanation for the elevated PTH was found, reinforcing the need for awareness of ectopic hormone production in the management of hypercalcemia in cancer patients. Early recognition and appropriate management of such atypical cases are crucial to avoid unnecessary interventions and to tailor treatment strategies effectively.



Figure 1. The Patient's Serum Calcium Levels

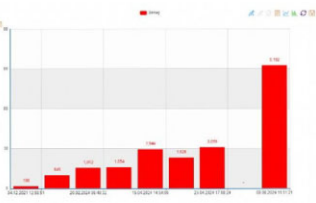


Figure 2. The Patient's Serum PTH Levels

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