Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Neuroendocrine Tumor Associated Severe Hypercalcemia of Pregnancy

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Background: Literature of pregnancy associated neuroendocrine tumors is limited. We report an unusual case of severe hypercalcemia of pregnancy secondary to a neuroendocrine tumor. **Clinical Case:** A 43-year-old lady presented to our center in her 28th week of pregnancy with complaints of worsening nausea and epigastric pain over few days. She had developed gestational diabetes at 24 weeks for which she was taking insulin. On examination she was tachycardic, mildly confused with a distended abdomen. Serum chemistries revealed serum calcium of 15.9 mg/dL, elevated PTHrP of 64 pg/mL and low PTH of 2 pg/mL consistent with PTHrP mediated hypercalcemia. She had low 25-OH- vitamin D (15 ng/mL) with normal 1–25- OH vitamin D. An abdominal ultrasound showed a solid, heterogenous, soft tissue mass interposed between the left kidney and spleen measuring 11 x 9 x 12 cm. This was confirmed on Abdominal MRI, which also showed multiple large hepatic metastasis. Tumor biopsy with pathology confirmed well differentiated neuroendocrine tumor of gastric or pancreatic origin. Her hypercalcemia was initially treated with infusions of IV normal saline, furosemide diuresis, calcitonin 200 units SC 12 hourly and a single dose of dexamethasone 10 mg IV. Calcitonin and diuresis were discontinued after 5 days once calcium improved to 10 mg/dL. Oncology deferred treatment of the tumor until she had delivered. Patient was discharged with serum calcium of 10.7 mg/dL and counselled for plenty of oral hydration. She was readmitted with nausea, vomiting and serum calcium level of 15.1 mg/dL at 32 weeks gestation. She was again treated with a similar regimen of IV saline and furosemide along with calcitonin 480 units SC 12 hourly and prednisone 10 mg daily, but calcium levels remained high (13.6 mg/dL) despite aggressive treatment. Labor was induced at 33 weeks and she delivered a healthy boy through an uncomplicated vaginal delivery. Remarkably, her serum calcium normalized on day two post-partum and remained so throughout. Her serum calcium remains normal at 2 months post-partum (10.3 mg/dL, PTHrP 36 pg/mL, PTH 7 pg/mL) despite the presence of unresected tumor. She is planned to undergo surgical resection of the tumor and hepatic metastatic lesions. **Conclusion:** Clinicians must be aware that severe hypercalcemia may be the presenting manifestation of neuroendocrine tumors in pregnancy. In this case, we hypothesize a potential placental contribution to her elevated PThrP given resolution of hypercalcemia post-delivery. However, persistent elevated PThrP post-partum may also be physiologic or related to the abdominal tumor. A placental histopathological could be performed due to logistic reasons.

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Night Sweats as the Presenting Symptom of Primary Hyperparathyroidism

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Background: Approximately 25–40% of patients report night sweats in the previous month during appointments with their primary care clinicians. The differential diagnosis for night sweats is broad, with hyperthyroidism, carcinoid syndrome, pheochromocytoma, medullary thyroid carcinoma, insulinoma, and acromegaly as established endocrine causes. We present a case of primary hyperparathyroidism (PHPT) in which the patient's chief complaint was night sweats and resolution occurred after parathyroidectomy. **Case.** A 39-year-old female reported one-year of daily night sweats that required changes of clothes and bedding. She denied excessive daytime sweating, frequent palpitations, tremors, nightmares, rashes, fevers, chills, cough, headaches, dizziness, abdominal pain, diarrhea, disrupted menses, or unintentional weight loss. Vital signs and examination were unremarkable. Hypercalcemia (11.0 mg/dL, 8.6–10.3) was noted and confirmed by additional serum calcium measurements. Intact PTH ranged from 27–33 pg/mL (12–88), and 24 h urine calcium (258 mg) excluded familial hypocalciuric hypercalcemia (FHH). Parathyroid scintigraphy and neck ultrasound identified a left neck mass, and the patient underwent successful resection of a left inferior parathyroid adenoma. Hypercalcemia and night sweats initially resolved after surgery, but the patient returned six weeks later with recurrence of night sweats. Reevaluation was notable for serum calcium 10.4 mg/dL, phosphorus 2.4 mg/dL (2.5–5.0), and intact PTH 104 pg/mL. A right superior parathyroid adenoma was identified on repeat parathyroidectomy, and the patient experienced durable resolution of night sweats and hypercalcemia following her second parathyroid surgery. She was screened for multiple endocrine neoplasia type 1 (MEN1) due to multiple parathyroid tumors, though no known pathogenic menin gene variants were identified. **Conclusions:** A title/abstract search in PubMed linking "hyperparathyroidism" and "hypercalcemia" to "night sweats," "sleep hyperhidrosis," "sweating," "hot flashes," "hot flushes," "diaphoresis" and "vasomotor symptoms" yielded only one relevant case of a postmenopausal woman with hot flushes unresponsive to hormone replacement that resolved after parathyroidectomy for PHPT. Hypercalcemia is known to affect central nervous system function. It is possible that in rare cases hypercalcemia alters function of the medial preoptic area, lowering the temperature threshold above which peripheral vasodilatation and perspiration occur to dissipate heat. The patient's predisposition to only night sweats is unclear, though unlike the first patient reported with PHPT and sweating, our patient is premenopausal. This case indicates that vasomotor symptoms may occur with PHPT and resolve after successful parathyroid surgery.

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Osteitis Fibrosa Cystica A Rare Presentation of Primary Hyperparathyroidism