left paratracheal mass excision with pathology confirming recurrent parathyroid carcinoma. After 4 years, her PTH and calcium levels rose. Sestamibi imaging showed abnormal activity in a 1.1 cm soft tissue mass near the sternal notch. The patient underwent complete excision of the left suprasternal mass and the left sternohyoid muscle with pathology confirming recurrent parathyroid carcinoma with lympho-vascular and perineural invasion. Follow-up Sestamibi imaging after a month showed no abnormal activity with recent labs of calcium level 10mg/dl and PTH 25 pg/ml.

Conclusion
Recurrent parathyroid carcinoma remains a challenge. Complete resection of the lesion along with care to avoid capsule rupture to prevent local seeding remains the treatment strategy. The main cause of morbidity and mortality is the sequelae of uncontrolled hypercalcemia rather than tumor burden. Management of hypercalcemia includes hydration, bisphosphonates and cinacalcet. Even though it is not radiosensitive, neck radiation after surgery may be helpful in preventing tumor regrowth. Clinical trials using nivolumab and ipilimumab are being conducted to treat parathyroid carcinoma (2).

Renal ultrasound showed no evidence of nephrocalcinosis or nephrolithiasis. A parathyroid scan was consistent with a left superior parathyroid adenoma. Patient underwent parathyroidectomy and became normocalcemic with normal serum PTH levels postoperatively. One year later she was diagnosed with a left sided bronchial carcinoma tumor. Surveillance Gallium-68 PET/CT scan done 2 years later was negative for any metastases. Twelve years later she reported to our clinic for follow up. She had no symptoms of hypercalcemia, fractures, nephrolithiasis, history of pyelonephritis, diabetes mellitus, analgesic use, or hypertension. Serum calcium was 9.1 mg/dL, serum phosphorous 3.8mg/dL, PTH 82 pg/mL, 25-OH vitamin D 34 ng/mL, 1.25-vitamin D 38 pg/mL, and a urorisk panel was normal except for a 24-hour urine calcium of 410 mg. However renal ultrasound showed bilateral RPN and this diagnosis was also confirmed by a CT scan. A urinalysis showed only microalbuminuria with no red cells. She had no history of any analgesic drug abuse, pyelonephritis, sickle cell disease, or diabetes mellitus. A glucose tolerance test was completely normal. Discussion RPN is characterized by coagulative necrosis of the renal medullary pyramids and papillae brought on by several associated disorders and toxins that exhibit synergism toward the development of ischemia. Although the initial kidney US was normal, a repeat US done 12 years later showed evidence of RPN. This finding along with hypercalcemia and a diagnosis of normocalcemic PHPT suggests that RPN may be associated with hypercalcemia and normocalcemic PHPT. Furthermore she had no other risk factors for RPN. Additional studies with large number of patients are needed to confirm the association between these 2 disorders.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

Renal Papillary Necrosis Associated with Normocalcemic Hyperparathyroidism
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SAT-363

Introduction
Hypercalciuria is generally considered to be the most common identifiable metabolic risk factor for calcium nephrolithiasis. Important renal manifestations of primary hyperparathyroidism (PHPT) include asymptomatic nephrolithiasis, hypercalciuria, nephrocalcinosis, and chronic renal insufficiency. However renal papillary necrosis (RPN) occurring in PHPT has not been reported previously. We report a 50-year-old woman who manifested RPN associated with hypercalcemia and normocalcemic PHPT. Case Report
A 50-year-old Caucasian woman was evaluated in 2006 for hypercalcemia. She had no history of nephrolithiasis, fractures, or symptoms of hypercalcemia. Laboratory: serum calcium 11.8 mg/dL, ionized calcium 6.3 mg/dL, phosphorus 1.8 mg/dL, intact PTH 98 pg/mL (ref 15–65), urine calcium 543 mg/24 hrs (ref <235).

Healthcare Delivery and Education

EXPANDING CLINICAL CONSIDERATIONS FOR PATIENT TESTING AND CARE

Healthcare Services Utilization and Costs Associated with the Management of Patients Living with Acromegaly
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MON-141

Background: Acromegaly (ACRO) is a rare, chronic growth hormone hypersecretory disorder associated with increased morbidity and mortality. Limited information is available on the utilization and costs of healthcare by patients with ACRO.

Aims: To assess the impact of ACRO healthcare utilization and costs by locations of care (LoC).

Methods: A US database of prescription (Rx) drug and medical claims from Jan 2010 to Apr 2019 was analyzed. Patients with an ACRO diagnosis (Dx) were identified based on claims with ICD-9/-10 codes 253.0x/E22.0. The 12-month study period followed each patient’s first ACRO Dx in the database (the index date). ACRO patients

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Adrenal

TRANSLATIONAL STUDIES ON ADRENOCORTICAL FUNCTION IN HEALTH AND DISEASE

Glucocorticoid Production in the Nervous and Immune Systems: Evidence for a Local HPA Axis Homolog

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OR19-04 Glucocorticoid Production in the Nervous and Immune Systems: Evidence for a Local HPA Axis Homolog

The hypothalamic-pituitary-adrenal (HPA) axis is a critical stress response system in vertebrates. The hypothalamus secretes corticotropin-releasing hormone (CRH), which binds its receptor (CRH-R1) in the anterior pituitary. The anterior pituitary then secretes adrenocorticotropic hormone (ACTH), which binds its receptor (MC2R) in the adrenal glands and stimulates secretion of glucocorticoids into the bloodstream. Glucocorticoids are critical modulators of neural and immune system development. During early development (postnatal day (PND) 2 to 12), mice show decreased adrenal glucocorticoid secretion at baseline and in response to stressors, termed the stress hyporesponsive period (SHRP) (1). Traditionally, glucocorticoids have been thought to be synthesized only in the adrenal glands. However, recent evidence demonstrates that glucocorticoids are also produced in extra-adrenal tissues, such as the brain and lymphoid organs (2). This may be of particular importance during the SHRP, as local production allows glucocorticoid modulation of specific tissues and cells, without general effects throughout the organism. Importantly, the factors that regulate local glucocorticoid production remain unknown. To study the regulation of local glucocorticoid production, we examined whether mediators of the HPA axis are locally expressed at baseline and in response to an immune stressor. We assessed systemic and local glucocorticoid levels in neonatal (PND5) C57BL/6J mice 4hr after an immune challenge with lipopolysaccharide (50µg/kg i.p.) or vehicle control. We examined blood, microdissected brain regions (prefrontal cortex, hippocampus, hypothalamus), and lymphoid organs (thymus, spleen, bone marrow). A panel of 7 steroids was measured via liquid chromatography tandem mass spectrometry (LC-MS/MS). Gene expression of Cnrh, Cnrh-R1, Pomp, and Mc2r was quantified via qPCR. Preliminary data indicate that corticosterone was 2-fold higher in tissues than in blood after an immune stressor. The thymus expressed all genes of interest, supporting the existence of a local HPA axis “homolog” in the thymus. Brain, spleen and bone marrow expressed a subset of the genes of interest. These exciting data demonstrate that all the mediators of the HPA axis are locally expressed within the thymus, likely to regulate thymocyte development and reactivity. Greater understanding of local glucocorticoid production will provide crucial insight into neural and immune development and function. Reference: (1) Sapolsky et al., Brain Res Rev. 1986 11(1):65–76. (2) Taves et al., Endocrinology. 2015 156(2):511–522.

Bone and Mineral Metabolism

OSTEOPOROSIS: DIAGNOSIS AND CLINICAL ASPECTS

Decisions to Accept or Decline Pharmacologic Osteoporosis Therapy After Attending a Novel Patient-Centred Educonsult Program for Osteoporosis (PEP-OP)

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SUN-374

Osteoporosis affects >200 million people, resulting in >8.9 million annual fragility fractures worldwide. Available medications can reduce fracture risk by 40–60%, although access to specialty osteoporosis services is limited, and many individuals remain unaware of their fracture risk and their treatment options. As the one-on-one ‘traditional consultation’ (TC) model of osteoporosis care is not time efficient (i.e. a single TC often requires >45 minutes), there is a need to identify innovative consultative models that can improve