wasting and inadequate bone mineralization. Complete resection can be curative; however, these tumors are typically difficult to identify due to size and location.

Clinical case

A 43-year-old man was referred to Endocrinology for evaluation of recurrent fractures and hypophosphatemia. First stress fracture was diagnosed at age 41, followed by musculoskeletal pain in several locations. Lower extremity MRI showed chronic left fibular stress fracture, new stress fracture in fifth metatarsal and right tibia. Tc99m-MDP bone scan revealed multiple foci of increased tracer uptake in bilateral ribs, hips, and lower extremities. Laboratory evaluation showed normal calcium (9.3 mg/dL, normal range [NR]: 8.7-10.2), low phosphorus (1.5 mg/dL, NR: 2.5-4.7), low 1,25-dihydroxyvitamin D (13 pg/mL, NR: 19.9-79.3), low 24-hour urine calcium (78mg), high phosphate excretion fraction in urine (27%, normal <5%), high ALP (163 U/L, NR: 38-126), and high FGF23 (238 RU/mL, NR<180). 25 OH vitamin D (36 ng/mL) and iPTH (5.7 pmol/L, NR: 1.6-6.9) were normal. Patient was started on calcitriol and phosphate supplements. Due to concern for TIO, a PET scan with 68Ga-DOTATATE was performed which showed multiple somatostatin avid lesions concerning for metastatic disease. However, after re-review with radiology, it was felt that other areas of uptake were due to fractures and not tumor given remarkably higher SUV in left acetabular lesion (SUV 20 vs 4-5). The left acetabular lesion was biopsied, followed by surgical resection. Pathology was consistent with phosphaturic mesenchymal tumor with uninvolved margins. FGF23 normalized within 24 hours after surgery (127 RU/mL) and calcitriol and phosphate supplements were discontinued on post-operative day 10.

Clinical lesson

TIO is a rare paraneoplastic syndrome commonly caused by phosphaturic mesenchymal tumors that secrete FGF23. Once the diagnosis of TIO is confirmed, the tumor is localized by anatomical or functional imaging. 68Ga-DOTATATE scan is currently the imaging modality of choice for localization. However, there are other pathologic processes, such as fractures, that could affect the interpretation of PET scans. Osteoblastic activity is increased in fractures, which results in increased uptake in Ga-DOTATATE PET scan since osteoblasts express somatostatin receptor. Our patient was initially thought to have multiple avid lesions concerning for metastatic disease, but culprit lesion was differentiated based on SUVs and confirmed with biopsy. Clinical and biochemical abnormalities resolved after surgery. Early recognition of culprit lesion in TIO is crucial, as successful surgery is curative and would lead to significant improvement in the quality of life of patients.

Cardiovascular Endocrinology

HYPERTRIGLYCERIDEMIA; INFLAMMATION AND MUSCLE METABOLISM IN OBESITY AND WEIGHT LOSS II

Immunologic Effects of GLP-1 Activation in Obese Adipose Tissue

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Background: Obesity is associated with systemic inflammation which is thought to stem, in part, from adipose tissue (AT). Obese AT is characterized by infiltration of pro-inflammatory T cells that promote macrophage activation and inflammation. GLP-1 has been shown to have anti-inflammatory effects in previous studies. We hypothesized that promotion of GLP-1 signaling with liraglutide or sitagliptin would reduce inflammation in association with an increase in the number of anti-inflammatory invariant natural killer T cells (iNKTs), group 2 innate lymphoid cell (ILC2s) and regulatory T cells (Treg) in blood and AT.

Methods: Obese adults with pre-diabetes were randomized to pharmacologic treatment resulting in increased GLP-1 signaling (liraglutide or sitagliptin, N=8), or hypocaloric diet (N=3). This ongoing study is blinded, so the effects of liraglutide and sitagliptin are combined in analyses and referred to as "drug". Subcutaneous abdominal AT and peripheral blood mononuclear cells (PBMCs) were collected at baseline ("pre") and after 12 weeks of therapy ("post"). Phenotypic marker expression of blood and AT T cells were characterized by flow cytometry. Whole AT inflammatory gene expression in the pre and post groups was assessed by Nanostring.

Results: Using the Nanostring inflammation panel, we found that a number of pro-inflammatory genes were significantly downregulated in whole AT after treatment with drug, including CD163, CD86, CCR1, MCP-2, and MCP-4. Blood ILC2s were significantly decreased with drug treatment (pre 3.95%±3.05, post 1.71% ±1.65, p=0.01), but not diet (pre 2.17% ±1.91, post 1.46% ±1.68, p=0.18). We did not detect a change in Treg numbers after treatment with either diet (pre 5.78% ±1.91, post 6.09% ±1.50, p=0.29) or drug (pre 6.49% ±2.29, post 5.94% ±2.15, p=0.12). Similarly, no difference in blood iNKT numbers was detected after diet (pre 0.063% ±0.044, post 0.082% ±0.049, p=0.11) or drug (pre 0.077% ±0.106, post 0.091% ±0.130, p=0.67).

As observed in the PBMCs, adipose ILC2s were decreased after drug (pre 2.04% ±1.67, post 1.32% ±1.58, p=0.07, N=4). We did not detect a change in AT Treg and iNKT numbers (Treg pre 9.24% ±5.36, post 6.23% ±1.55, p=0.14; iNKT pre 0.12% ±0.08, post 0.09% ±0.05, p=0.47, N=4).

Conclusions: In a small pilot study of obese pre-diabetic patients treated with drugs that activate GLP-1 signaling (liraglutide or sitagliptin) or hypocaloric diet, global transcriptional analysis of whole AT suggested decreased inflammation with drug therapy. However, we found decreased percentages of ILC2 cells (considered anti-inflammatory in adipose) in both blood and AT after drug treatment. Future experiments will further characterize the function of these cell types, and evaluate other immune subsets in PBMC and AT that may be responsible for decreasing inflammation.

Adrenal

ADRENAL - CORTISOL EXCESS AND DEFICIENCIES

Clinical Features of Immune Checkpoint Inhibitor-Related Adrenal Insufficiency: A Retrospective Analysis

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