

macroadenoma, with features of acromegaly and hyperthyroidism. **Case report:** A 75 years' old man presented with new onset atrial fibrillation. He had high FT4 with normal TSH. His ultrasound scan of the neck showed a solitary nodule. He had ablation twice and was started on bisoprolol and anticoagulant. He had MRI scan for headaches and this showed a pituitary macroadenoma. He had high IGF-1. His oral glucose tolerance showed failure of GH suppression. His FT4 was persistently high with normal TSH and he had high  $\alpha$  subunits. This suggested the diagnosis of TSH and GH secreting pituitary adenoma. **Discussion:** TSH-secreting pituitary adenomas are rare and not uncommonly, they co-secrete other pituitary hormones including growth hormones. Somatotrophs and lactotrophs share common transcription factors with thyrotrophs. TSH-secreting adenomas are benign but 60% of them are locally invasive. TSH-secreting pituitary adenomas typically present with either symptoms of tumor growth like headache or visual field disturbance or symptoms of hyperthyroidism. Thyroid nodules are common in patients with TSHomas. In patients with TSH-secreting pituitary adenomas, majority will need only surgery and radiation. The medical treatment used to normalize TSH and FT4 levels is somatostatin analogs. This is effective in about 90% of patients with TSH secreting pituitary adenomas TSHoma should be differentiated from resistance to thyroid (RTH). The main difference between TSHoma and RTH is the presence of signs and symptoms of hyperthyroidism in patients with TSHoma, absence of a family history, normal thyroid hormone levels in family members, and the presence of an elevated glycoprotein  $\alpha$ -subunit in patients with pituitary tumor. **Reference:** H Adams and D Adams. A case of a co-secreting TSH and growth hormone pituitary adenoma presenting with a thyroid nodule. EDM case reports 2018 Hatem.eid1@nhs.net

## Diabetes Mellitus and Glucose Metabolism

### DIABETES COMPLICATIONS II

#### *Might Podocyturia Be an Early Marker for Diabetic Nephropathy in Males*

Emre Durcan, MD<sup>1</sup>, Ozge Polat Korkmaz, MD<sup>1</sup>, Ahmet Murt, MD<sup>2</sup>, Halil Ibrahim Saygi, PhD<sup>3</sup>, Serdar Sahin, MD<sup>1</sup>, Tamer Dincer, MD<sup>2</sup>, Serbay Ozkan, PhD<sup>3</sup>, Alev Bakir, Asst. Prof.<sup>1</sup>, Hande Mefkure Ozkaya, MD<sup>1</sup>, Sinan Trabulus, MD<sup>2</sup>, Emine Elif Guzel Meydanli, MD<sup>3</sup>, Nurhan Seyahi, MD<sup>2</sup>, Mustafa Sait Gonen, MD<sup>1</sup>.

<sup>1</sup>Division of Endocrinology and Metabolism, Department of Internal Medicine, Cerrahpasa Medical Faculty, Istanbul University-Cerrahpasa, Istanbul, Turkey, <sup>2</sup>Division of Nephrology, Department of Internal Medicine, Cerrahpasa Medical Faculty, Istanbul University-Cerrahpasa, Istanbul, Turkey, <sup>3</sup>Department of Histology and Embryology, Cerrahpasa Medical Faculty, Istanbul University-Cerrahpasa, Istanbul, Turkey.

#### MON-680

Renal involvement can develop before detection of microalbuminuria in type 2 diabetes. There is an interest in finding biomarkers to detect diabetic nephropathy (DN) earlier and identify progression risk. Podocyturia emerge

as a marker for early kidney damage however standardization problems hamper its widespread use. We aimed to investigate the value of podocyturia for the detection of early DN. Herein we report our preliminary results.

Our study population was composed of three type 2 diabetic patient groups and a healthy control group. Diabetic groups were defined as follows; group 1: patients without microalbuminuria who had HbA1c <7%; group 2: patients without microalbuminuria who had HbA1c > 8.5%; group 3: patients with diabetic retinopathy who had proteinuria >1g/day and/or microalbuminuria >300 mg/day and group 4: healthy volunteers without any known disease. Patients with glomerular filtration rate (GFR) below 30 ml/min were excluded.

GFR was calculated using the abbreviated MDRD formula. Microalbuminuria was measured in 24 hour urine. Number of podocytes in the urine was determined by immunocytochemical staining of podocalyxin. Due to the known expression of podocalyxin in the female genital tract, only males were included. Statistical analyses were carried out using Statistical Package for the Social Sciences version (SPSS) 24.0 and statistical significance was set as p<0.05.

We examined a total of 119 patients (mean age 57.35 ± 12.75 yrs.). Patient distribution in each group was as follows; group 1: 24(20%); group 2: 26(22%); group 3: 24(20%) and group 4: 45(38%) patients. There was no significant difference in mean age (p=0.582) and duration of diabetes (p=0.517) between the diabetic groups. The mean GFR was significantly lower in group 3 than in group 1 and 2 (p<0.001, p<0.007; respectively). The median podocyte measurement in urine was 0,25 (IQR: 0- 2.68) podx/ml in group 1; 0,37 (IQR: 0-2.12) podx/ml in group 2; 1,37 (IQR: 0.56-5.18) podx/ml in group 3; 0.0 (IQR: 0-0.75) podx/ml in group 4. The mean number of podocytes in urine was significantly different between the 4 groups (p=0.001). In posthoc analysis with Bonferoni correction, the mean podocytes measurement was significantly higher in group 3 than in group 1 and 4 (p=0.033, p=0.001; respectively).

According to our preliminary results; podocyturia assessed by podocalyxin immunostaining does not seem to be increased in male diabetic patients without proteinuria. Further studies on larger patient groups and using different podocyte markers might clarify the value of podocyturia as an early marker of DN.

## Bone and Mineral Metabolism

### BONE AND MINERAL CASE REPORTS I

#### *Pyridoxal 5'-Phosphate Cerebrospinal Fluid Abnormalities in Hypophosphatasia Before and After Enzyme Replacement Therapy*

Jordan J. Wright, MD, PhD<sup>1</sup>, Jiun-Ruey Hu, MD, MPH<sup>1</sup>, Zahra Shajani Yi, PhD<sup>1</sup>, Kathryn McCrystal Dahir, MD<sup>2</sup>.

<sup>1</sup>Vanderbilt University Medical Center, Nashville, TN, USA, <sup>2</sup>Vanderbilt Univ, Nashville, TN, USA.

#### SAT-382

**Introduction:** Hypophosphatasia (HPP) is an inborn error of metabolism due to deficiency of tissue non-specific alkaline phosphatase (TNSALP), characterized by a wide range