An unusual cause of polyuria

Sir,

Thyrotoxicosis due to Graves' disease is an uncommon albeit known cause of polyuria. Mechanism underlying this manifestation has been studied in rat models. We present a young male who presented with polyuria as the first manifestation of thyrotoxicosis.

A 29 year old male presented with history of passing excessive urine of 1-month duration. He was passing urine about 7-8 times during day and 4 - 5 times during night. This was associated with excessive thirst and loss of weight of 6 kg with normal appetite. He denied having fever, headache, visual symptoms, heat intolerance or hyperdefecation. He was not taking any drugs prior to these symptoms. On examination, his weight was 60 kg (66 kg premorbid), pulse 106/min, regular, fine finger tremors, soft diffuse goiter grade II with no ophthalmopathy or markers of autoimmunity. Urine volume estimation was 5500 ml in 24 hrs suggestive of polyuria with water intake of 5750 ml. On investigation, he had thyrotoxicosis (TSH < 0.01 mIU/l, total T4 - 14.5 mcg/dl, T3 – 400 ng/dl). Urine for spot sodium was 68 mMol/l. Technitium Pertechnate scan for thyroid showed diffuse increased uptake 7% suggestive of hyperthyroidism. Rest of the biochemical parameters including blood glucose levels, serum electrolytes and renal functions were normal. On water deprivation, he showed normal response with urine concentrating to Urine osmolality of 618 mOsm/kg with calculated plasma osmolality of 286 mOsm/kg ruling out diabetes insipidus. He was started on carbimazole 30 mg daily. On evaluation after 8 weeks, he was clinically and biochemically euthyroid (TSH 2.97 mIU/l). He had regained 4 kg weight and his daily urine output had reduced to 2000-2400 ml over 3 days.

Our patient was a case of Graves' disease with thyrotoxicosis who presented with polyuria and natriuresis as initial presentation, which completely resolved on achievement of euthyroid status. Other causes of polyuria were excluded.

Polyuria is a known albeit uncommon manifestation of thyrotoxicosis.[1,2] The mechanism of polyuria in Graves' disease is not clear. It has been noted in rats induced thyrotoxic by thyroxine injection that there is increased mean arterial pressure and renal blood flow. Excessive water intake along with increased solute excretion was also noted in them. This was associated with significant decreases in Aquaporin (AQP1) and AQP2 water channel expression in renal cortex and medulla. Thyrotoxicosis was also associated with increased Na- K-2Cl cotransporter expression in these rats. Arginine vasopressin (AVP) - independent downregulation of aquaporins, the observed increase in renal arterial pressure, and decrease in filtration fraction are thought to be contributing to polyuria and increased solute excretion in spite of enhanced ion transporters in thyrotoxicosis.[3] Similar studies in human are lacking. Lymphocytic hypophysitis rarely associated with Graves' disease may cause central diabetes insipidus which may present with polyuria in such a setting.[4] This was ruled out by a negative water deprivation test in our patient.

Polyuria should be recognised as a presenting feature of thyrotoxicosis after ruling out commoner causes like hyperglycemia, diuretics and diabetes insipidus.

Jayaraman Muthukrishnan, Dawra Saurabh[1]

Departments of Medicine and Endocrinology, and [1]Medicine, Command Hospital, Chandimandir, Panchkula, Haryana, India

Corresponding Author: Dr. J. Muthukrishnan, Department of Medicine and Endocrinology, Command Hospital, Chandimandir, Panchkula, Haryana, India.
E-mail: jmuthukrishnan@hotmail.com

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