Hypernatraemia with reset osmostat associated with secondary hypogonadism

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Introduction

We report two cases of hypernatraemia with reset osmostat and evidence of abnormal pituitary function.

Case 1

- A 35-year-old male was referred with Graves' thyrotoxicosis associated with hypokalaemic periodic paralysis and an incidental finding of serum sodium 154mmol/L
- He complained of a long history of polyuria and nocturia, denied excessive thirst and was otherwise well
- Height was 193cm with BMI 29.5
- He had gynaecomastia and sparse body hair
- He had a small 6ml right testicle (originally undescended) and 15ml left testicle
- A very high arched, possibly cleft palate was noted suggesting a midline defect (Figure 1)

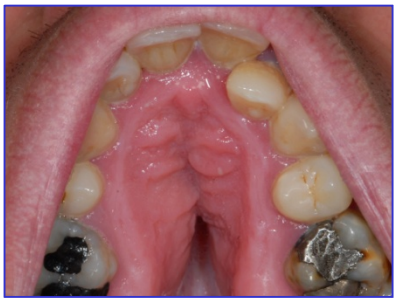

Figure 1. High arched palate

Investigations

- Serum osmolality was 314mOsm/kg with urine osmolality 1025mOsm/kg
- Testosterone 5.0nmol/L (9.9-27.8), LH 5.6IU/L (1.7-8.6) and FSH 5.0IU/L (1.2-12)
- Prolactin, IGF-1, synacthen test, renin and aldosterone were normal
- Pituitary MRI was normal
- He had undergone a water load/deprivation test (Table 1)

Time	Plasma Osmolality (mmol/kg)	Urine Osmolality (mmol/kg)	Plasma AVP (pg/ml)
10:00	303	891	1.3
11:00		387	
12:00		245	
13:00		462	
14:00	304	866	2.5
15:00		916	
16:00		946	
17:00	304	930	2.4

Table 1. The water load/deprivation test shows a prompt reduction in urine osmolality in response to the water load and urine concentration then increased during subsequent fluid deprivation. There is reasonable evidence of osmoregulatory control over AVP.

Case 2

- An 18-year-old male was admitted following an incidental finding of serum sodium 163mmol/L whilst being investigated for joints pain
- He otherwise feels well and denied excessive thirst or polyuria
- Examination showed BMI 40.4, short stature (154cm), small hands, size 6 feet and gynaecomastia
- Testicular volumes were 15ml bilaterally

Investigations

- Serum osmolality was 330mOsm/kg, urine osmolality 731mOsm/kg and urine sodium 172mmol/L
- Initial hospital treatment with 5% Glucose reduced his serum sodium to 152mmol/L and urine osmolality to 114mOsm/kg
  His urine output increased to 3 litres/day whilst he remained hyperosmolar with a calculated serum osmolality of 316mOsm/kg
- Prolactin was 2000mIU/L (86-324), LH 6.3IU/L (1.7-8.6), FSH 1.0IU/L (1.2-12) and Testosterone 5.7nmol/L (9.9-27.8)
- IGF-1 was low with peak GH 4.5ug/L on ITT
- TSH, FT4 and synacthen test were normal
- Pituitary MRI was normal

Discussion

- Both cases of hypernatraemia were incidentally found in asymptomatic patients
- Increased urine output and reduced urine osmolality following a fluid load was demonstrated in both cases whilst they remained hypernatraemic and hyperosmolar
- In the first case, there was direct evidence of osmoregulatory control over AVP levels consistent with reset osmostat
- In both cases there was evidence of pituitary dysfunction
- A congenital midline defect is the most likely cause in the first case
- The second patient has been commenced on dopamine agonist therapy
- Both patients have been maintained on a fixed daily fluid intake

Conclusion

- **"Essential hypernatraemia" is rare and usually secondary to traumatic brain injury or surgery**
- **It represents a complex diagnostic challenge which needs to be made to avoid subsequent misinterpretation of the fluid status**