CASES

A 3-year-old female presented with anterior neck swelling, weight loss, and persistent tachycardia. Biochemical evaluation confirmed severe thyrotoxicosis (T4 152.3 pmol/L, TSH <0.008 mIU/L) with markedly elevated anti-thyroglobulin and anti–thyroid peroxidase antibodies. Thyroid ultrasonography demonstrated a diffusely enlarged, hypervascular gland with nodular changes, consistent with Graves' disease. During longitudinal follow-up, she developed polyuria, polydipsia, and hyperglycemia, with positive pancreatic autoantibodies confirming type 1 diabetes mellitus. Notably, serial early morning cortisol measurements were persistently low, raising concern for evolving adrenal insufficiency and suspicion of autoimmune polyglandular syndrome type 2. Comprehensive endocrine evaluation is ongoing.

A 4-year-old male presented with a 1-year history of recurrent self-limiting fever followed by significant fatigability for 1 month. Salient examination on admission revealed tachycardia, anterior neck swelling, and an incidental pansystolic murmur. Thyroid function tests confirmed thyrotoxicosis (T4 38.6 pmol/L, TSH <0.008 mIU/L). Serial serum glucose was markedly elevated on admission, prompting further testing that confirmed type 1 diabetes mellitus with positive pancreatic autoantibodies. Echocardiography demonstrated moderate-to-severe mitral regurgitation with heart failure secondary to prolonged uncontrolled hyperthyroidism state. Cardiac function improved with restoration of a euthyroid state, highlighting the reversible nature of thyrotoxic heart failure.

CONCLUSION

These cases highlight the progressive nature of childhood autoimmunity, where Graves' disease may precede the development of type 1 diabetes mellitus and evolving features of autoimmune polyglandular syndrome type 2. They further emphasize that thyrotoxic heart failure is potentially reversible with restoration of a euthyroid state, underscoring the importance of early recognition and vigilant long-term surveillance.

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NOT JUST DEHYDRATION: A CASE OF EARLY ONSET PERSISTENT HYPERNATREMIA IN A PRETERM BABY

Sin Yin Gan[1], Wan Nurzahiah Wan Zakaria[1], Zurina Zainudin[1], Yee Lin Lee[1]

[1]Department of Paediatrics, Hospital Sultan Abdul Aziz Shah, Universiti Putra Malaysia

INTRODUCTION

Hypernatremia in preterm babies is often due to insensible water loss, inadequate fluid intake or sodium imbalance due to immature kidneys. Congenital nephrogenic diabetes insipidus (CNDI) is an uncommon cause of hypernatremia in neonates and presents shortly after birth. Early recognition is important to prevent severe dehydration, electrolytes imbalance and neurological complications.

CASE

We report a case of a preterm 32-week female infant, with a birth weight of 1.14 kg with persistent hypernatremia (146–156 mmol/L) from day 4 of life. She received total parenteral nutrition since birth and was started on breastmilk since day 5 of life. The baby was mildly dehydrated with weight loss and high urea (6.5 mmol/L) at day 7 of life. Total fluids were increased to 160 mL/kg/day but serum sodium remained elevated even though the urea had normalized. The infant was also noted to have high urine output (5–6 mL/kg/hour) since day 3 of life and suboptimal weight gain.

Further evaluation at age 1 month revealed urine osmolality 71 mOsm/kg, serum osmolality 308 mOsm/kg and urine sodium <20 mmol/L when serum sodium was 150 mmol/L, suggestive of DI. A trial of desmopressin showed unchanged serum sodium (Na), suggesting nephrogenic DI. Administration of intravenous fluids of 1/5NSD10% resulted in further increase of both sodium (159 mmol/L) and serum osmolality (326 mOsm/kg) with low urine osmolality (111 mOsm/kg). Intravenous fluids were discontinued and she was started on hydrochlorothiazide (1 mg/kg/dose bd), resulting in gradual normalization of sodium 138 mmol/L. Post discharge, she had good weight gain and normal developmental milestones at corrected age of 1.5-month-old. Due to early onset hypernatremia, the infant was referred for genetic testing to rule out CNDI.

CONCLUSION

Early onset persistent hypernatremia and high urine output despite adequate fluid management should prompt evaluation of DI. Early diagnosis is crucial to prevent a chronic state of hypernatremia that can lead to growth and developmental delay.