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ADIPSIC DIABETES INSIPIDUS IN LOCALLY ADVANCED NASOPHARYNGEAL CARCINOMA

https://doi.org/10.15605/jafes.036.S37

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INTRODUCTION

Pituitary metastasis in nasopharyngeal carcinoma (NPC) is extremely rare with early detection and presentation of NPC. Up to 30% of patients present with diabetes insipidus (DI). The inability to sense thirst or adipsic DI may result in electrolyte imbalance and hypovolemia.

RESULTS

A 29-year-old woman presented with a short history of dysphagia and hoarseness of voice followed by a week of right eye and facial swelling. Further enquiry revealed a one-year history of unexplained epistaxis and loss of appetite, and weight loss of 5 kg in one month. Physical examination revealed right cranial nerve II to VI, VIII and bulbar palsies. Nasopharyngeal assessment showed a mass over the right posterior choana with right vocal cord palsy. She was admitted to the ENT ward for a suspicion of nasopharyngeal carcinoma with intracranial extension. Initial preoperative medical review revealed persistent tachycardia and significant polyuria. Subsequent diagnosis of central DI was confirmed with findings of high serum osmolality, low urine osmolality and hypernatremia. Tachycardia was attributed to hypovolemia and responded to fluid correction. Intermittent doses of desmopressin controlled urine output and improved hypernatremia. As the patient could not sense thirst, she was unable to actively consume fluids. There was extreme difficulty in normalising her sodium and maintaining hydration. Delicate balance of supervised fluid consumption with supportive intravenous fluids and desmopressin was required. Cranial MRI later confirmed a locally advanced nasopharyngeal tumour with pituitary gland and stalk metastasis and invasion into both cerebello-pontine angles and hypothalamus. Her initial anterior pituitary hormone function was intact.

CONCLUSION

DI in a locally advanced nasopharyngeal carcinoma is a poor prognostic indicator. The presence of adipsic diabetes insipidus further complicate the delicate management of hydration and sodium balance in such patient.

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NEPHROGENIC DIABETES INSIPIDUS AND RHABDOMYOLYSIS WITH SEVERE HYPERNATREMIA REQUIRING HEMODIALYSIS TREATMENT

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INTRODUCTION

Severe hypokalaemia may present with respiratory muscle paralyisis and respiratory failure. In rare instances, it results in rhabdomyolysis and nephrogenic diabetes insipidus (NDI). The management of NDI with severe hypernatraemia, metabolic acidosis and renal impairment is challenging and potentially complicated.

RESULTS

A 21-year-old male presented with acute shortness of breath, fever, cough and generalised body weakness. He had severe hypokalemia (potassium level of 2.0 mmol/L) and compensated metabolic acidosis. He rapidly deteriorated requiring ventilatory support and ICU admission. His hypokalaemia was resistant to correction and he required repeated bolus potassium chloride correction. His increasing serum sodium trend subsequent polyuria (urine output between 150 to 200 mL/hour prompted the suspicion of DI. Urine output was reduced only after high dose intravenous desmopressin 4 µg. Simultaneously, he required regular potassium supplementation via intravenous and nasogastric route. Peculiarly, the patient had dark-coloured urine, which increased in intensity after desmopressin. Elevated creatinine kinase and myoglobinuria indicated rhabdomyolysis. Serum sodium trend continued to increase to a peak of 179 mmol/L. Coupled with metabolic acidosis, haemodialysis was opted as a method to reduce the sodium level. Following haemodialysis, sodium level gradually decreased with normalisation of urine output and potassium level. He made a remarkable recovery and was discharged well. Three weeks after discharge, he was readmitted for symptomatic hypokalemia with normal anion gap metabolic acidosis. The final diagnosis of renal tubular acidosis (RTA) was ascertained.

CONCLUSION

Undiagnosed RTA resulted in severe hypokalemia that led to life threatening respiratory depression, rhabdomyolysis and NDI. Haemodialysis in specific situations can be used as treatment for severe hypernatraemia.