

Case Report

Central diabetes insipidus (CDI) in very low birth weight neonate: case report

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Abstract

Diabetes Insipidus (DI) is rare, with an estimated incidence of 1 in 25,000 cases; less than 10% of cases are hereditary. CDI accounts for over 90% of DI cases and can appear at any age, depending on the cause. There is no indication that hereditary CDI causes are frequent. Central diabetes insipidus (CDI) is uncommon in neonates, particularly in very low-birth-weight newborns, and the great majority of cases are caused by disorders such as ischemia damage or bleeding brain injury. CDI is caused by a deficiency of antidiuretic hormone (ADH) as a result of posterior pituitary and/or hypothalamus dysfunction. Our kid was delivered in Sultan Qaboos Hospital in Salalah at 28 weeks gestational age, with a very low birth weight of 1001 grams. She also suffered polyuria and hypernatremia. The baby was identified with central diabetes insipidus early on and was effectively treated with oral desmopressin.

Keywords: Central diabetes insipidus; Desmopressin; Sultan Qaboos Hospital; Salalah

Introduction

Diabetes Insipidus (DI) is rare, with an estimated incidence of one in every 25,000 people; less than 10% of cases are hereditary. CDI accounts for over 90% of DI cases and can appear at any age, depending on the cause. There is little indication that hereditary CDI causes are widespread [1,2].

Central diabetes insipidus (CDI) is uncommon in neonates, particularly in very low-birth-weight infants, and the great majority of cases are caused by disorders such as ischemia damage or bleeding brain injury [3-7].

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Citation: Abuhamda AFM (2023) Central diabetes insipidus (CDI) in very low birth weight neonate: case report. J Neonatol Clin Pediatr 10: 107.

Received: May 26, 2023; **Accepted:** June 15, 2023; **Published:** June 21, 2023

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CDI is caused by a deficiency of antidiuretic hormone (ADH) as a result of posterior pituitary and/or hypothalamus dysfunction. CDI is an uncommon disease that causes increased urine production and hypernatremia. Infant water and electrolyte balance are affected by postnatal physiological adaptations, and proper treatment is crucial, especially for severely ill newborns. Dehydration with hypernatremia can occur in neonatal CDI cases, followed by insensible water loss, increased urine output, and a reduced capacity to release salt. Meticulous monitoring is essential to maintain hydration and electrolyte balance in new-born CDI [8].

Background

When caring for a very preterm newborn with a low birth weight, it is critical to maintain optimal hydration and electrolyte balance. Our situation was difficult since she received appropriate hydration and salts but still had hypernatremia and polyurea. The case was diagnosed at the time and treated satisfactorily with oral desmopressin.

Case presentation

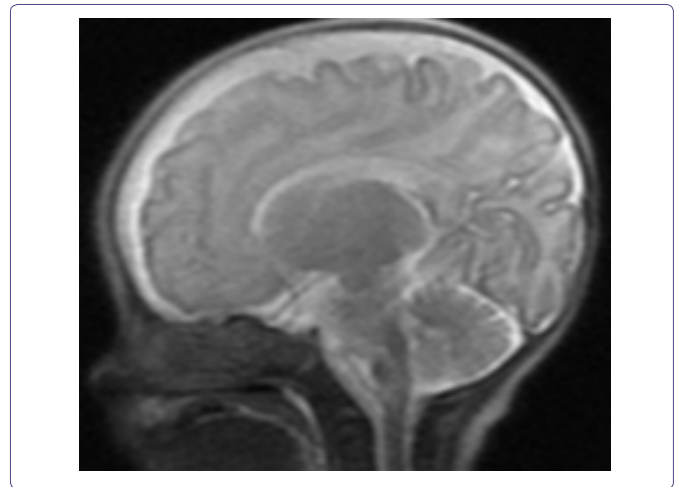
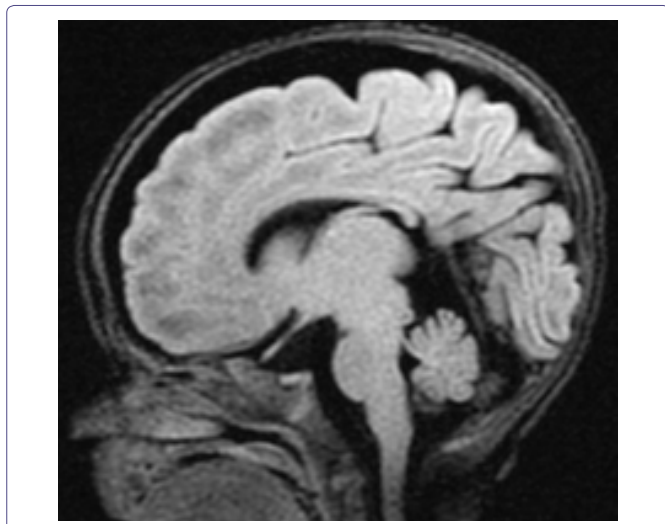
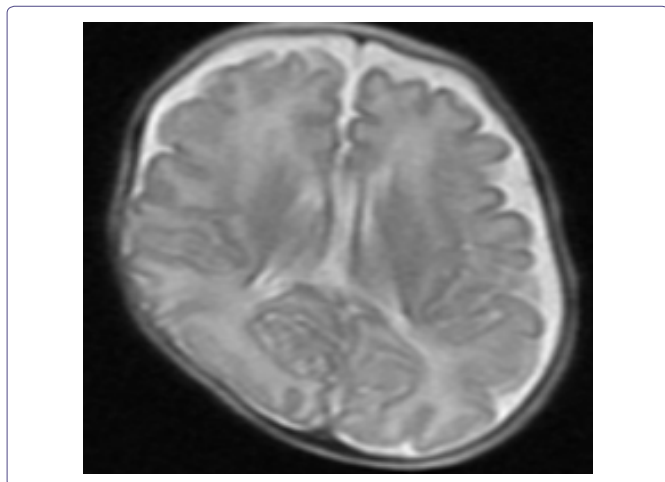
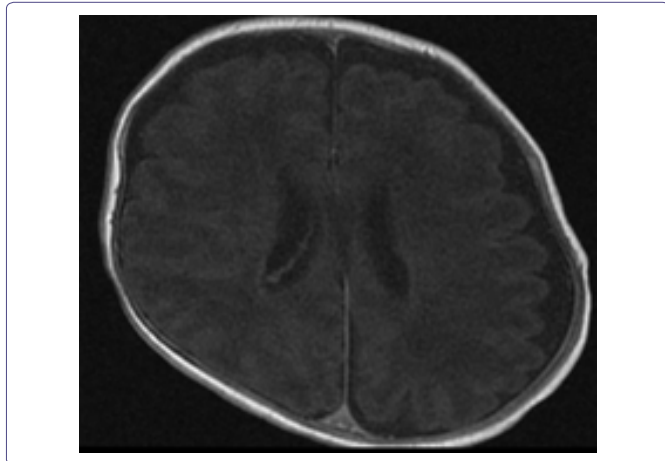
The parents were not consanguineous, and there was no family history of diabetes insipidus. The mother was G5P2, had gestational diabetes, had a preterm rupture of membranes for 7 days, and was given two doses of dexamethasone before birth. She was a girl baby born at 28 weeks gestational age through normal delivery and had a birthweight of 1001 grams on the 50th percentile, a circumference of the head of 25 cm on the 50th percentile, and a length of 33 cm above the 10th percentile. She lacked dysmorphic characteristics. The newborn was intubated in the birth room, then transferred to the NICU and connected to mechanical ventilation. The baby got two doses of surfactant and was kept on mechanical ventilation for 17 days before being extubated. The baby required IV inotropes throughout the first four days of life owing to hemodynamic instability, initial screening brain U/S revealed IVH grade 1, and all blood cultures were negative.

The newborn got enough fluid, receiving 100 ml/kg/day on the first day of birth and 190 ml/kg/day on the third day of life, with polyuria up to 10 ml/kg/h and serum sodium of 159 mEq/L (a typical range of 135 to 145). The newborn had received 260 ml/kg/day IV fluid by the age of 10 days, serum sodium was 158 mEq/L, urine output was 9 ml/kg/hr, and urine specific gravity was 1.0050 (normal range, 1.002 to 1.030). The baby was diagnosed with diabetes insipidus at this age. Staff attempted to manage the case by increasing fluid intake and compensating for polyurea, but the case was still uncontrolled at 25 days of age while the infant required 290 ml/kg/day of fluid, serum sodium was 151 mEq/L, and the baby had polyuria. The paediatric endocrinology team suggested that desmopressin be given to the patient. At 28 days, the newborn took oral desmopressin 3 microgram bid, as well as the dose, was titrated based on the baby's overall condition, oedema, urine output, and serum sodium in blood gases.

Oral desmopressin allowed blood sodium to be kept within a normal range with minimum variation, while also providing the infant with an average fluid intake and controlling polyuria. Initially, the

case required periodic blood gases, at least twice daily, to monitor serum sodium and regulate the dose of desmopressin by increasing or decreasing the dose.

Brain MRI [Images 1-4] at the age of 78 revealed that generalized relative simplified gyral pattern, generalized enlarged subarachnoid spaces as well as enlarged bilateral sylvian fissures, no diffusion restriction, no intracerebral haemorrhage, pituitary fossa appears normal in size, the posterior pituitary spot is not well defined.



The infant was released home at the age of 82 days; she was a feeder and grower, weight was 2110 grams, and head circumference was 33.5 cm, active, sucking well, and breathing effortlessly in room air. She was discharged on desmopressin 4 micrograms orally supplied, and serum sodium was regulated with the same dose of desmopressin for several days before discharge.

The mother was taught how to care for her infant, how to evaluate hydration status, how to prepare, and how to administer desmopressin. The infant had been scheduled for appointments at the paediatric endocrinology clinic and the new-born clinic.

Discussion

Since her admittance, this infant has got standard care for a very preterm and very low birth weight neonate, and she has received average intravenous fluids since the first day. By day three, the baby had received 190 ml/kg/day IV fluid and had polyurea and serum sodium of 159 mEq/L. Since admission, the baby's neurological status has been stable; a simple screening of the brain revealed an IV grade 1, blood sugar and serum electrolytes were normal, the baby had normal saturation throughout the hospital stay and was not subjected to hypoxia; just for the first four days of her life, she required inotropes, due to hemodynamic instability which she then discontinued.

By 10 days, the infant was still on high IV fluid, had polyuria and hypernatremia, and her urine specific gravity was 1.0050, confirming the diagnosis of diabetes insipidus [9].

The team managed the case by hydration and replacing urine output with IV fluid and feeding, but when we reached 290 ml/kg/day of IV fluid, the kid was still polyureic exhibiting hypernatremia. We began with desmopressin 3 mcg bid (tablet 200 mcg dissolved in 20 cm water) with strict monitoring of hydration status, urine output, and capillary blood gases for sodium before the dose since the infant was very low birth weight and was still fragile.

We were able to manage hypernatremia and polyuria with desmopressin and weaned the infant from a high fluid intake to a normal fluid intake per day [10-14]. The desmopressin response validated the diagnosis of central diabetes insipidus. Brain MRI data were also indicative of a central origin, particularly the posterior pituitary spot, which was not well delineated. In comparison with previously reported cases, we controlled the case with a little dosage of oral desmopressin, and oral desmopressin is more practical than intranasal

dDAVP treatment [15,16], and oral desmopressin administration and modification of dosage is easier than nasal application [17].

Conclusion

A very premature infant born at 28 weeks gestational age with a birth weight of 1001 grams was diagnosed with central diabetes insipidus and treated safely and effectively with oral desmopressin. Desmopressin therapy necessitates frequent monitoring of hydration status, polyuria, serum sodium, and dosage calibration as needed.

Consent

Approval from hospital administration of Sultan Qaboos Hospital in Salalah (SQH) and consent was signed by the father for publication of brain MRI images and medical details of the case.

Conflict of Interest

Author declares no conflict of interest.

Acknowledgments

The author is grateful to all medical staff; doctors and nurses, in the neonatal intensive care units, for their exceptional care of a so complicated cases.

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