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LETTER TO THE EDITOR

Extensive subclinical venous sinus thrombosis in the dehydrated infant

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Sir,

Hypernatremic dehydration is a common presentation in the neonate who has failed to establish breastfeeding. Cranial venous sinus thrombosis is a rare and serious complication of hypernatremic dehydration in the neonate. We describe a case of a neonate presenting in a hypernatremic, hyperosmolar state secondary to severe dehydration. Despite the absence of any neurological signs or symptoms, the child developed an extensive venous sinus thrombosis. We propose that venous sinus thrombosis secondary to dehydration may be more prevalent than previously thought due to its subclinical nature and suggest a high clinical suspicion of cerebral venous thrombosis in infants with severe hypernatremic dehydration, even in the absence of neurological symptoms or signs.

An 8-day-old male infant presented to the Accident and Emergency department with a history of poor feeding. He was reported to have been 'floppy' that morning. He was born at term weighing 3 kg and was discharged home on day 2 of life. Ante and perinatal history was unremarkable. A detailed history revealed that the infant's mother had been struggling to establish breast feeds. Postnatal assessments by the community midwife had been missed on two consecutive days.

On examination the patient had signs consistent with severe dehydration. He was floppy and lethargic with a sunken fontanelle, decreased skin turgor, dry mucous membranes and capillary refill time of greater than 5 s. He had lost 23% of his birth weight. He was breathing spontaneously but hadperiodic, spontaneously resolving apnoeic episodes. Access was established by inserting an intraosseous needle and two 20 ml/kg fluid boluses were administered. The patient became more responsive but continued to have apneas. He was intubated and transferred to Pediatric Intensive Care (PICU). His biochemistry confirmed hypernatremic dehydration with an initial serum sodium of 165 mmol/l and urea of 25 mmol/l. The patient received another 40 ml/kg of fluid to restore perfusion and was then started on an enteral rehydration regime (as per pediatric department protocol) to slowly correct the serum sodium over the subsequent 48 h. His presenting platelet count was 91.

After biochemical parameters had been corrected, the patient was extubated uneventfully on day 3 of admission and showed no signs of neurological sequelae such as seizures or abnormal tone or posturing. There were no clinical signs of end-organ damage. However, it was noted that the platelet count had decreased further to 72 on day 3. This raised the concern of an underlying thrombosis and an MRI of the brain was arranged urgently. The MRI showed extensive venous sinus thrombosis with involvement of superior sagittal, transverse and sigmoid sinuses [Figures 1(a) and 1(b)]. The patient remained clinically stable with no neurological signs. A repeat MRI on day 7 showed persistent thrombosis. There were no signs of thrombosis in any other sites.

Following discharge from PICU, the pediatric neurologists anticoagulated the patient, starting on subcutaneous heparin at an initial dose of 75 IU/kg twice daily, which was subsequently increased to 150 IU/kg. After the patient had established adequate breastfeeding and the parents had been educated in administration of subcutaneous heparin injections, the patient was discharged home.

Regular neurodevelopmental follow-up and outpatient monitoring of Factor Xa levels was arranged. To date, at 3 months post-event there is no evidence of neurological sequlae. A repeat MRI at 3 months showed that all the major sinuses had re-opened and that the brain parenchyma looked normal. A full coagulopathy and thrombophilia screen demonstrated no abnormalities, and therefore it was considered that this thrombotic event was directly attributable to the hypernatremic dehydration.

In a review of the literature there are reported cases of venous sinus thrombosis associated with hypernatremic dehydration. Karadag et al. [1] reported a case of sagittal sinus thrombosis as a complication of hypernatremic dehydration in a 3-day-old girl. Subsequently Fawke and Whitehouse [2] reported three cases of infants with cranial venous thrombosis secondary to hypernatremic dehydration. Prior to this, we could find only one other report of cranial sinus thrombosis directly related to hypernatremic dehydration [3]. Our patient differs from the reported cases in that he did not develop seizures or have any clinical neurological signs. Our only reason to suspect the diagnosis was the presence of a low platelet count in the presence of severe hypernatremic dehydration.

Hypernatremic dehydration as a complication of breastfeeding is showing an increasing incidence [4]. This case highlights a severe complication of hypernatremic dehydration. While 'breastfeeding is best', hypernatremic dehydration as a result of inadequate breast feeding may go undetected until the infant becomes symptomatic. Inexperienced parents may be unaware of or mis-interpret the worrying signs of dehydration. Professional surveillance

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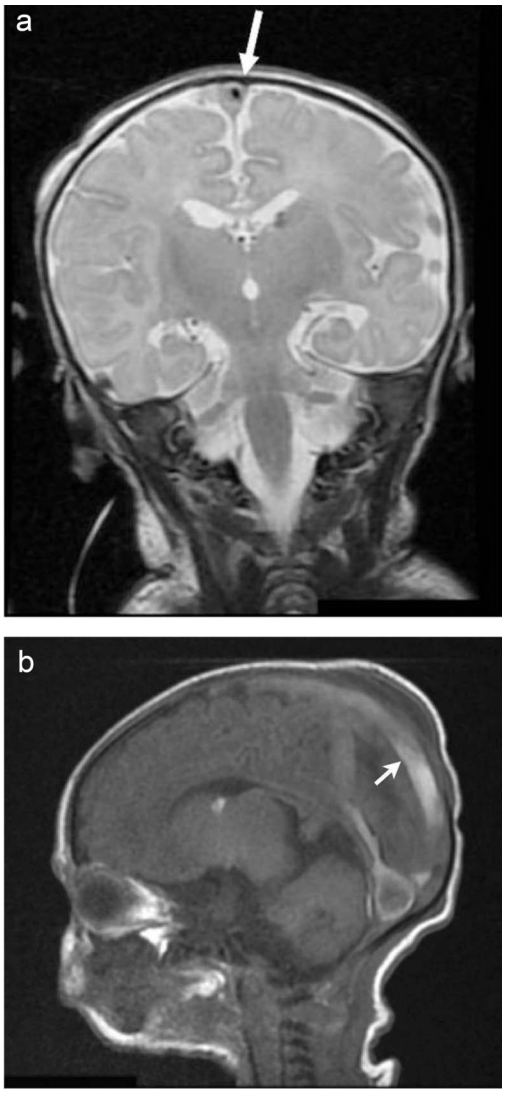

Figure 1. (a) Coronal T2-weighted MRI showing saggital sinus thrombosis (arrow). (b) Saggital T1-weighted MRI showing saggital (arrow) and straight sinus thrombosis.

including the weighing of a newborn infant and appropriate intervention in the first few days of life is essential in preventing hypernatremic dehydration. The importance of this has been previously highlighted [2], and as pointed out in Karadag et al.'s letter, the American Academy of Pediatrics has recommended that all breastfeeding newborns should be evaluated and weighed by a pediatrician or other knowledgeable health care professional within 3–5 days of age [1,5].

We feel this case highlights the need for high clinical suspicion of cerebral venous thrombosis in infants with severe hypernatremic dehydration, even in the absence of neurological symptoms or signs. We postulate that there may be a cohort of asymptomatic infants with undiagnosed cerebral venous thrombosis secondary to hypernatremic dehydration. Whether such a diagnosis has long-term implications or even requires treatment remains to be established.

We suggest that neonates presenting with severe hypernatremic dehydration should have brain imaging; further studies in evaluating the threshold for such imaging are needed. Longitudinal neurodevelopmental follow-up of babies with hypernatremic dehydration is required to gain further insight into the effects of subclinical venous sinus thrombosis in later life.

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