

# Subclinical Venous Sinus Thrombosis Secondary to Hypernatraemic Dehydration

C LOCKE, S DEPANI, A GOUR, M GRAY

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## Abstract

An 8-day-old infant was admitted to a paediatric intensive care unit with severe hypernatraemic dehydration. An MRI brain was performed which revealed extensive venous sinus thromboses. This is a recognised but rare complication of hypernatraemic dehydration and the importance of early detection and intervention of this condition is again highlighted. This is the first case report of venous sinus thrombosis secondary to hypernatraemic dehydration in a patient with no clinical neurological signs. We postulate that there may be a cohort of infants with subclinical cerebral venous thrombosis secondary to hypernatraemic dehydration.

## CASE REPORT

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

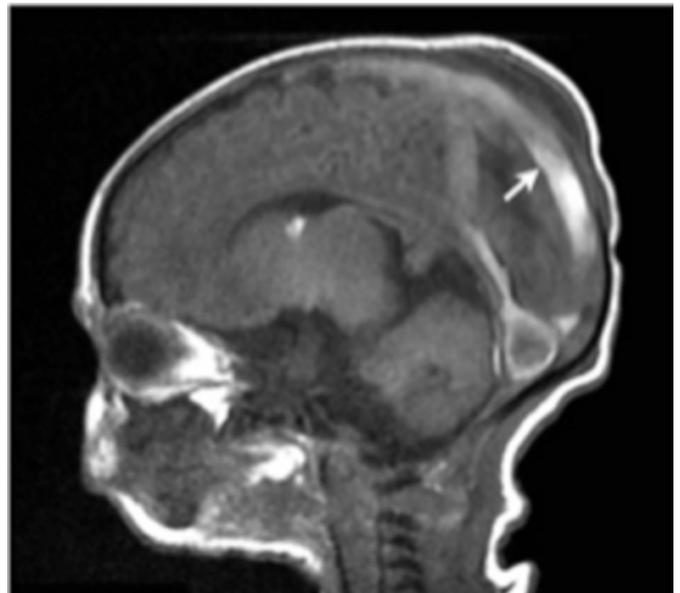
On examination the patient had signs consistent with severe dehydration. He was floppy and lethargic with a sunken fontanelle, decreased skin turgor, dry mucous membranes and capillary refill time of greater than 5seconds. He had lost 23% of his birth weight. He was breathing spontaneously but had periodic,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

After biochemical parameters had been slowly corrected, the patient was extubated uneventfully on day 3 of admission

and showed no signs of neurological sequelae. There were no signs of any 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.

## Figure 1

Figure 1. MRI demonstrating saggital (arrow) and long sinus thrombosis



The MRI showed extensive venous sinus thrombosis with involvement of superior sagittal, transverse and sigmoid sinuses (Figure 1). The patient remained clinically stable, with no neurological signs. A repeat MRI on day 7 showed

some improvement but persistent thrombosis. There were no signs of thrombosis in any other sites.

After discharge from PICU, the paediatric neurology team anticoagulated the patient, starting on subcutaneous heparin at an initial dose of 75IU/kg twice daily, which was subsequently increased to 150IU/kg. After the patient had established adequate breastfeeding and the parents had been educated in administration of subcutaneous heparin injections, the patient was discharged with regular follow up and outpatient monitoring of Factor Xa levels arranged. To date there have been no seizures or evidence of neurological fallout.

There were no other risk factors for this condition and full coagulopathy and thrombophilia screen have shown no abnormalities, therefore it is considered that this thrombotic event was directly attributable to the hypernatraemic dehydration.

### **DISCUSSION**

In a review of the literature there are several reported cases of venous sinus thrombosis associated with hypernatraemic dehydration. Karadag et al reported a case of sagittal sinus thrombosis as a complication of hypernatraemic dehydration in a 3 day-old girl.<sup>1</sup> Subsequently Fawke et al reported 3 cases of infants with cranial venous thrombosis secondary to hypernatraemic dehydration.<sup>2</sup> Prior to this we could find only one other report of cranial sinus thrombosis directly related to hypernatraemic dehydration.<sup>3</sup> Our patient differs from the reported cases in that it is the only case that 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 hypernatraemic dehydration.

Hypernatraemic dehydration as a complication of breastfeeding is showing an increasing incidence<sup>4</sup>. This case highlights a severe complication of hypernatraemic dehydration. Whilst “breastfeeding is best”, hypernatraemic dehydration as a result of inadequate breast feeding may go undetected until the infant becomes symptomatic. Inexperienced parents may be unaware of or misinterpret the

worrying signs of dehydration. Professional surveillance including the weighing of a newborn infant and appropriate intervention in the first few days of life is essential in preventing hypernatraemic dehydration. The importance of this has been previously highlighted<sup>2</sup>, 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 paediatrician or other knowledgeable health care professional within three to five days of age<sup>1,5</sup>

### **CONCLUSIONS**

This case highlights the need for high clinical suspicion of cerebral venous thrombosis in infants with severe hypernatraemic 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 hypernatraemic dehydration. Whether such a diagnosis has long term implications or even requires treatment remains to be established.

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

### **References**

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**Author Information**

**C LOCKE**

Department of Anaesthesia, St George's Hospital

**S DEPANI**

Department of Paediatrics, St George's Hospital

**A GOUR**

Paediatric Intensive Care, St George's Hospital

**M GRAY**

Paediatric Intensive Care, St George's Hospital