

Complicated Hypernatremic Dehydration in an Exclusively Breast-Fed Neonate: A Success Story of a Common Pathology from the Developing World

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Abstract

Hypernatremic dehydration in a neonate can occur due to inadequate feeding in developing world. We reported a case of a neonate with hypernatremic dehydration causing acute renal failure due to significant aortic and bilateral renal artery thrombosis. Basic supportive care and concurrent anticoagulation therapy using low molecular weight heparin for 6 weeks resulted in complete recovery in our patient. This case highlights the significance of timely intervention with anticoagulation therapy along with supportive care as an effective strategy in improving the survival and recovery of renal function despite prolonged ischemic renal injury.

Keywords: Hypernatremic Dehydration; Thrombosis; Neonate; Acute Renal Failure; Low Molecular Weight Heparin

Introduction

Hypernatremic dehydration (HD) in exclusively breast-fed neonates is common but is often underestimated due to fluid shifts [1]. During the past few years, there has been a rise in frequency of this diagnosis, most likely due to increased awareness about the condition [2]. HD is frequently found in neonates in primigravida mothers who lack experience in breastfeeding, leading to inadequate intake of free water by the baby [2,3]. The cause of HD in exclusively breast-fed infants is a free water deficit secondary to inadequate fluid. The diagnosis is usually made incidentally in the first neonatal visit where they present with > 10% weight loss in first 7 - 10 days of life after discharge. Some of these neonates present in emergency departments with signs of deep clinical jaundice with or without fever, due to dehydration. The severity of hypernatremia plays critical role in treatment and prognosis; therefore, it is also suggested that newborns must be followed up within 48 - 72 hours of discharge to assess the adequacy of breast-feeding and weight loss [3]. Literature reveals that clinical examination of these newborn babies suggests that they may present with high sodium and are usually active, very eager to suck, which might be misleading both for the mother and physician about the clinical severity of the condition [2]. Neurological complications of severe HD may be due to cerebral venous thrombosis, cerebral pontine myelinolysis due to quick correction of sodium, which may present with cerebral edema, seizures and eventually death [4,5]. To the best of our knowledge, this is the first clinical case report, of a neonate with severe HD, leading to both aortic bilateral renal artery and aortic thrombosis which was successfully managed with supportive care and antithrombotic treatment.

Case Summary

A 10-day old full-term male baby was admitted to an outside hospital with a 48-hours history of lethargy, poor feeding and low urine output. The baby was born to a 30-year-old healthy primigravida mother at 39-week gestation via emergency lower segment caesarian section due to ruptured uterine fibroid with a birth weight of 3700 gm. He remained well after birth and was discharged on day 3rd day of life at a weight of 3480 gm. On arrival to hospital, the mother was unable to describe the details of breastfeeding, and the numbers of wet diapers in a day. Reportedly the baby breastfed well every 2 - 3 hourly and appeared hungry even after feeds.

Examination revealed a cachectic, afebrile, irritable and severely dehydrated neonate based on findings of dry mucus membrane, decreased skin turgor, delayed capillary refill time (CRT) of 3 seconds and remarkable weight loss of almost 1000 gm compared to his birth weight. His vitals were unstable with borderline tachycardia with pulse of 166 beats/min and shallow breathing of 25 breaths per minute.

Initial laboratory studies revealed a serum sodium of 191 mEq/l, potassium 4.8 mEq/l, creatinine 5.1 mg/dl, blood urea nitrogen 50 mg/dl and blood glucose of 100 mg/dl. The hemoglobin was 14.4 g/dl, platelets 164,000, white blood cell count 23,100 with 67% polymorphonuclear leukocytes, 18% lymphocytes and 8% monocytes. The activated partial thromboplastin time (aPTT) was 31.5 seconds and the prothrombin time (PT) was 15 seconds. Both within normal limits.

As per the limited information, the baby was managed with intravenous antibiotics and intravenous fluids. Serial electrolytes and creatinine were monitored showing progressive improvement. After these clinical measures the baby's urine output increased to 3 ml/kg/h with progressive improvement in renal profile (Creatinine 1.1 mg/dl, BUN 25 mg/dl).

On 2nd day of admission, the baby developed issues of lethargy and desaturation requiring intubation and transfer to our tertiary care hospital for further evaluation and management.

On admission in our unit, baby appeared wasted with minimal fat tissue. He was pale, lethargic and bradypnea. Anterior fontanelle was open and flat. Pupils were bilaterally equal and reactive to light. Systolic blood pressure was 80 mmHg and pulse of 30/min. The kidneys were not palpably enlarged.

The repeated investigations, at emergency department after initial management from previous hospital revealed a serum sodium of 142 mEq/l, chloride 110 mEq/l, potassium 2 mEq/l, calcium 7.5 mg/dl, magnesium 1.4 mg/dl, BUN 12 mg/dl, creatinine 0.6 mg/dl. The hemoglobin was 10.2 g/dl, wbc 10 and platelets 70,000. Procalcitonin was unremarkable.

The baby was kept intubated in our neonatal intensive care unit (NICU); ventilator support was continued. The baby was evaluated for possible nosocomial infection. Triple tap was done along with Tracheal cultures for microbiology that subsequently turn out to be negative. Renal ultrasonography was normal. MRI Brain with contrast also reported to be normal. On the third day of admission, there were of persistent hypertension with blood pressures of 105/70 mmHg (> 95th centile), that was managed accordingly on hydralazine, amlodipine and spironolactone after having cardiology input. Echocardiography was done showed moderately reduced cardiac functions (EF 40%), so intravenous (IV) milrinone infusion was started. Added work up for persistent hypertension showed significantly raised serum renin and aldosterone, along with Doppler renal ultrasound findings of increased resistance and reversal of diastolic flow in both renal arteries, in the presence of normal renal parenchyma, which were suggestive of renal vascular pathology (Figure 1). Detailed CT Angiography revealed the filling defect, identified in aortic lumen at T10 level and T12-L1 level (abdominal aorta from supra renal to its bifurcation) and non-visualization of bilateral renal arteries suggestive of thrombosis (Figure 2). At this occasion a hematology opinion was taken and LMWH (enoxaparin) was started in therapeutic doses. The work up for inherited hypercoagulable state (Protein C and S antithrombin 3, homocysteine and factor V laden), was unremarkable. The baby clinically responded well on the given therapy, so IV milrinone was stopped, Orogastric tube (OG) feed was progressed to target, and a successful extubation trial was given after 14 days of intubation.

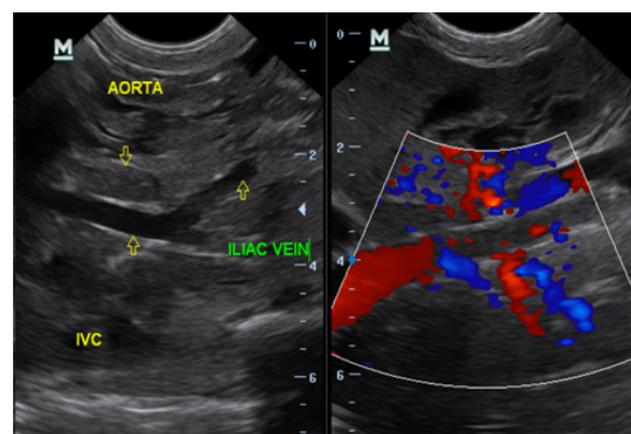


Figure 1: Grey scale and doppler ultrasound examination showing echogenic thrombus in abdominal aorta. No color flow is demonstrated in abdominal aorta on Doppler examination.

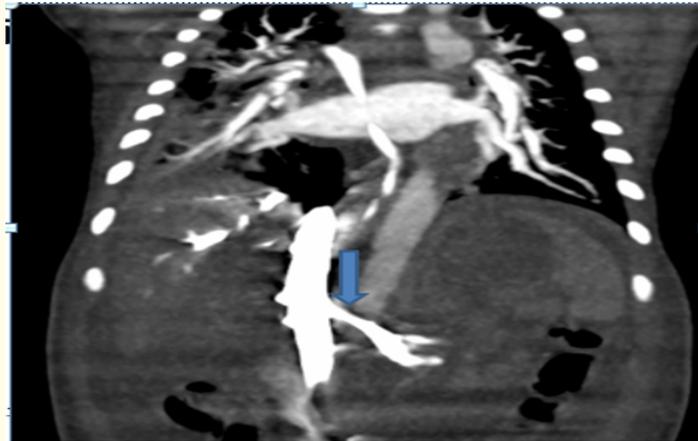


Figure 2: CT-scan coronal section with contrast showing non-visualisation of bilateral renal arteries suggestive of thrombosis. Contrast reflux from IVC into the left renal vein is noted (arrow).

The baby was discharged home on OG feed along with amlodipine, enoxaparin and micronutrient supplements.

Enoxaparin was continued for total of 6 weeks keeping anti-Xa levels therapeutic between 0.5 - 1 IU/ml. The thrombus was followed with repeated Doppler US scan. There was absence of any residual filling defect suggestive of thrombus resolution. Hypertension gradually improved so antihypertensive were restricted to low doses of amlodipine only which was subsequently stopped on follow up.

Discussion

Hypernatremia is defined as a serum sodium level greater than 145 mmol/L [mEq/L]. In neonates, predominant water deficit in relation to total body sodium, is an established cause of hypernatremia [6]. HD in a newborn, is usually suspected when weight loss is more than 10% of the birth weight at the end of first week of life or if there is clinical findings of dehydration with hypernatremia, like refusal to feed, lethargy, decreased urine output, fever or jaundice [7-9]. Our patient presented with the same signs and symptoms. In recent days, the incidence of HD has increased significantly [2]. This is probably because of decrease awareness and underestimation regarding the condition, as a result of which many such cases are either missed, or wrongly diagnosed as sepsis due to overlapping clinical feature [10]. This case report of HD present unique complications of both renal artery and aortic thrombosis leading to Acute renal failure.

HD is potentially a lethal condition in neonate which adversely affects central nervous system, leading to devastating consequences like intracranial hemorrhage, thrombosis, neurological deficit with seizures and even death [11]. Commonly reported complications of HD include Acute kidney injury (AKI), hypoglycemia, seizures, jaundice and Disseminated intravascular coagulation (DIC). Our baby also had AKI and jaundice along with a relatively uncommon complication of aortic and bilateral renal artery thrombosis.

Thrombotic disease is uncommon in newborns; however, it is increasingly recognized as a complication of modern neonatal care in NICU settings. Thrombosis contributes to significant morbidity and mortality especially in infants, less than 1 year of age [13]. Incidence of thrombotic events varies significantly in neonate which may be due to various methodological differences, including study designs, definitions, and criteria for neonatal thrombosis. Recently two neonatal registries from Canada and Germany have shown their incidence of clinically apparent thrombosis as 2.4/1000 and 5.1/100000 respectively [14,15]. Both registries found that the use of intravascular catheters especially in NICU, is the most common risk factor for development of thrombosis. Other risk factors were identified, including

asphyxia, septicemia, dehydration and maternal diabetes. In our case, clinical and laboratory proven dehydration was the cause of thrombosis and it was secondary to lactation failure, poor fluid intake and a delay in reaching for medical help.

The treatment approaches vary depending on the location and extent of the thrombus which must be balanced against with the risks and benefits ratio. As per the ACCP guidelines [16] we manage asymptomatic neonatal thrombosis by close monitoring of thrombus progression and by providing supporting care, while severe symptomatic thromboembolic events are typically treated with anticoagulants and/or fibrinolytic agents under supervision of qualified hematologist. In our baby, we used LMWH (Enoxaparin) for total of 42 days to achieve recanalization of aorta and bilateral renal arteries.

In lower middle-income countries (LMIC) awareness of HD in neonate during first 7 to 10 days of life is lacking. The review is written to orient our health professionals particularly pediatricians and neonatologists regarding diagnosis and timely management of HD so that devastating consequences in terms of morbidity and mortality in neonate can be avoided.

Conclusion

Breastfeeding is superior than any other form of feeding and have advantages both for mother and the baby. A rise in the incidence of breast feeding associated HD is due to lack of proper education and training of primigravida mothers particularly in the LMIC. Thus, stressing the importance of early follow up of breast fed neonates with heightened awareness amongst health care providers for this diagnosis.

Conflict of Interest

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

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