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Subcutaneous fat necrosis of the newborn and nephrolithiasis

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Abstract

Objectives: Subcutaneous fat necrosis of the newborn (SCFN) is a rare panniculitis that can affect newborn patients who have experienced perinatal stress, hypoxia, or hypothermia. Risk factors include gestational diabetes and pre-eclampsia. This condition is usually self-limiting; however, it can lead to severe complications, including hypercalcemia. After diagnosis, it is important to monitor calcium levels. There is no current standard for how frequently these levels should be monitored.

Case presentation: We present a full-term African American male with multiple risk factors, who was diagnosed with SCFN. The patient's hypercalcemia secondary to SCFN led to renal complications.

Conclusions: This case highlights the importance of early and frequent monitoring of calcium levels in patients with SCFN.

Keywords: dermatology; nephrolithiasis; panniculitis.

Introduction

Subcutaneous fat necrosis of the newborn (SCFN) is a rare panniculitis that occurs within the first few weeks of life, often in full-term neonates who experience perinatal stress, hypoxia, or hypothermia [1]. This condition presents as erythematous nodules or plaques on the cheeks, back, buttocks, or proximal extremities [2]. SCFN is self-limiting; however, patients can develop complications including hypercalcemia and thrombocytopenia [1]. Although the etiology of this condition is unclear, it is thought to be due to perinatal stress interfering with the blood supply of the fat

tissue, which leads to inflammation and necrosis [2]. Granulomatous infiltrates within these lesions increase the expression of 1-alpha-hydroxylase, which activates vitamin D3 and leads to the increased release of calcium [3]. When calcium levels exceed the kidneys' excretion capacity, renal complications such as nephrolithiasis can occur [4]. Maternal risk factors include gestational diabetes, pre-eclampsia, cocaine and cigarette exposure, calcium blocker use, and familial dyslipidemia disorders [2].

SCFN can be diagnosed clinically or via biopsy, with radially arranged crystals in fat cells and lymphocytes on histopathology [3]. Treatments for cutaneous symptoms consist of supportive care and pain management as the lesions resolve within weeks [5]. Calcium monitoring is recommended for six months due to potential complications of hypercalcemia including renal failure, cardiac arrest, and metastatic calcifications [2, 6]. Recommended managements for hypercalcemia consist of calcium-wasting loop diuretics, steroids, bisphosphonates, and calcitonin depending on the severity of lab abnormalities [5]. Despite the potential complications of hypercalcemia, there are no current standards for the frequency of calcium monitoring. This case presents an infant with hypercalcemia and renal complications secondary to SCFN, which highlights the importance of early and frequent monitoring of calcium levels in patients with this diagnosis.

Case presentation

A 2.905 kg African American male was delivered by a 42-year-old G2P1 female at 37 weeks with a history of chronic hypertension with superimposed severe pre-eclampsia, gestational diabetes, tobacco use during pregnancy, and a BMI greater than 40 kg/m². The patient was delivered via primary cesarian section due to non-reassuring fetal heart tones and lack of cervical dilation. The patient was initially hypotonic and apneic with APGAR scores of 2, 6, and 9 at 1, 5, and 10 min, respectively. Resuscitation efforts included deep suction, positive pressure ventilation for 2 min, and continuous positive airway pressure for 5 min, before transitioning to room air. He was transferred to the ICU for treatment of hypoglycemia despite feedings, hypothermia, respiratory distress,

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and concern for sepsis. His lowest serum glucose measurement was <5 mg/dL at 2 h of life, which was treated with two boluses of 6 mL of Dextrose 10% in water. The patient's hypoglycemia management required a peripherally inserted central catheter with a maximum glucose infusion rate of 11.3 mg/kg/min for 12 h on his second day of life, his blood glucose stabilized six days later. On day 4, dermatology was consulted for new tender, flat, violaceous, plaque-like lesions on the bilateral cheeks, shoulders, and buttocks (Figure 1). The facial and shoulder lesions were firm and well demarcated (Figure 2). They determined that the diagnosis was likely SCFN and recommended supportive care with serial calcium monitoring. His serum calcium was within normal limits on day 4 and 5, but he had thrombocytopenia with a platelet count of $23 \times 10^9/L$, which was attributed to SCFN.

On day 12, dermatology was consulted again to examine a scaly eruption of unknown etiology over the affected areas which was managed with hydrocortisone 2.5% cream and hydrophilic topicals (Figure 3). A punch biopsy was obtained, which confirmed subcutaneous fat necrosis with crystals and focal lymphohistiocytic inflammation (Figure 4). Dermatology again recommended daily calcium monitoring with pediatric endocrinology follow up. His serum calcium was next checked on day 14 and 17, both levels were elevated at 11.9 and 13.2 mg/dL, respectively (normal 8.8–11.3 mg/dL) [7]. His ionized calcium was also

Figure 1: A tender, flat, violaceous, plaque-like lesion on the left cheek that appeared on day 4.

Figure 2: A firm, well demarcated plaque-like lesion on the right shoulder.

elevated to 1.64 mmol/L at this time, but serum magnesium, phosphorous, alkaline phosphatase, and albumin were within normal limits. Throughout this time, the patient did have symptoms of hypercalcemia, as he was exhibiting feeding intolerance due to signs of gastrointestinal reflux and immature oromotor skills requiring partial gavage. On day 17, pediatric endocrinology was consulted for hypercalcemia; he was treated with furosemide and switched to a low calcium formula. His platelet count normalized on day 18. His serum calcium levels were monitored every 12 h and peaked at 13.9 mg/dL on day 20. A 2 mg/kg per day dose of oral prednisolone was added to his treatment regimen for two months, then decreased to 0.4 mg/kg for two weeks before discontinuing.

Figure 3: A scaly eruption on the previously affected areas of the left cheek.

Figure 4: Histology of the left hip revealed subcutaneous fat necrosis with crystals and focal lymphohistiocytic inflammation.

On day 22, nephrology was consulted, and the patient received a renal ultrasound which showed 3 mm non-obstructive renal calculi without nephrocalcinosis, likely secondary to SCFN related hypercalcemia. The patient was also hyponatremic and hypokalemic due to the low calcium formula. They recommended a three-month ultrasound follow-up, as the calculi would likely pass on their own. By day 26, his serum calcium levels had decreased to 10.6 mg/dL and he was able to discontinue furosemide on the next day.

Conclusions

The patient had multiple risk factors for SCFN including perinatal stress, hypoxia, hypothermia, maternal diabetes, pre-eclampsia, and cigarette use during pregnancy, and was diagnosed on initial presentation. However, he was not closely monitored for hypercalcemia and was 0.1 mg/dL below the threshold for a hypercalcemic crisis at his peak [6]. Although his serum calcium was within normal limits at the time of his diagnosis, it was likely trending upward for several days until it was checked again nine days later. A systematic review showed that more than half of infants who are diagnosed with SCFN develop hypercalcemia [6]. In 95% of these cases, hypercalcemia developed within 60 days after the onset of the skin lesions [6]. This indicates the importance of monitoring calcium, even in asymptomatic SCFN patients, given the high rate of

elevated levels. Several studies have recommended six months of calcium screening after diagnosis of SCFN, however there is no standard of care for the frequency of these screenings in an inpatient setting.

In this case, late identification of increased calcium levels may have led to renal complications. Although the resulting calculi were small and did not require extensive treatment, if they progressed to 5 mm, they would require surgical intervention [8]. Untreated hypercalcemia can also lead to seizures, electrolyte imbalances, arrhythmias, hypotonia, and failure to thrive [4]. This case indicates that daily screening and early identification of hypercalcemia after a diagnosis of SCFN can lead to earlier treatment and prevention of sequelae.

This case highlights the importance of early identification and management of SCFN, which can help prevent potentially severe complications, including hypercalcemia. Due to the high rates of hypercalcemia associated with this condition, it is important to closely monitor patient's calcium levels after their initial diagnosis. Parents of patients with many risk factors for SCFN should also be educated on signs of these lesions after discharge to assist with early identification and monitoring.

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