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Case report

Subcutaneous fat necrosis of the newborn complicated by severe hypercalcemia and hypertriglyceridemia

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Abstract

Subcutaneous fat necrosis of the newborn (SCFN) is a rare acute hypodermatitis characterized by firm, indurated plaques and subcutaneous nodules with purplish-red color, typically occuring between the 1st and 6th weeks of life. While generally benign and self-resolving, this condition can occasionally lead to severe complications.

We report the case of a female newborn with a maternal history of hypothyroidism and severe preeclampsia, and a personal history of perinatal asphyxia and sepsis, admitted on the 20th day of life for fever and dehydration. Dermatological examination revealed indurated purplish-red nodules on the back and arms. Laboratory tests showed severe hypercalcemia and hypertriglyceridemia, while renal ultrasound revealed nephrocalcinosis. A diagnosis of SCFN, complicated by severe hypercalcemia, hypertriglyceridemia and nephrocalcinosis, was made. The patient was treated with intravenous fluids, diuretics, and corticosteroids, resulting in clinical and biological improvement.

Although SCFN often resolves spontaneously, severe complications like hypercalcemia may require intervention. Early diagnosis and

management, particularly in neonates with multiple risk factors, are crucial to prevent complications. Further research is needed to optimize treatment strategies and improve outcomes for affected infants.

Keywords

Neonate, hypercalcemia, subcutaneous fat necrosis of the newborn, hypertriglyceridemia.

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Introduction

Subcutaneous fat necrosis of the newborn (SCFN) is a rare condition characterized by firm, red or purple subcutaneous nodules, most commonly located on the trunk, buttocks, cheeks, and extremities [1]. This acute form of hypodermitis typically occurs in the neonatal period, often within the first month of life [2]. While the exact cause remains unclear, neonatal stress and other predisposing factors have been identified.

SCFN is generally considered a benign condition in which lesions resolve spontaneously within a few weeks to 6 months [3]. However, complications can occur, including local tissue breakdown, hypoglycemia, anemia, thrombocytopenia, and less commonly hypertriglyceridemia. Hypercalcemia occurs in almost 50% of reported SCFN cases, although it is rarely severe [4]. Symptoms typically occur within the first weeks of life and may include polyuria, gastrointestinal discomfort, dehydration, and weight loss [5]. Both pediatricians and dermatologists should be aware of this condition and should suspect it when appropriate, given the possibility of severe complications.

In this report, we describe the case of a 3-week-old female with severe neonatal hypercalcemia, hypertriglyceridemia, and nephrocalcinosis as complications of SCFN.

Case presentation

We report the case of a 20-day-old female newborn, admitted for dehydration and fever. She was the firstborn child of a 39-year-old mother with hypothyroidism and no family history of dyslipidemia. The patient was born at 38 weeks of gestation by cesarean section due to severe preeclampsia, with an Apgar score of 6 in the 1st minute and 7 in the 10th minute. She had a birth weight of 3.9 kg and was admitted within the first hour of life for management of grade 1 perinatal asphyxia and neonatal respiratory distress secondary to transient tachypnea of the newborn.

On the 8th day of her hospital stay, the patient contracted a healthcare-associated infection with extended-spectrum beta-lactamase (ESBL)-producing *Klebsiella pneumoniae*. She was treated with intravenous antibiotics for 10 days, resulting in good clinical and biological recovery.

On examination, she was slightly dehydrated with a weight loss of 300 grams, but otherwise vitally stable. She was afebrile, and had no neurological symptoms. Dermatological examination revealed multiple purple-red nodules on her back and both arms (**Fig. 1**). These lesions were warm and tender upon palpation.

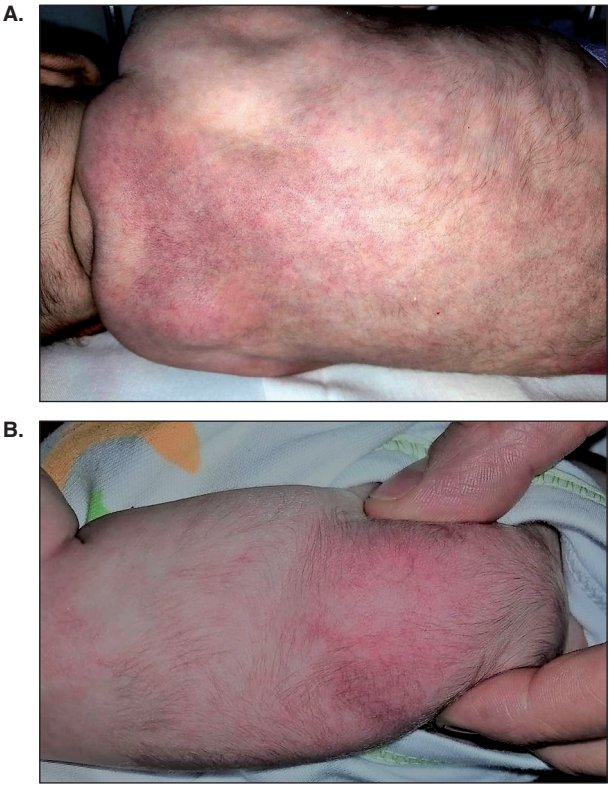

Figure 1. A. Subcutaneous fat necrosis on the back. **B.** Subcutaneous fat necrosis on the arm.

Based on her medical history and clinical findings, a diagnosis of SCFN was made. Blood tests revealed a calcium level of 3.35 mmol/L (normal range: 2.2-2.7 mmol/L), a triglyceride level of 2.15 mmol/L (normal range: 0.35-1.45 mmol/L) and a normal platelet count (**Tab. 1**). Renal ultrasound showed early signs of nephrocalcinosis.

Initial management included intravenous rehydration and diuretics (furosemide). However, due to persistent hypercalcemia, oral prednisolone therapy was started at a dose of 2 mg/kg daily. This resulted in normalization of calcium levels within 3 days (**Fig. 2**). Follow-up monitoring was conducted weekly at first, and then monthly, during which calcium levels remained within the normal range.

The skin lesions resolved after about 2 months of treatment. During this period, the prednisolone dose was gradually reduced as calcium levels normalized and lesions disappeared. Three weeks after resolution of the lesions, follow-up renal ultrasound revealed no evidence of nephrocalcinosis. No other sites of calcium deposition were identified in this case.

Table 1. Blood tests results.

Test	Value	Reference range
Calcium (mmol/L)	3.35	2.2-2.7
Magnesium (mmol/L)	0.74	0.65-1.1
Phosphate (mmol/L)	2.07	1.35-2.3
Creatinine (umol/L)	33	27-77
Urea (mmol/L)	2.7	1-4.2
Alkaline phosphatase (U/L)	140	100-400
Vitamin D (ng/mL)	28	30-80
TSH (mUI/L)	3.88	0.16-16
Sodium (mmol/L)	138	133-146
Potassium (mmol/L)	4.81	3.7-5.7
Chloride (mmol/L)	101.2	95-110
Serum triglycerides (mmol/L)	2.15	0.35-1.45
Serum cholesterol (mmol/L)	4.53	1-4.8
C-reactive protein (mg/L)	17.3	< 5
Hemoglobin (g/dL)	11.9	14-23
White cell count (K/uL)	13.390	10-25
Platelet count (K/uL)	434.000	150-450

TSH: thyroid-stimulating hormone.

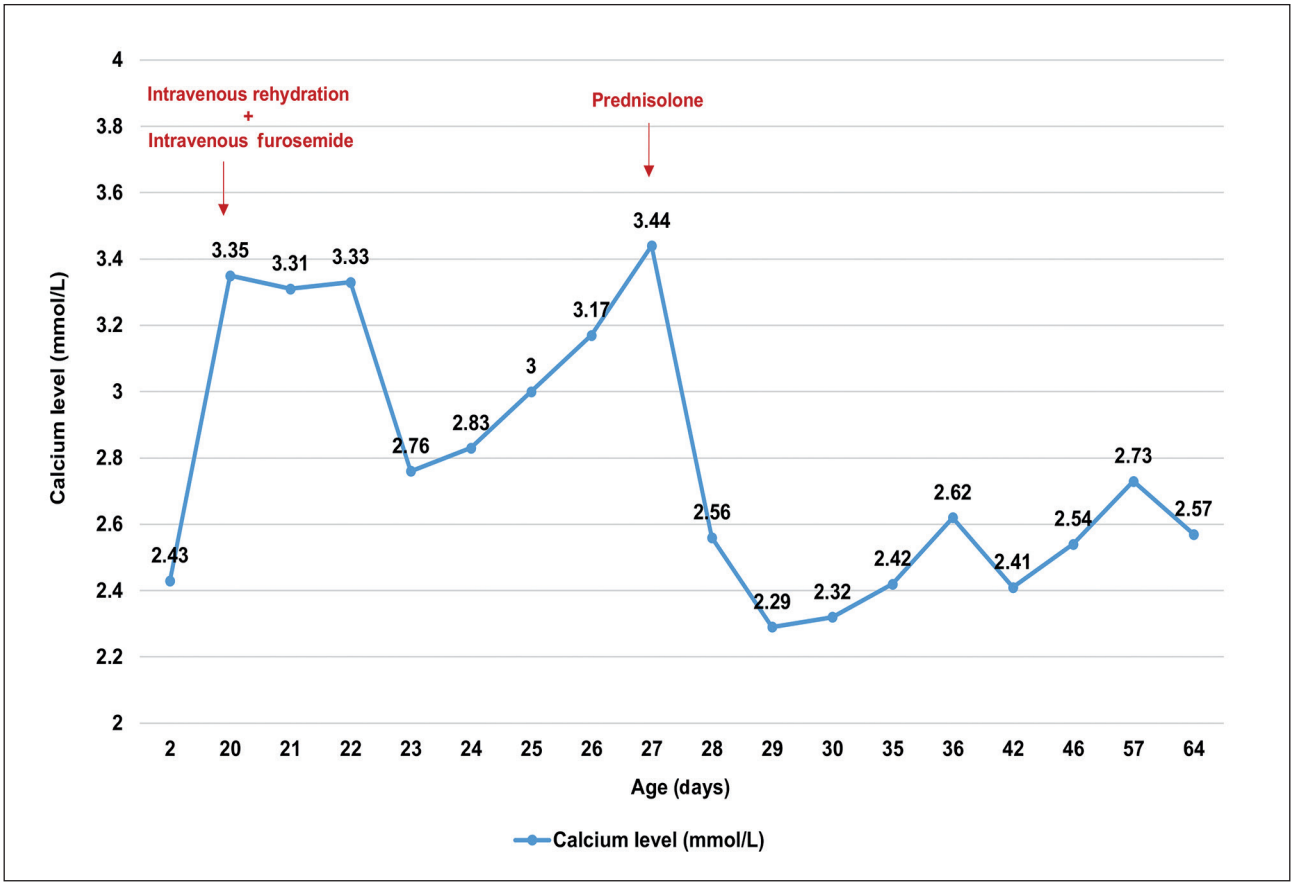

Figure 2. Total serum calcium levels during follow-up.

Discussion

SCFN is a rare form of panniculitis typically observed in term newborns [1]. Although the exact etiology is unclear, perinatal stress leading to hypoxia and hypothermia can trigger inflammation and necrosis in immature adipose tissue. Multiple risk factors have been associated with SCFN, including perinatal asphyxia, localized skin trauma, obstetric complications, macrosomia, preeclampsia, meconium aspiration, sepsis, maternal gestational diabetes, maternal hypothyroidism and use of drugs like cocaine or calcium channel blockers, as well as familial dyslipidemia [1, 6, 7]. Our patient had, in fact, four identified risk factors: perinatal asphyxia, preeclampsia, maternal hypothyroidism, and sepsis attributable to a healthcare-associated infection with ESBL-producing *Klebsiella pneumoniae*.

Clinically, SCFN typically presents with firm, erythematous or violaceous indurated plaques which evolve into painful subcutaneous hard nodules, often located on the back, shoulders, upper limbs and thighs [8]. Our patient had lesions on the back and both arms, which are commonly affected sites, which is consistent with reported cases [6]. While the skin lesions are often characteristic, differential diagnoses such as cellulitis, erysipelas, sclerema neonatorum, and cold panniculitis must be considered [9].

Although SCFN is generally benign, resolving spontaneously within a few months, it can lead to severe complications that require close monitoring. The most common complications include thrombocytopenia, hypoglycemia, hypertriglyceridemia, and, most notably, hypercalcemia [4]. Hypercalcemia is observed in 25% to 65% of SCFN cases, usually within the first month of life [4, 6, 8]. It is defined as a serum calcium concentration that is 2 standard deviations above the normal mean, typically > 2.60 mmol/L, or an ionized calcium level > 1.32 mmol/L (normal range: 1.16-1.32 mmol/L). Measuring ionized calcium is particularly important to distinguish true hypercalcemia from elevated total calcium due to increased protein binding [10]. Therefore, when SCFN is suspected, ionized calcium levels should be measured, with ongoing monitoring during the first 3 months of life, even if hypercalcemia is not initially detected [11].

Several mechanisms have been proposed to explain hypercalcemia in SCFN. One theory suggests elevated prostaglandin E levels, which stimulate osteoclastic bone resorption, thus increasing serum calcium levels. Another hypothesis involves the release of calcium from necrotic adipocytes, although the delayed onset of hypercalcemia relative to adipocyte necrosis makes this less likely. The most widely accepted explanation is the extrarenal synthesis of 1,25(OH)2-vitamin D3 by lipophagic granulomas, which increases intestinal calcium absorption [4, 12]. In most cases, hypercalcemia is moderate (< 3 mmol/L) and asymptomatic, though rare cases of severe hypercalcemia (> 3 mmol/L) have been reported [7].

Calcium deposits can occur in the kidneys, myocardium, liver, vessels and gastric mucosa [7]. In our case, nephrocalcinosis was detected on renal ultrasound, but no clinical signs suggested calcium deposition in other organs, so further systematic evaluation was not deemed necessary [12, 13].

Hypertriglyceridemia is a rarely reported complication of SCFN and typically resolves spontaneously. However, its presence must lead to seeking family history of dyslipidemia [6, 7]. Our patient had no family history of dyslipidemia, and yet she developed hypertriglyceridemia, which resolved spontaneously. No further SCFN complications were observed in our patient.

The initial approach to treating hypercalcemia involves prompt hydration to manage dehydration and prevent acute kidney injury. Diuretics, such as furosemide, are administered to lower serum calcium levels, along with reducing calcium and vitamin D intake [14]. Glucocorticoids, such as prednisolone, are recommended as second-line therapy for hypercalcemia in SCFN, as they inhibit the activation of 25-OH-vitamin D3, thereby preventing calcium accumulation [13]. Pamidronate, a bisphosphonate, has been reported to be effective in cases where conventional treatments fail, and it may be considered as first-line therapy for severe hypercalcemia to prevent nephrocalcinosis and avoid the use of glucocorticoids. Pamidronate has been shown to be effective and well tolerated at a dose of 0.25-0.5 mg/kg [4, 15]. In our case, pamidronate was not necessary, as hypercalcemia was quickly controlled with glucocorticoids.

Conclusion

SCFN presents a unique clinical challenge due to the risk of complications. Although the condition often resolves spontaneously, severe cases may occur, requiring timely intervention.

Management strategies should prioritize early detection and recognition in neonates with multiple risk factors to prevent and control complications such as hypercalcemia. Further research is crucial to optimize treatment protocols and improve outcomes for infants affected by this rare but potentially serious condition.

Informed written consent

Informed written consent was obtained from the patient's family for publication of this case report and any accompanying images.

Declaration of interest

The Authors declare that there is no conflict of interest.

References

1. Mitra S, Dove J, Somisetty SK. Subcutaneous fat necrosis in newborn – an unusual case and review of literature. Eur J Pediatr. 2011;170(9):1107-10.
2. Messaoudi S, Seddiki AE, Chaalal M, Amrani R. [Fat necrosis of the newborn: report of two cases]. [Article in French]. Pan Afr Med J. 2015;22:34.
3. Sultana J, Yasmeen S, Nazme NI, Moslem MHM, Haque ZS, Fatema NN. Subcutaneous Fat Necrosis of the Newborn: A Case Report. J Bangladesh Coll Physicians Surg. 2017;35(4): 192-5.
4. Martinelli S, Pitea M, Gatelli IF, Raouf T, Barera G, Vitelli O. Safety and Efficacy of Pamidronate in Neonatal Hypercalcemia Caused by Subcutaneous Fat Necrosis: A Case Report. Front Pediatr. 2022;10:845424.
5. Thomas JM, Bhandari J, Rytina E, Gass JK, Williams RM, Burrows NP. Subcutaneous Fat Necrosis of the Neonate with a Delayed Second Eruption. Pediatr Dermatol. 2016;33(2): e134-6.
6. Del Pozzo-Magaña BR, Ho N. Subcutaneous Fat Necrosis of the Newborn: A 20-Year Retrospective Study. Pediatr Dermatol. 2016;33(6):e353-5.
7. Mahé E, Girszyn N, Hadj-Rabia S, Bodemer C, Hamel-Teillac D, De Prost Y. Subcutaneous fat necrosis of the newborn: a systematic evaluation of risk factors, clinical manifestations, complications and outcome of 16 children. Br J Dermatol. 2007;156(4):709-15.
8. Lorenzo C, Romana A, Matias J, Calhau P. Subcutaneous fat necrosis of the newborn – An atypical case with typical complications. Clin Case Rep. 2021;9(4):2069-73.
9. Regaieg C, Thabet AB, Kolsi N, Sellami K, Turki H, Charfi M, Hmida N. Congenital liquified subcutaneous fat necrosis in a newborn: an unusual case. Dermatol Online J. 2024;30(1):D330163295.
10. Stokes VJ, Nielsen MF, Hannan FM, Thakker RV. Hypercalcemic Disorders in Children. J Bone Miner Res. 2017;32(11):2157-70.
11. Siegel LH, Fraile Alonso C, Tuazon CFR, Mancini AJ, Kruse LL, Miller JL, Wagner AM, Yun D, Kenner-Bell BM, Paller AS, Chamlin SL. Subcutaneous fat necrosis of the newborn: A retrospective study of 32 infants and care algorithm. Pediatr Dermatol. 2023;40(3):413-21.
12. Dudink J, Walther FJ, Beekman RP. Subcutaneous fat necrosis of the newborn: hypercalcaemia with hepatic and atrial myocardial calcification. Arch Dis Child Fetal Neonatal Ed. 2003;88(4):F343-5.
13. Tran JT, Sheth AP. Complications of subcutaneous fat necrosis of the newborn: a case report and review of the literature. Pediatr Dermatol. 2003;20(3):257-61.
14. Rice AM, Rivkees SA. Etidronate therapy for hypercalcemia in subcutaneous fat necrosis of the newborn. J Pediatr. 1999;134(3):349-51.
15. Alos N, Eugène D, Fillion M, Powell J, Kokta V, Chabot G. Pamidronate: Treatment for severe hypercalcemia in neonatal subcutaneous fat necrosis. Horm Res. 2006;65(6):289-94.