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Case Report

Subcutaneous Fat Necrosis of the Newborn Presenting as an Axillary Mass Following Therapeutic Hypothermia

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Abstract

Subcutaneous fat necrosis (SCFN) of the newborn is a self-limited form of panniculitis associated with peripheral perfusion disturbances which in consequence lead to crystallization of free fatty acids and subcutaneous fat. The skin lesions are mainly localized on the back, limbs and buttocks. We present a SCFN, with a rare localization confirmed by histologic investigation in an infant with a history of perinatal asphyxia and therapeutic hypothermia.

Keywords: *Axillary Mass; Newborn; Subcutaneous Fat Necrosis; Therapeutic Hypothermia*

Introduction

Subcutaneous fat necrosis (SCFN) of the newborn is a self-limited form of panniculitis associated with peripheral perfusion disturbances which in consequence lead to crystallization of free fatty acids and subcutaneous fat. Lesions are mainly localized on the back, limbs and buttocks [2]. We present a SCFN, with a rare localization. Although self-limited, it is critical to recognize SCFN, yet affected cases require monitoring for associated life threatening complications [1,2].

Case Report

A 20-day-old, full-term infant boy presented to neonatal follow up clinic with a 1-day history of axillary swelling. Physical examination revealed a hard, fixed, 3-cm mass palpable in the right axilla with reddish discoloration of the overlying skin. Infant had skin temperature of 36.7°C, heart rate of 120 beats per minute, respiratory rate of 40 breaths per minute, and blood pressure of 80/45 mmHg. Oxygen saturation was 99% on pulse oximetry. On radiography, subcutaneous calcification in the right axillary area was noticed (Figure 1).

Previous medical records revealed therapeutic hypothermia history due to perinatal asphyxia after placental ablation. He was born with an Apgar score of 1/1/2. Umbilical cord pH was 6.7 (BE - 18 mEq/l) after successful neonatal resuscitation in the delivery room, infant was transported to neonatal intensive care unit and whole-body cooling was initiated at the first hour, and continued for the next 72 hours of life. He was intubated and mechanically ventilated for the first day of his life and then he was successfully extubated straight to room air. He did not develop any other complications including seizures or any findings of organ failure. Minimal enteral feeding was started on the second day of life and gradually increased to full enteral feedings. His cranial magnetic resonance imaging revealed a mild increase in signal intensity in the white matter. He was discharged from neonatal intensive care unit on 10th day of life and his first control in neonatal follow up clinic on the 12th days of life revealed normal physical findings with normal blood biochemistry including calcium.

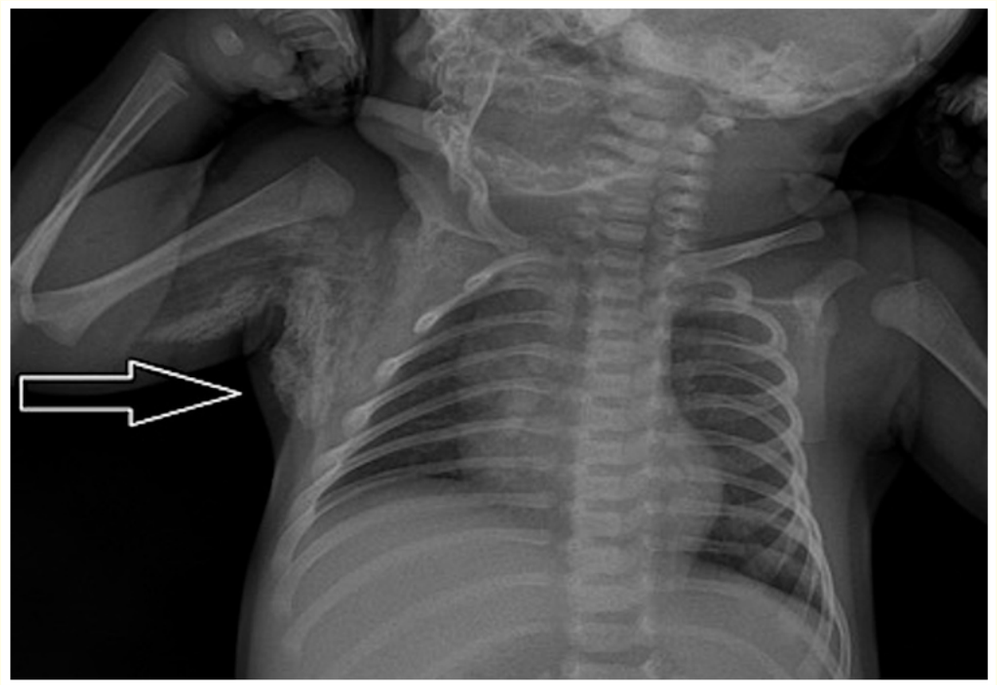

Figure 1: *Subcutaneous calcification on radiography.*

After hospitalization, biopsy was performed in order to illuminate the pathology, along with empirical intravenous ampicillin and gentamicin therapy. Blood and local cultures were negative. Subsequent serum calcium and triglyceride levels, as well as complete blood count and C-reactive protein remained within normal limits. Axillary mass resolved gradually within four weeks.

Results

Histologic examination revealed subcutaneous fat necrosis with calcification, clusters of adipocytes, histiocytes, fibroblasts, and foreign body giant cells (Figure 2).

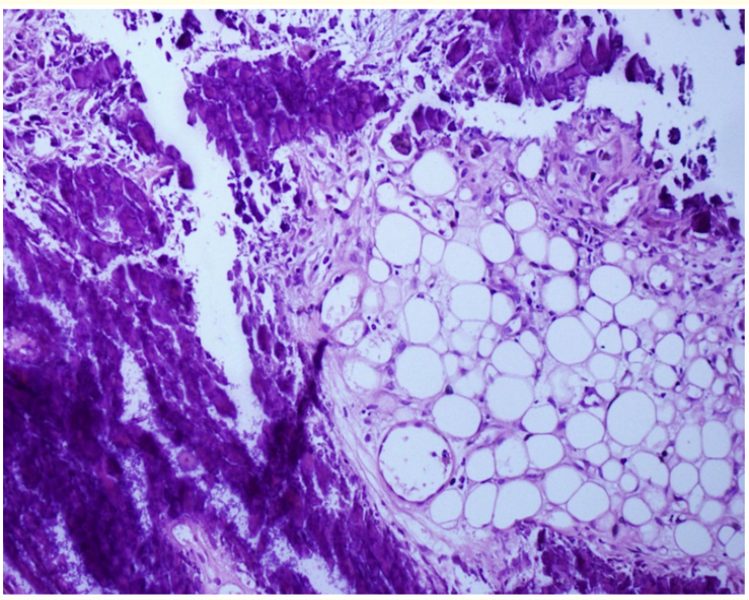

Figure 2: *Calcium deposits next to the fat necrosis (Hematoxylin Eosin X 200).*

Discussion

We present a SCFN case, with a rare localization confirmed by histologic investigation in an infant with a history of perinatal asphyxia and therapeutic hypothermia. It has been suggested that therapeutic hypothermia for management of hypoxic ischemic encephalopathy of newborn is an actual risk factor for subcutaneous fat necrosis [3,4].

Although the pathogenesis of SCFN remains obscure, there are several attractive hypothesis trying to illuminate the mechanism. One hypothesis put the blame on the local tissue hypoxia and mechanical pressure; another underlines the predisposition of neonatal adipose tissue to crystalize at low temperatures due to abundance of stearic acids and saturated palmitic acids rather than oleic acid in brown fat of infancy [1-3]. Hypothermia may worsen skin perfusion that is already compromised by perinatal asphyxia, which ultimately end up with fat necrosis [4]. Pressure stress, may create an additional local ischemia that compounds the tissue injury [1-3].

Axillary area is a rare localization for SCFN, however, in our case, hypothermia blanket was wrapped to the body of the infant below the armpits which resulted in exposure of cold to the axillary and pectoral region. Local pressure by hypothermia blanket may well be a contributing factor to the lesion.

SCFN can cause soft-tissue calcification and recognition of this entity can be possible based on features revealed by radiology which is also important in diagnosing accompanying visceral fat necrosis [5,6].

Hypercalcemia associated with increased calcium absorption due to unregulated production of vitamin D, may accompany some cases of SCFN [4]. Blood biochemistry should be monitored closely in these infants. In our case, blood calcium levels found to be normal during hospital stay and follow up period.

Conclusion

Although rare, SCFN should be kept in mind in differential diagnosis of axillary lumps, especially infants with history of therapeutic hypothermia and risk groups should be checked for the presentation of skin findings even after hospital discharge.

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Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Defne Engür has written the article. Defne Engur, Mehmet Yekta Oncel, Elif Yigit, Meltem Koyuncu-Arslan, Sumer Sutcuoglu have managed the case clinically in neonatal intensive care unit. Gülden Diniz performed the pathological examination.

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