Original Article

Subcutaneous fat necrosis of the newborn, report of five cases

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Abstract

Subcutaneous fat necrosis of the newborn (SCFN) is a rare, self-healing condition affecting full-term or post-term newborns within the first weeks of life. Although the etiology is unknown, this disorder is associated with neonatal hypoxia, hypothermia, obsteric trauma, anemia, thrombocytopenia, gestational diabetes, pre-eclampsia and maternal exposure to use of cocaine or calcium channel blockers during pregnancy. Hypercalcemia is the most serious although rare complication and may occur up to six months after the skin lesions appear. We report five patients with diagnosis of subcutaneous fat necrosis of the newborn detected during 2001-2008. All patients had history of perinatal hypoxia, four of the pregnancies presented with hypertension and no child had complications (Dermatol Argent 2009;15(3):200-204).

Key words: *subcutaneous fat necrosis, hypercalcemia, newborn.*

ABREVIATURAS

SCFN : subcutaneous fat necrosis of the newborn

Introduction

Subcutaneous fat necrosis of the newborn (SCFN) is a rare disorder affecting full-term or post-term newborns in the first weeks of life.[1-4] Clinical features include nodules or indurated erythematous plaques. The most frequent location is the trunk, cheeks and lower limbs. The evolution is favorable, generally self-healing in the first months of life.

Although the etiology is not completely clarified yet, it has been associated with various risk factors:

- Neonatal factors: neonatal hypoxia,[4] hypothermia,[2]local trauma,[3,5] anemia[3] and thrombocytopenia.[4]
- Maternal factors: gestational diabetes, pre-eclampsia during pregnancy. Recently, smoking and familiar history of thrombosis were described as risk factors.[4]

Normal adipose tissue contains mainly triglycerides, especially palmitic, stearic, and oleic acids, in a variable ratio, depending on whether it is adult or neonatal fat tissue. The inversion of the index oleic/palmitic that normally occurs in the newborn due to the higher proportion of saturated to insaturated fatty acids determines a higher melting point and a lower solidification point of the adipose tissue. These features of neonatal fat

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imply a higher tendency to crystallization when exposed to risk factors.[6]

We evaluated five patients with clinical and/or histopathological diagnosis of SCFN detected in 2001-2008.

The objective of this work is to describe risk factors, clinical presentation, and evolution of our patients.

Clinical cases

We describe five patients with diagnosis of SCFN (**Table 1**)

Case 1

A 28-day-old female newborn; developed at 25 days of life, a firm nodule on the neck.

Perinatal history: Full term birth weight appropriate for gestational age, vaginal delivery, controlled pregnancy, abruptio placentae, cigarette-smoking mother. She stayed hospitalized for 7 days for birth asphyxia requiring halo oxygenotherapy (72 hours) and luminotherapy for 4 days. Laboratory tests were within normal ranges. Lesions regressed at 45 days of life.

Histopathological: areas of subcutaneous tissue necrosis with inflammatory cells and giant cells. Remaining adipocytes show fusiform spaces with radially arranged needle shaped clefts. Diagnosis: SCFN.

Case 2

A 19-day-old male patient; at 4 days of life he showed indurated, erythematous plaques with painful skin colored nodules located in the upper back area, left arm and left cheek. Perinatal history: ful term neonate, birth weight was 4.060, born by cesarean section; controlled pregnancy; maternal hypertension; thick, meconial amniotic fluid; and fetal bradycardia. During the perinatal period, he developed hypoglycemia, hypothermia, moderate pulmonary hypertension, and mild hypertrophy of left ventricle; other laboratory tests were within normal ranges. Histopathology: No significant epidermal or dermal alterations were observed; adipocyte necrosis with septum breakdown and the presence of discrete lymphohistiocytic inflammatory infiltrate were found in the fragment corresponding to subcutaneous tissue. Remaining adipocytes showed fusiform spaces with radiated layout. Diagnosis: SCFN.

Case 3

A 4-day-old boy showed an indurated erythematous plaque located on trunk and right upper limb, which appeared at 2 days of age. Perinatal history: Full term, birth weight 4.330, born by cesarean section, controlled pregnancy, gestational hypertension. Was reffered to neonatal unit care for transient respiratory distress requiring oxygen for 24 hours. Laboratory tests were within normal ranges.

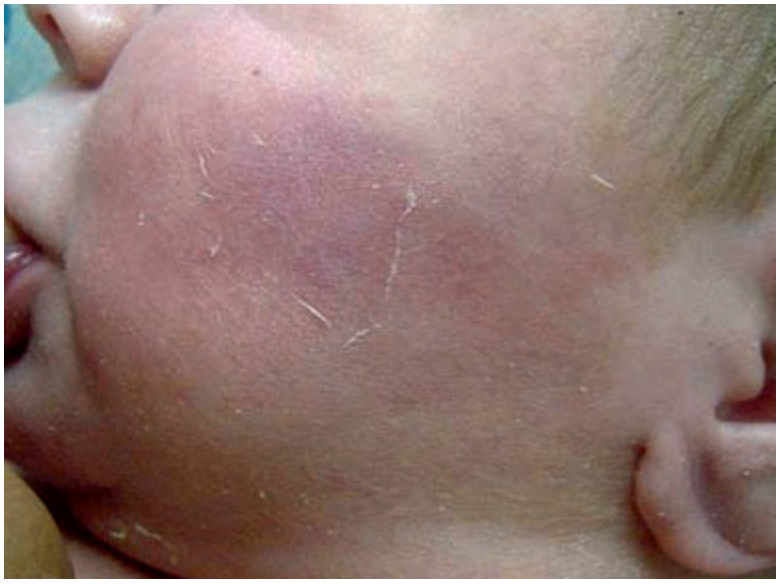

Figure 1. *Patient 2. Erythematous plaque on left cheek.*

TABLE 1. FILIATION DATA AND PERSONAL MATERNAL AND NEONATAL HISTORY.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Gender	Female	Male	Male	Male	Female
Age	28 days	19 days	4 days	32 days	18 days
Age at Onset	25 days	4 days	2 days	24 days	15 days
Delivery	Vaginal	Cesarean section	Cesarean section	Cesarean section	Cesarean section
Pregnancy history	Abruptio placentae	Gestational pre-eclampsia	Gestational pre-eclampsia	Gestational pre-eclampsia	None
Perinatal history	40 weeks Weight 3.950 Perinatal asphyxia	41 weeks Weight 4.060 Meconial amniotic fluid. Hypoglycemia, hypothermia, pulmonary hypertension, left ventricle hypertrophy.	Full term Weight 4.330 Transient respiratory distress	Perinatal asphyxia Seizures. Admitted for bronchiolitis at 28 days of age.	42 weeks Weight 2.740 Respiratory distress. Halo for 48 hours.
Age of lesion disappearance	45 days of age	Unknown	Unknown	60 days of age	60 days of age

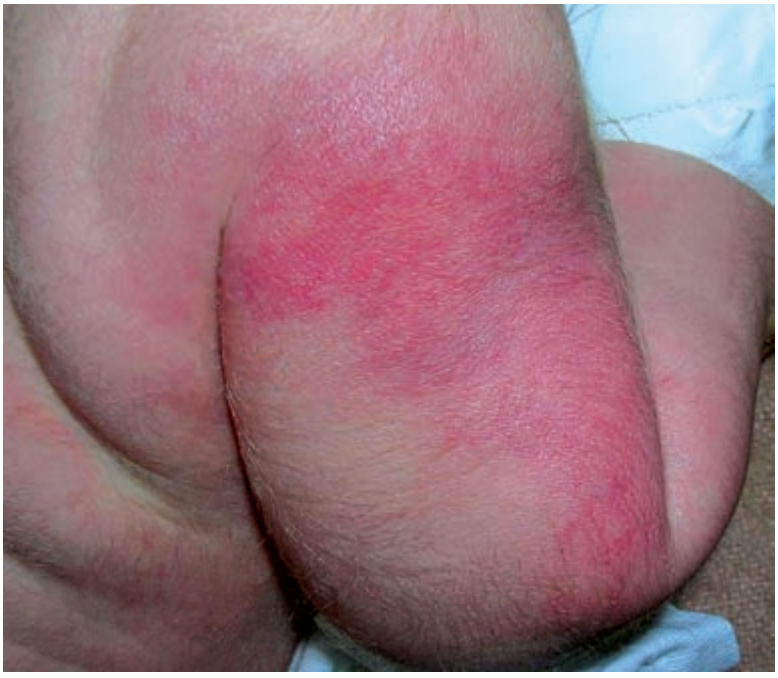

Figure 2. *Patient 3. Erythematous plaque on right upper limb.*

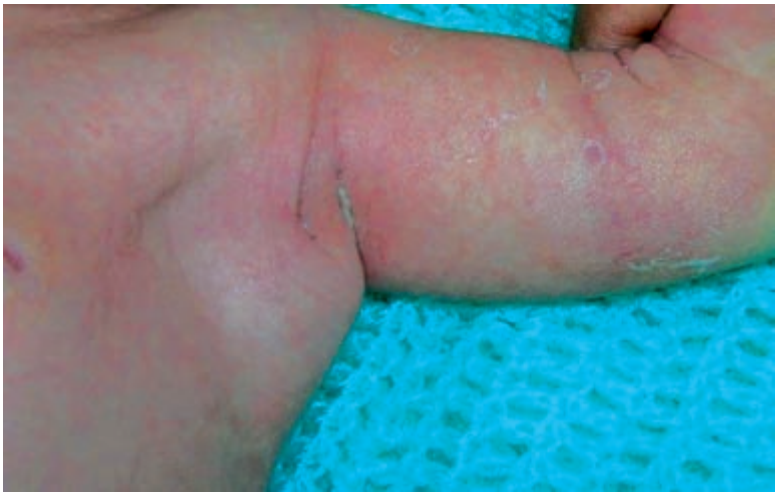

Figure 3. *Patient 4. Indurated plaque with superficial telangiectasia on left upper limb.*

Histopathology: areas of subcutaneous cell tissue necrosis with mild inflammatory infiltrate and giant cells. Remaining adipocytes showed needle-shaped clefts. Diagnosis: SCFN.

Case 4

A 32-day-old male patient; he showed an indurated plaque of hard, stony consistency, 2 x 2 cm in diameter, with superficial telangiectasia, surrounded by a whitish halo, located on the medial aspect of the left arm, appearing at 24 days of life. Perinatal history: full term neonate with adequate weight for gestational age born by cesarean section after a controlled pregnancy with maternal hypertension. He stayed hospitalized for 14 days for perinatal asphyxia requiring halo oxygen therapy. He was readmitted at 28 days of age for bronchiolitis. Laboratory tests were within normal ranges. Lesions resolved at about two months of age.

Histopathology: areas of subcutaneous tissue with adipocyte necrosis and septa rupture. Remaining adipocytes showed fusiform spaces with radiated layout. Mild lymphohistiocytic infiltrate. Diagnosis: SCFN.

Case 5

An 18-day-old female patient; she showed erythematous and indurated plaques located on trunk and left cheek, appearing at 15 days of age. Perinatal history: Posterm (42 weeks of gestation),birth weight was 2.740 g, born by cesarean section, controlled pregnancy. She stayed hospitalized for transient respiratory distress for 48 hours. Laboratory tests were within normal ranges. Lesions disapeared within the second month of life. The parents refused to perform skin biopsy.

Discussion

Risk factors, clinical manifestations, complications, and outcome of five SCFN patients were assessed in this work.

SCFN is a rare disorder occurring in Full term or post term neonates.

Four of the five patients studied were full term babies, 3 with adequate weight and one large for gestational age; the remaining patient was a post term girl with adequate weight for gestional age.

Although the etiology of this disorder is unknown, it was associated with perinatal hypoxia,[4,7] aspiration of meconial amniotic fluid, hypothermia,[7] local trauma,[4,5] and anemia.[3] In our series, all patients showed perinatal hypoxia, one patient presented meconium aspiration, and another hypothermia. Although trauma has been described as a triggering factor for this disorder, a higher incidence has been reported in children born by cesarean section than by vaginal delivery. Four of the 5 patients were born by cesarean section.

With reference to maternal risk factors, the following are described: gestacional diabetes gestational hypertension, exposure of cocaine or calcium channel, use of blockers during pregnancy, and cigarette-smoking.[4] In our series, 3 mothers had gestational hypertension, one mother was a smoker, but none had gestional diabetes or used toxic substances.

Clinically, SCFN is characterized by multiple nodules or indurated erythematous plaques in trunk, buttocks, limbs, thighs, and cheeks. Some lesions may contain calcium, as in patient 1. The presence of calcifications justifies the use of sim-

ple X-rays for study purposes. Lesions typically develop in the first 6 weeks of age,[1] and may appear from the first 7 days to12 months of life, like in our patients. In most cases, lesions are spontaneously self-limiting in 2 to 4 weeks with no atrophy or residual scar.[6] All our cases resolved in the first 2 months of age (about 20 days after onset). In some cases, lesions may be very painful and require opioid management.[2]

Hypercalcemia is the most severe but infrequent complication developed by these patients. It has been described in about 25% of the cases, generally in the presence of more extended lesions.[7-12] Mechanisms involved in hypercalcemia include increase in prostaglandin E, alteration of parathormone homeostasis and aberrant levels of vitamin D.[1] Hypercalcemia may appear up to 6 months after lesion onset, therefore all newborns must be adequately monitored in this period of time.[9] Treatments described for this complication include: adequate rehydration, loop diuretics, prednisolone, and etidronate.[2,10] If hypercalcemia occurs, extracuataneous calcification foci must be searched for at renal, myocardial, and hepatic level. Platelet and lipid levels must also be monitored. Thrombocytopenia is an early systemic complication of unknown origin.[4,9] A relationship with history of familial dyslipemia exists in SCFN children. Hypertriglyceridemia may develop after skin lesions appear and resolve subsequent to their regression.[4] None of our patients had these complications.

Histologically, normal epidermis and dermis is noted, with mixed inflammatory infiltrate and abundant histiocytes. Hypodermis shows adipocyte necrosis with multinucleated giant cells forming needle-shaped cleft granulomas in radial layout.[8] Biopsy confirmed diagnosis in 4 of our patients.

Main differential diagnoses are rhabdomyosarcoma, myofibromatosis, infantile hemangioma, neurofibromas and escleredema neonatorum.[12,13] Although clinical presentation of these lesions and general condition involvement of the newborn are very different, image diagnosis methods (ultrasonography, computerized axial tomography, and nuclear magnetic resonance),[13] and finally biopsy may be resorted to in order to attain certainty diagnosis.

In conclusion, we wish to highlight that our case series reflects what is published in the literature. It is important to understand the associated risk factors and complications in order to reach adequate diagnosis and follow-up.

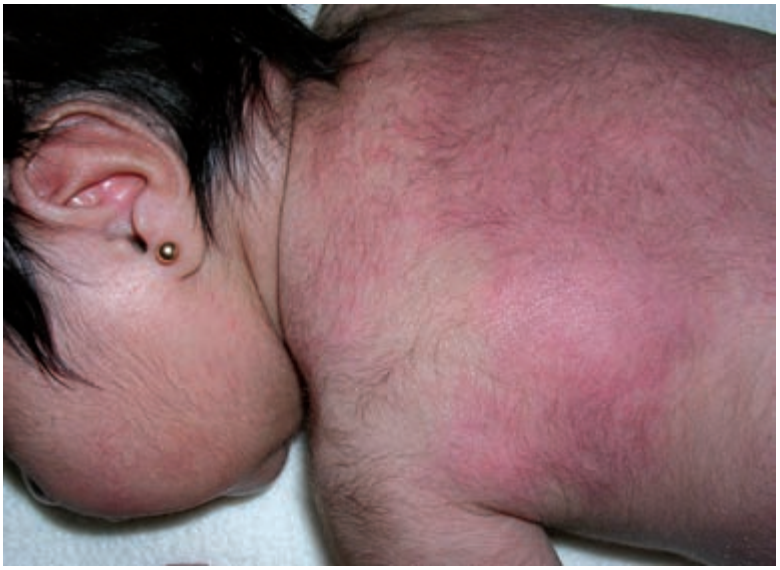

Figure 4. *Patient 5. Erythematous and indurated plaque on the back.*

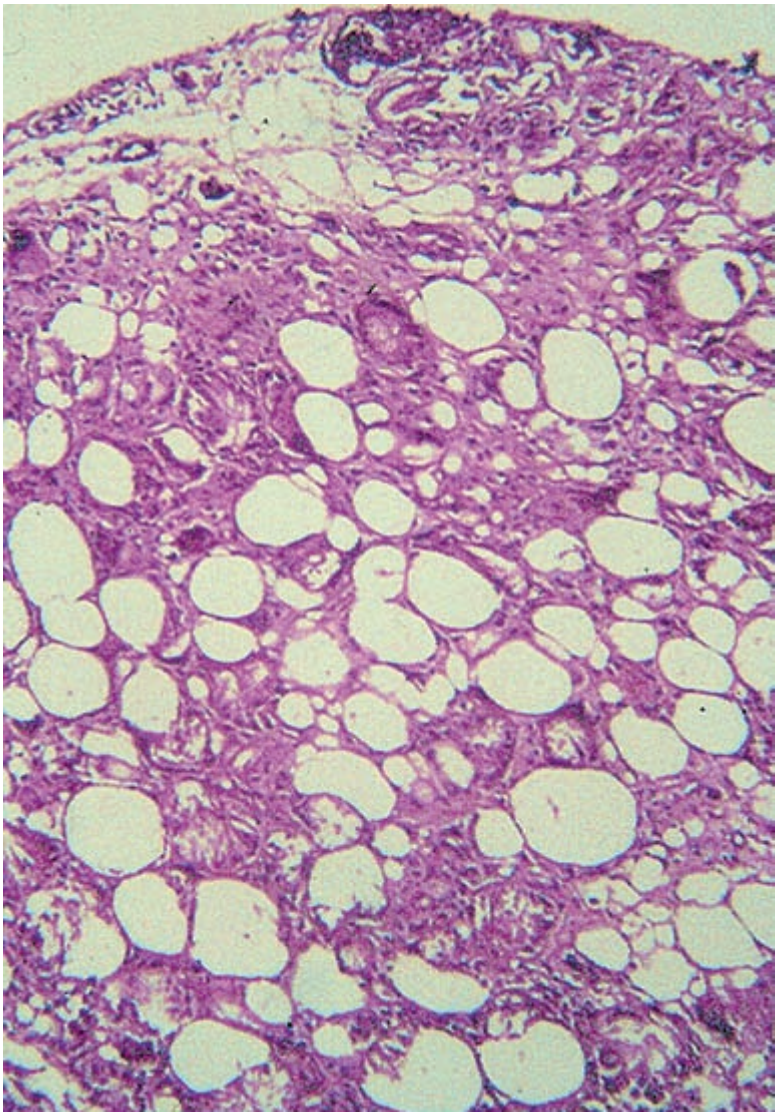

Figure 5. *Histology: Adipocyte necrosis with granuloma formation (H-E 100×).*

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