CASE REPORT

Subcutaneous fat necrosis in a newborn
Necrose de tecido adiposo subcutâneo em um recém-nascido

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ABSTRACT
We report the case of an asphyxiated newborn with subcutaneous fat necrosis and a favorable outcome up to the present. Some pathophysiological, clinical and therapeutic aspects of this condition are discussed.

Keywords: Necrosis; Adipose tissue/pathology; Adiponecrosis; Newborn; Hypercalcemia; Perinatal asphyxia; Hypothermia

RESUMO
É descrito um caso de necrose do tecido subcutâneo em um recém-nascido asfixiado com evolução satisfatória até o presente momento. Alguns aspectos fisiopatológicos, clínicos e terapêuticos da afecção são discutidos.

Descritores: Necrose; Tecido adiposo/patologia; Adiponecrose; Recém-nascido; Hipercalcemia; Asfixia neonatal; Hipotermia

INTRODUCTION
Subcutaneous fat necrosis (SFN) or adiponecrosis of the newborn is a rare, acute hypodermatitis with spontaneous resolution that appears in the first several weeks of life. Probably several factors leading to a prolonged and complicated delivery interact in the development of SFN. The lesion presents as red-purplish indurated subcutaneous nodules and plaques. Hypercalcemia is a complication frequently associated with this condition and may present as lethargy, irritability, hypotony, vomiting, polyuria, polydipsia, dehydration, constipation, and failure to thrive¹. The diagnosis is clinical and confirmed by histological examination of the lesion. The course of the disease is usually benign, but special attention should be given to the prevention and treatment of hypercalcemia.

CASE REPORT
The patient was a 4065g male infant, born by cesarean section indicated because of fetal bradycardia and intrauterine meconium. His mother was a multiparous woman who had attended two prenatal visits. She reported being smoker, was on the 39 1/7 week gestation based on her last menstrual period, and had ruptured membranes 5 hours earlier. The 1, 5 and 25-minute Apgar scores were 2, 6, and 7, respectively. The patient was assisted by neonatologists who visualized his trachea and performed tracheal suction of a large amount of meconium. He was transferred to the neonatal ICU and was placed on mechanical ventilation. He had a seizure one hour after birth and hypoglycemia three hours after birth, reversed with intravenous administration of sodium phenobarbital and glucose. Biochemical determinations on the first day of life revealed normal sodium, potassium, and total calcium levels, and low magnesium level which was adequately treated.

Transfontanellar ultrasonography (US) on the second day of life demonstrated hyperechogenicity of the parenchyma and cerebral edema. Repeated three days later, the test revealed that the cerebral lesions had resolved.

On the seventh day of life, the presence of several nodules was observed. They were of different sizes, indurated, firm, with no increased local heat and mildly erythematous in the posterior region of the trunk, compatible with adiponecrosis (figure 1). Microscopic

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examination revealed the presence of adipocytes with refringent crystals with a radial arrangement which confirmed the presumptive diagnosis (figure 2).

With 27 days of life, the US revealed normal renal tissue and total serum calcium level was 9.6mg/dL. He was discharged the next day with 4900g, persistence of the dorsal nodular lesions and was exclusively breast-fed with no vitamin supplementation.

We have been following his clinical and laboratory outcome and, up to the last visit, when he was two months old, the dorsal lesions, although smaller, still persisted and the serum calcium level remained within normal limits.

This benign disease of unknown etiology may go unnoticed because it improves without treatment\(^5\). Its frequency is unknown, and it affects different races and both genders equally\(^5\).

The exact pathophysiology of SFN is unknown, and different mechanisms for its development have been proposed\(^5\). It is attributed to cold, obstetric trauma, fetal distress, sepsis, and peripheral circulatory collapse\(^4\,7\,9\).

Physiological immaturity in the development of enzyme systems affects fatty acid desaturation resulting in a predominance and persistence of saturated fatty acids in the neonate, with a relatively high melting point within the subcutaneous tissue\(^3\,4\). Local pressure trauma during delivery, hypoxia, and hypotermia may lead to a decrease in blood supply to fat tissue as a consequence of the peripheral vasoconstriction\(^10\). Since fat is very sensitive to lack of oxygen, vasoconstriction induces lesion, resulting in its solidification\(^3\,5\,6\).

The infants usually appear well and are afebrile\(^5\). The lesion starts as an area of edema progressing to non-painful, indurated, well-circumscribed subcutaneous nodules and plaques of varying sizes and low mobility over deeper planes and with no increased local heat\(^3\,9\). The overlying skin is red or purple and may be taut and shiny\(^4\,6\,9\).

Although the patient has not developed hypercalcemia up to the present moment, this is a well-known complication\(^1\,3\,4\,7\). It can develop asymptotically 1-2 months following SFT or be life threatening due to cardiac and neurological complications. Renal alterations may develop due to persistent nephrocalcinosis and nephrolithiasis\(^2\,5\).

The pathogenesis of hypercalcemia is unknown. The occurrence of an extra-renal deregulated production of 1,25 (OH)\(_2\) vitamin D by granulomatous cells that appear in the sites with SFN is accepted\(^6\,11\).

Seizures presented by this newborn were not related to hypercalcemia and were interpreted as a result of post-asphyxia cerebral edema.

The diagnosis is essentially clinical. Biopsy or fine-needle aspiration may be performed in uncertain cases and especially when normocalcemia is present, as in this case\(^5\).

Histologically, giant cells and lipocytes with varying sizes, ruptured, with intra-adipocyte doubly refractile crystals on polarized light microscopy, with a bundle or star-like arrangement are patognomonic\(^3\,5\,6\). These findings were present in this case.

SFN usually has a benign self-limited course with spontaneous resolution within a few weeks\(^5\,6\). Serum calcium levels should be monitored periodically until the skin lesions disappear\(^3\,5\).

Skin lesions require only symptomatic treatment, which was the management followed in this case\(^5\).

**DISCUSSION**

SFN of the newborn was first described by Harrison and McNee, in 1926\(^2\). It is an acute, rare, localized, well-circumscribed hypodermatitis that appears in the first several weeks of life and usually affects full-term or post-term infants\(^3\,7\).
Hypercalcemia, if present, is treated with forced diuresis using hydration and/or diuretics, with prednisone in the most severe cases\(^{(3-5, 8)}\).

The prognosis of SFN is excellent and usually with no sequelae\(^{(5)}\).

REFERENCES


