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Case Report

Subcutaneous fat necrosis in newborn: case report

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ABSTRACT

Subcutaneous fat necrosis of the new-born (SCFN) is an unusual form of panniculitis, with few cases described in medical literature. The disease affects new-borns at term or post-term, with normal general health. We describe a case of 5 day old new-born affected by the disease since birth. Also, we discuss diagnostic approach and the differential diagnosis.

Keywords: Subcutaneous fat necrosis (SCFN), Newborn

INTRODUCTION

Subcutaneous fat necrosis of the newborn is a rare disease, characterized by being a 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, obstetric trauma, anemia, thrombocytopenia, and gestational diabetes, preeclampsia and maternal exposure to use of cocaine or calcium channel blockers during pregnancy. Although rare, the most serious complication is the development of hypercalcemia that my occur up to six months after the skin lesions appear.

We report a case of subcutaneous fat necrosis of the newborn with no detectable risk factor.

CASE REPORT

A 5-day-old male newborn, product of uncomplicated and unassisted normal vaginal delivery, to a primigravida mother, Full term pregnancy (38 weeks gestation), born with an APGAR score 9 and 10 at 1 and 5 minutes respectively (Birth weight was 3700 grams).

Presented with history of swelling on both cheeks since the first day of life, that increased in size in the following few days. Upon a presentation, physical examination revealed the presence of hard, mobile, well circumscribed non tender oval-shaped subcutaneous nodules, measuring approximately 2x1 cm over right and left cheeks .The overlying skin was normal. Otherwise, the physical exam was unremarkable.

MRI of the neck showed deep seated right sided 17x16x14 mm mass, abutting the ramus of the mandible; it is relatively well defined of mixed low and fatty signal. The adjacent muscles and osseous structures are intact .A similar one is present on the left side. This is associated with an excess of subcutaneous fat, bilaterally, more pronounced on the right. The findings were considered compatible with subcutaneous fat necrosis (Figure 1). Initial investigations revealed normal calcium level (9 mg/dl).

Over the next 4 weeks, the masses continued to enlarge slightly; after that each mass had reduced to approximately 50% of its former size. During this interval, the patient's calcium levels were monitored and remained normal.

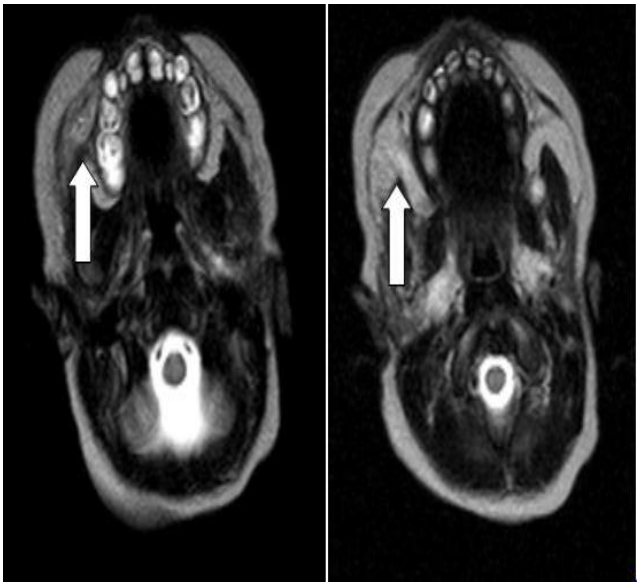

Figure 1: MRI of the neck showed deep seated right sided 17x16x14 mm mass, abutting the ramus of the mandible; with mixed low and fatty signal.

DISCUSSION

Subcutaneous fat necrosis affects full-term or postterm infants and is characterized by the appearance, days to weeks after a complicated perinatal period, of one or more well-defined, nonsuppurative, erythematous or violaceous, mobile subcutaneous masses, often with taut overlying skin. Lesions typically develop in the first 6 weeks of age.[1]

It may appear from the first 7 days to12 months of life. In most cases, lesions are spontaneously self-limiting in 2 to 4 weeks with no atrophy or residual scar.[2]

Although the etiology of this disorder is unknown, the causes could be attributed to perinatal hypoxia,[3,4] aspiration of meconial amniotic fluid, hypothermia,[4] local trauma[3,5] and anemia.[6]

The maternal risk factors that contribute to the development of SCFN include gestational diabetes, gestational hypertension, exposure of cocaine or calcium channel, use of blockers during pregnancy, and cigarette-smoking.[3]

Typically, the lesions develop on the shoulders, back, buttocks, thighs, and cheeks. It has been postulated that these lesions result from localized tissue hypoxia and mechanical pressure, which further compromise the local circulation.[7,8]

In the majority of cases, the lesions are spontaneously self-limiting in 2 to 4 weeks with no atrophy or residual scar.[2]

Hypercalcemia is considered the most severe complication, and has been described in about 25% of the cases.[4,9]

Although the pathogenesis of hypercalcaemia in subcutaneous fat necrosis of the newborn is not clear, several hypotheses have been proposed.[10-12]

The first one proposes release of calcium from the resolving subcutaneous plaques or nodules, but most cases do not develop hypercalcemia or have calcium deposition in the plaques. In another hypothesis, PTH and prostaglandin E2 have been proposed to stimulate bone resorption. According to the most accepted theory, hypercalcaemia in subcutaneous fat necrosis is related to aberrant extra renal production of 1, 25(OH) 2D3 from the granulomas of the lesions, stimulating intestinal calcium uptake.

Precursor 25(OH) D3 is metabolised to 1, 25(OH) 2D3 by the enzyme one alpha-hydroxylase (a process stimulated by PTH) which is mostly renal in origin but also detectable in some extra renal locations such as macrophages, keratinocyte, dendritic and placental cells. Farooque et al. demonstrated the presence of extra renal one alpha hydroxylase in immune cells associated with SCFN.

Hypercalcemia may appear up to 6 months after lesion onset, therefore all newborns must be adequately monitored in this period of time.[12]

Platelet and lipid levels must also be monitored. Thrombocytopenia is an early systemic complication of unknown origin. A relationship with history of familial dyslipemia exists in SCFN children. Hypertriglyceridemia may develop after skin lesions appear and resolve subsequent to their regression.[3]

Clinical diagnosis reveals a typical history of maternal complications such as gestational diabetes associated with obstetric trauma (usually dystocia) or fetal distress, coupled with typical clinical findings.

Imaging tests are usually not required for diagnosis, although they may play an important role in excluding other differential diagnoses.[9,14]

The diagnosis can be confirmed by aspiration cytology or skin biopsy. The histology is typical, showing areas of fat necrosis surrounded by a granulomatous reaction consisting of histiocytes, macrophages and giant cells and associated with calcification foci, with normal epidermis and dermis.[13]

The main differential diagnoses are rhabdomyosarcoma, myofibromatosis, infantile hemangioma, neurofibromas and scleredema neonatorum.[9,15]

Table 1: Main differential diagnosis with subcutaneous fat necrosis.

Differential diagnosis	Age	Features
Embryonal rhabdomyosarcoma	Appears after the first few months of life	Solitary, rapidly enlarging, firm mass that involves a striated muscle. On imaging, it is a densely cellular infiltrating neoplasm that may have sites of hemorrhage and necrosis.[16]
Infantile myofibromatosis	Neonatal period	The typical lesions are in the skeleton, lungs, heart, and gastrointestinal tract. The more superficial lesions tend to be desmoid fibromatoses, and these tend to develop in somewhat older children. On CT: Fairly homogeneous soft-tissue attenuation, usually with infiltrating margins that involve adjacent muscles and often bone.[16]
Hemangioma	It appears when the patient is several months old	Histopathology: Can be multiple, enlarge during the first year of life, and then involute nonencapsulated masses and dense cords of mitotically active, plump endothelial cells in close association with pericytes. Special stains reveal well-developed basement membranes around primitive vessels.[17]
Sclerema neonatorum	It is associated with prematurity	Diffuse hardening of the subcutaneous adipose tissues. Histopathology : The subcutaneous fat may appear normal or may have only sparse inflammation, which, when present, consists of lymphocytes, histiocytes, and multinucleated giant cells. This is thought to reflect the poor immunologic response of the infant. The most consistent findings are edema, a thickening of the subcutaneous fibrous septa, and a radial array of fine, needlelike clefts in the fat cells, representing former sites of fat crystals.[17]

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