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Complication of Subcutaneous Fat Necrosis of the Newborn: A Case Report and Review of the Literature

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Abstract: Subcutaneous Fat Necrosis (SCFN) of the newborn is uncommon, self-limited disorder that occurs in full term infant who experienced a perinatal distress in the first weeks of life. It can be complicated by life threatening hypercalcemia and other rare complication such as hypoglycemia, thrombocytopenia, hypertriglyceridemia, anemia and fever. SCFN with hypercalcemia frequently has been reported. We describe a case of subcutaneous fat necrosis with all of the above complications and discuss the etiology, clinical finding and complication of the disease.

Key words: Subcutaneous fat necrosis, newborn, hypercalcemia, hypoglycemia

INTRODUCTION

Subcutaneous Fat Necrosis (SCFN) of the newborn is a rare, transient, selfe-limited pathology of the adipose tissue which appears in full term infants who experienced perinatal distress in the first weeks of life. It is characterized by firm, erythematous nodules or plaques on the trunk, arms, thighs and cheeks. The condition may be associated with maternal disorders such as diabetes, preeclampsia or perinatal complication such as hypothermia, meconium aspiration, hypoglysemia and hypoxemia (Katz *et al*., 1984; Chuang *et al*., 1991; Burden and Krafchik, 1999). SCFN is usually a harmless, temporary condition but may be complicated by hypercalcemia, hypertriglyceridemia, thrombocytopenia, anemia and fever (Tran and Sheth, 2003; Hicks *et al*., 1993). Hypercalcemia is a rare complication, which can be life-threatening if not treated adequately (Cunningham and Paes, 1991). Even though spontaneous resolution without sequelae is the normal, patients should be followed for development of late complication of SCFN. We report a case of SCFN following perinatal distress from meconium aspiration, complicated by hypercalcemia, fever, hypoglycemia, thrombocytopenia and hypertri-glyceridemia.

CASE REPORT

The patient was a full term female infant born by cesarean section, with a birth weight of 3 kg. Delivery was complicated by meconium aspiration. Her Apgar score were 3 and 6 at 1 and 5 min. She had respiratory distress and required ventilatory support. On the second day of life, she had a focal seizure and was treated with phenobarbital, on the 17th day of life, she was discharged. The infant was readmitted to the same hospital 5 days after discharged from the hospital for skin lesions and fever. After evaluation for sepsis, since, she was suspected of having sepsis antibiotics were started but fever continued in spite of antibiotic therapy. On the 32 day old she was admitted to our hospital for evaluation of hypoglycemia, hypercalcemia, nephrocalcinosis and fever. On admission, she weighted 3100 g, her length and head circumference were 51 and 34 cm, respectively. Body temperature was 39.5°C, heart rat was 150 beats min^{-1} and respiratory rate 45 min. Multiple non tender, firm and violaceous subcutaneous plaques were noticed on her back (Fig. 1). Diffuse pitting edema was present on the back, thorax, face, palms and soles. A clinical diagnosis of subcutaneous fat necrosis was made. The baby 's mother refused a skin biopsy. The hemo globin level was 9 g dL^{-1}, leukocyte count 18700 mm^3 with 45% neutrophils, 50% lymphocytes, platelet count of 105000 mm^3, C-reactive proteine positive (3+). All of the cultures (blood, urine, cerebrospinal fluid) were negative. Serum calcium was 15.6 mg dL^{-1}, phosphotus 5 mg dL^{-1}, glucose 23 mg dL^{-1}, cholesterol 78 mg dL^{-1}, triglyceride 319 mg dL^{-1}, total protein 5.4 g dL^{-1}, albumin 3.4 g dL^{-1} and urine ca/creatinine: 0.6 (normal<0.2). The intact parathyroid hormone (iPTH) was appropriately suppressed at 2.5 pg mL^{-1} (normal 8.8-76.6 pg mL^{-1}). The 25 (OH) D3 level was normal at 20 ng mL^{-1} (normal 7-52 ng mL^{-1}) but the 1, 25 (OH) 2D3 level was elevated at 98 ng mL^{-1} (normal 9-40 ng mL^{-1}). A abdominal ultrasonography showed bilateral medullary nephrocalsinosis and Computed Tomoghraphy (CT) scan of the head was showed calcifications in the brain.

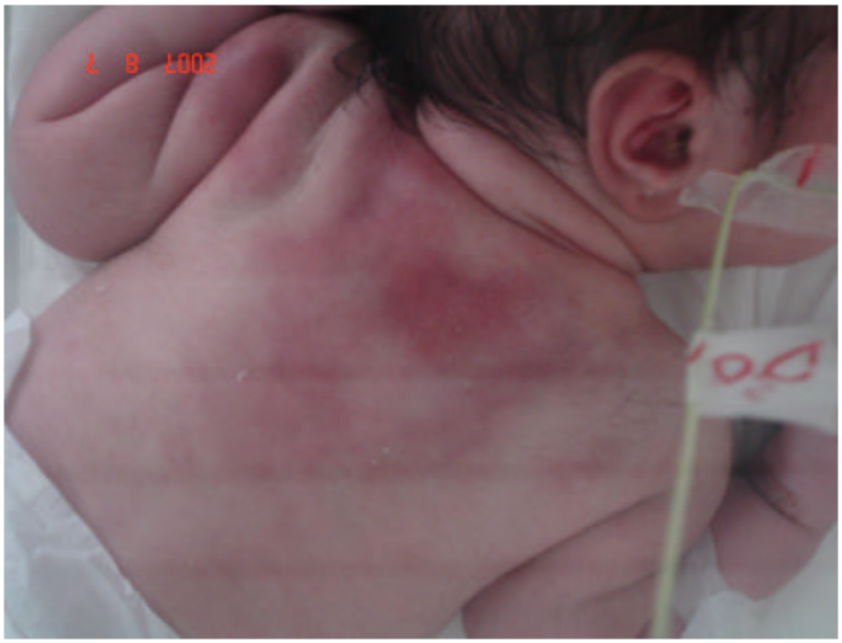

Fig. 1: Firm and violaceous subcutaneous plaques

The infant was treated with intravenous salin and glucose followed by IV furosemide 2 mg/kg/dose every 12 h and prednisolon 4 mg/m^2/d Po every 8 h. Fever continued along 28 days in spite of antibiotic therapy. Hypoglycemia controlled after 30 days.

The patients was discharged home after 1.5 month of hospitalization with serum calcium level of 10.8 mg dL^{-1}, urin Ca/creatinine: 0.2, serum triglyceride 180 and glucose 56 mg dL^{-1}. The skin lesions disappeared by the third month. The patient was followed by renal ultrasonography for nephrocalsinosis.

DISCUSSION

Subcutaneous Fat Necrosis of the Newborn (SCFN) is a rare disorder characterized by firm, erythematous nodules or plaques over the trunk, arms, buttocks, thighs and checks of full term newborn in the first several weeks of life (Katz *et al*., 1984; Chuang *et al*., 1991; Burden and Krafchik, 1999). In our patient lesions appeared after 3 weeks of birth. It was first described by Cruise in 1875 and initially was called scleroderma of the newborn infants (Klaiudom, 1999).

The cause of SCFN is unknown. Neonatal stress and hypothermia from various sources, such as Rh factor incompatibility, meconium aspiration (as our patient), hypoxia, seizures and maternal disorders such as gestational diabetes play some role in investigating the processes (Katz *et al*., 1984). Lesions resolve spontaneously in several weeks to 6 months without any treatment, but may be complicated by hypercalcemia and other metabolic abnormalities (Freedberg *et al*., N.D.). Hypercalcemia is the most frequently reported, so hypoglycemia, hyperlipidemia, fever, anemia and thrombocytopenia have been reported.

The exact pathogenesis of hypercalcimia in SCFN is not known. It is postulated that cold or stress-induced injury to immature fat results in the development of solidification and necrosis. A granulomatous infiltrate forms subsequently and nonrenal absorption of calcium increases (Taieb *et al*., 1987; Silverman *et al*., 1986). Ghirri *et al*. (1999) reported hypercalcemia associated with extrarenal 1,25 $(OH)_2$ vitD production from the granolomatous cell of fat necrosis that stimulated intestinal calcium uptake. Other mechanisms include elevated prostaglandin E2 activity leading to osteoclastic activation resulting in increased bone turnover (Harrison and Harrison, 1979). Normally, PTH stimulates the production of 1,25 $(OH)_2D_3$ by the kidney. In our patient elevated 1,25 $(OH)_2D_3$ and low serum concentration of iPTH supports the hypotesis of extrarenal production of 1,25 $(OH)_2D_3$.

Infants with hypercalcemia may have poor feeding, vomiting, dehydration failure to thrive, irritability, constipation, electrocardiography changes (Hisks *et al*., 1993). Hypercalcemia usually develops 4-6 weeks after the appearance of skin lesions as in our patient (Lewis *et al*., 1994). Therefore, these infants should be followed closely

until the fat necrosis resolves. The parents of newborns should be educated about the sings and symptoms of hypercalcemia. Persistant hypercalcemia may result in nephrocalsinosis, cerebral, skin and myocardium calcification (Gu *et al*., 1995). Our case was complicated nephrocalsinosis and cerebral calcification. The aggressive treatment is required and includes hydration, low calcium and vitD diet and furosemide (Ghirri *et al*., 1999). Corticosteroids, calcitonin and bisphosphonate are alternative if the above measures fail, we were not able to have a decrease in serum calcium level with hydration and furosimde. Although, prednisolon added and it was able to control calcium level. Oral prednisolon effectively lowers serum calcium levels by interfering with metabolism of vitD to the active form 1,25 dihydroxy vitamin D. Prednisolon may also inhibit production of this metabolit by macrophage involved in the granolomatous inflammatory process. In contrast, etidronate reduced bone calcium turnover (Rice and Rivkees, 1999).

Nephrocalcinosis and nephrolitiasis secondary to hypercalcemia occur in 4-6 months of onset of hypercalcemia. Our patient presented in 4 weeks of age with hypercalcemia, hypercalciuria and nephrocalcinosis. Probably, the skine lesions were not noticed earlier.

Thrombocytopenia is another important complication due to sequestration within the subcutaneous tissue and elevated levels of prostaglandin E (Burden and Krafchik, 1999; Chen *et al*., 1981). It appears before or at the same time as the onset of cutaneous lesions. Our case had mild thrombocytopenia and it is unclear whether the thrombocytopenia is a consequence of subcutaneous fat necrosis or perinatal hypoxia, or both.

The inflammatory process, as high fever and elevated acute phase reactants may be due to high prostaglandin released from necrotic fat tissue that leading to unnecessary antibiotic therapy as in our case. We were not able to perform prostaglandin measurements.

The other complication of SCFN is hypoglycemia. Several authors reported, hypoglycemia in their patients with SCFN born to diabetic mothers and hypoglycemia was secondary to being an infant of a diabetic mother (Fretzin and Arias, 1987; Lusk and Greiman, 1988). Our case borne to non diabetic mother and developed hypoglycemia in the 4 weeks of life. In our patient hypoglycemia may be result of perinatal distress or complication of SCFN.

To date 2 reported case had associated hypertriglyceridemia (Vonk *et al*., 1993; Juli and Antia, 2003). In the case of reported by Vonk *et al* (1993), hypertriglyceridemia was noted during week 11 of life when the fat necrosis was still active and it was due to an elevated concentration of Very Low Density Lipoproteine (VLDL). In our case, hypertriglycerdemia developed during 5th week of life and similar to the case reported by Juli and Antia (2003) it was transient. The increased level of triglycerides may be due to mobilization of fatty acids from the adipose tissue.

CONCLUSION

SCFN is an uncommon condition with spontaneous resolution over several weeks to as 6 months. However, there are rare and life threatening complications, for which these patients must be regularly monitored: thrombocytopenia, hypoglycemia, hypertriglyceridemia and most importantly for signs and symptoms of hypercalcemia for up to 6 months after the appearance of the skin lesions.

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