

Case Report

Subcutaneous Fat Necrosis in a Term Male Neonate with Perinatal Asphyxia : A Case Report

Swathi Chacham^{1*}, Syed Muzammil Hassan², Jakkampudi Nagasravani³, Jillalla Narsing Rao⁴, Uppin Narayan Reddy⁵, A.S. Kumar⁶

¹Assistant Professor, Department of Pediatrics, Princess Esra Hospital, Deccan College of Medical Sciences, Hyderabad, India

²Senior Resident, Department of Pediatrics Princess Esra Hospital, Deccan College of Medical Sciences, Hyderabad, India

³Junior Resident doctor, Department of Pediatrics Princess Esra Hospital, Deccan College of Medical Sciences, Hyderabad, India

⁴Professor, Department of Pediatrics, Princess Esra Hospital, Deccan College of Medical Sciences, Hyderabad, India

⁵Professor and Head, Department of Pediatrics Princess Esra Hospital, Deccan College of Medical Sciences, Hyderabad, India

⁶Professor and Head, Department of Dermatology, Princess Esra Hospital, Deccan College of Medical Sciences, Hyderabad, India

***Corresponding author**

Swathi Chacham

Email: swathi.m.lahiri@gmail.com

Abstract: Subcutaneous fat necrosis (SCFN) is an uncommon and yet, a benign condition seen in full term neonates who experience perinatal asphyxia and or traumatic delivery. It is temporary, self limited condition of unknown pathophysiology. Characteristic lesions are indurated, erythematous nodules or plaques over the areas prone for pressure such as back, arms, buttocks, thighs and face. This condition is known to be associated with maternal disorders such as diabetes, pre-eclampsia and maternal substance abuse which leads to fetal hypoxia. Also, it is seen in perinatal complications like perinatal asphyxia, meconium aspiration syndrome, hypothermia, hypoxemia and hypoglycemia. The associated complications are anemia, thrombocytopenia, hypo or hypercalcemia. While the cutaneous lesions of SCFN are self limiting, the hypercalcemia might have a fatal outcome if unnoticed and untreated. Hereby we are reporting a 29 days old, term, male neonate with subcutaneous fat necrosis. A 29 days old male neonate was admitted with multiple, painful, erythematous and tender nodular lesions over the back and shoulders from tenth day of life. The neonate was born to a primi-gravida at term gestation by vaginal delivery, with birth weight of 2700gms. There was a history of prolonged labour and the neonate had perinatal asphyxia, no other maternal risk factors could be elicited. There was no history of birth trauma or other perinatal morbidities. However the infant had hypothermia. Investigations revealed positive sepsis screen, normocalcemia with anemia. Skin biopsy was consistent with SCFN. Infant received supportive care and the lesions regressed in follow up over a period of three months. In conclusion, we report a rare case of Subcutaneous fat necrosis in a term male neonate with perinatal asphyxia, hypothermia with normocalcemia and anemia.

Keywords: Hypercalcemia, Neonate, Perinatal asphyxia, Subcutaneous Fat Necrosis.

INTRODUCTION

Subcutaneous fat necrosis (SCFN) of the neonate is a rare dermatoses, which was described by Harrison and McNee in 1926[1]. It is a temporary, self limited panniculitis of unknown patho-physiology. This panniculitis is associated with traumatic delivery, perinatal asphyxia which result in hypoperfusion of subcutaneous tissue leading to necrosis and calcification of subcutaneous fat [2].

SCFN is characterized by benign, indurated, erythematous nodules or plaques over back, arms,

buttocks, thighs and face. Though these lesions are benign and resolve spontaneously during infancy, they can be complicated by hypocalcemia or life threatening hypercalcemia which requires close monitoring [1].

CASE REPORT

A 29 days old male neonate was brought, with multiple, painful erythematous nodular lesions over the back and shoulders from tenth day of life. The neonate was born to a primigravida at term gestation by vaginal delivery. There was history of prolonged labour and the neonate had delayed cry with low APGAR scores

(APGAR score : 4,6,7 at 1,5&10 minutes of life respectively). Soon after birth the neonate required hospitalization for respiratory distress and had neonatal jaundice on third day of life. It was treated with double surface phototherapy and other supportive measures and was discharged by fifth day of life. There was no history of maternal diabetes, cocaine abuse or maternal smoking or Rh-incompatibility. There was no history of birth trauma and administration of drugs known to cause cutaneous lesions. At admission vitals were stable and mild pallor was present. However other systemic examination (including neurological system was normal). Cutaneous examination revealed multiple, erythematous, firm nodular lesions over the back and shoulders (as shown in Fig. 1) which were tender with normal overlying skin. Subcutaneous fat necrosis was

considered on the clinical grounds and the infant was investigated. Investigations revealed positive sepsis screen (CPR-24 ug/dl, WBC count-15,000/cumm) and low Hemoglobin (hemoglobin was 11 gm%) with normal serum calcium levels (8.9 mg/dl). However blood culture was sterile. Infant was started on supportive treatment along with antimicrobial agents. Skin biopsy was done to confirm SCFN and the histology was consistent with SCFN (skin biopsy shows areas of fat necrosis surrounded by granulomatous reaction, consisting histiocytes, macrophages.). Infant was periodically followed-up clinically and biochemically and the lesions subsided over a period of three months and the serum calcium levels remained normal throughout.



Fig -1: back of the neonate showing erythematous, nodular lesions suggestive of SCFN.

DISCUSSION

Subcutaneous fat necrosis (SCFN) is a rare form of panniculitis seen in term and post term neonates due to perinatal asphyxia, traumatic delivery and other antenatal complications (gestational diabetes, maternal cocaine abuse, smoking, pre-eclampsia and Rh-incompatibility). This usually occurs in the initial few weeks of life and resolves spontaneously during infancy. SCFN is often complicated by hypothermia, hypoglycemia, meconium aspiration syndrome. The severity of etiological factors leading to SCFN also determines the severity of its complications [3].

A neonate has physiological immaturity of enzymes which cause fatty-acid desaturation. Generalized or local tissue hypoperfusion leads to deposition of these saturated fatty acids and necrosis of fatty tissue. Hypothermia leads to crystallization and necrosis of adipocytes. The brown fat of infancy is shown to have

higher percentage of the saturated palmitic and stearic acids than the oleic acid (a major component of adult yellow fat). These fatty acids have a higher melting point and tend to crystallize with hypothermic injury [4, 5]. Similarly, pressure injury (birth trauma) such as the obstetrician's hands or forceps also enhances the risk of fat necrosis. Other postulations include impairment of fat or triglyceride metabolism (exacerbated by neonatal stress) along with elevated prostaglandin E levels [4, 6].

Clinically, the lesions are characterized by areas of erythema, edema which progress to hard mobile, distinct painful plaques or subcutaneous nodules in areas subjected to trauma such as the back, buttocks, thighs and arms. These lesions tend to be multiple, well defined, non-suppurative, erythematous or violaceous, mobile subcutaneous masses with taut overlying skin. The nodules of SCFN may enlarge for

several weeks to months but ultimately regress spontaneously.

SCFN should be differentiated from other conditions like sclerema neonatorum, bacterial infections (erysipelas and cellulitis), CMV infection. Lipogranulomatosis (farber's disease), deep hemangioma and sarcomas (including rhabdomyosarcoma) also mimic the lesions of SCFN [2]. Sclerema neonatorum is a rare condition of the neonate manifesting as diffuse hardening of the subcutaneous adipose tissues. However, it is distinct from the localized lesions of subcutaneous fat necrosis and associated with prematurity and poor prognosis (in contrast to uncomplicated SCFN which has a good prognosis) [7].

The diagnosis depends on the clinical history (characteristic history of antenatal complications like gestational diabetes mellitus along with shoulder dystocia or fetal distress) along with other typical characteristics of the lesions. Imaging modalities like ultrasonography, CT scan aid in differentiating SCFN from other lesions [8, 9]. The definitive diagnostic modality is aspiration cytology and skin biopsy. Histopathology reveals areas of fat necrosis surrounded by granulomatous reaction, which consists of histiocytes, macrophages, giant cells along with foci of calcification.

The index case had typical history of perinatal asphyxia which is the most commonly reported perinatal risk factor for SCFN. However, other risk factors (GDM, birth trauma) were not associated with the index case. SCFN was diagnosed in the index case depending on clinical history, characteristic findings and was confirmed by skin biopsy.

Though SCFN regresses spontaneously in infancy, it can be complicated by systemic manifestations like hypoglycemia, anemia, thrombocytopenia, hypo and hypercalcemia and local complications include epidermal atrophy, ulceration, scarring or infections [3]. Hypercalcemia is the most common as well as life threatening complication occurring within six months of onset of lesions [1]. It manifests with lethargy, irritability, hypotonia, vomiting, polydypsia and polyuria.. However asymptomatic hypercalcemia can occur in neonates which also has high morbidity and mortality requiring immediate intervention. Etiology of hypercalcemia remains unknown, though it appears to be due to excess prostaglandin secretion from macrophages which in turn leads to increase in 1,25dihydroxycholecalciferol disregard of renal absorption and parathormone levels. The severity and duration of hypercalcemia is related to severity of skin lesions [10].

Hence, infants with hypercalcemia are closely monitored for both clinically and biochemically until

the skin lesions have completely disappeared. The management of the skin lesions (in the absence of complications) is symptomatic and supportive. As the index neonate did not have any associated complications it was managed with supportive measures.

CONCLUSION

A term male neonate with perinatal asphyxia, presenting with a rare manifestation of subcutaneous fat necrosis along with normocalcemia in the fourth week of life.

ACKNOWLEDGEMENTS

We are thankful to Mr. Mujahid for helping with the formatting of the case.

REFERENCES

1. Oliveira AC, Selores M, Pereira O; Fat necrosis of the newborn: report on two cases. *An Bras Dermatol.*, 2011; 86(4 Suppl 1): S114-117.
2. Fenniche S, Daoud L, Benmously R, Ben Ammar F, Khelifa I, Chaabane S *et al.*; Subcutaneous fat necrosis: report of two cases. *Dermatol Online J.*, 2004; 10(2):12.
3. Akın MA, Akın L, Sarıcı D, Yılmaz I, Balkanlı S, Kurtoğlu S; Follow-up during early infancy of newborns diagnosed with subcutaneous fat necrosis. *J Clin Res Pediatr Endocrinol.*, 2011; 3(4): 216-218.
4. Hicks MJ, Levy ML, Alexander J, Flaitz CM; Subcutaneous fat necrosis of the newborn and hypercalcemia: case report and review of the literature. *Pediatr Dermatol.*, 1993;10(3): 271–276.
5. Darmstadt GL, Kanzler MH; Pathological case of the month. *Arch Pediatr Adolesc Med.*, 1994; 148(1): 61-62.
6. Sharata H, Postellon DC, Hashimoto K; Subcutaneous fat necrosis, hypercalcemia, and prostaglandin E. *Pediatr Dermatol.*, 1995; 12(1): 43–47.
7. Jardine D, Atherton DJ, Trompeter RS; Sclerema neonatorum and subcutaneous fat necrosis of the newborn in the same infant. *Eur J Pediatr.*, 1990;150(2): 125–126.
8. Norton KI, Som PM, Shugar JMA, Rothchild MA, Popper L; Subcutaneous Fat Necrosis of the Newborn: CT Findings of Head and Neck Involvement. *AJNR Am J Neuroradiol.*, 1997; 18(3): 547–550.
9. Vasireddy S, Long SD, Sacheti B, Mayforth RD; MRI and US findings of subcutaneous fat necrosis of the newborn. *Pediatr Radiol.*, 2009; 39(1): 73-76.
10. Farooque A, Moss C, Zehnder D, Hewison M, Shaw NJ; Expression of 25-hydroxyvitamin D3-1alpha-hydroxylase in subcutaneous fat necrosis. *Br J Dermatol.*, 2009; 160(2): 423-425.