

An Interesting Neonatal Presentation

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Abstract

We present a 1 week old female new-born with extensive subcutaneous fat necrosis. Due to extensive necrosis, excision and skin grafting was done.

Keywords: Swelling; Subcutaneous; Fat; Necrosis; Hypercalcemia; New-born

Case Report

Our case was a full term female, 38 weeks of gestation, 7 days old, delivered by caesarean section after an attempt of vaginal delivery, 1st order of birth of non-consanguineous parents of low average socioeconomic class. She was presented with multiple swellings on the back, buttocks, left arm of one day duration, with rapid onset and progressive course. No maternal history of gestational diabetes or preeclampsia, with proper antenatal care. Patient was admitted to another NICU during the first 72 h of life due to respiratory distress diagnosed as transient tachypnea of the newborn. No history of trauma. No family history of similar condition, no history of diabetes, hypertension or blood diseases.

By examination; she was 3.8 kg in weight, 50 cm in length, with occipitofrontal circumference of 38 cm, anterior fontanel 5 × 5 cm, full. Her pulse was 170/min, regular with normal capillary refill time, blood pressure was 65/40, respiratory rate was 40 breath/min and temperature was 37.4°C. She was pale, with no jaundice, no cyanosis. Liver was 3 cm below costal margin, soft, not tender, rounded border, no splenomegaly. Chest and cardiac examination showed no abnormality. She had multiple huge swellings overlying back of left arm, back and gluteal region, purplish, mildly tender, firm with overlying edema at the periphery, with areas of necrosis (Figures 1 and 2).

Her blood count showed leucocytosis, total leukocytic count: $22.8 \times 10^3/\text{cm}^3$, hemoglobin: 6.5 g%, platelet count: $128 \times 10^3/\text{cm}^3$, C-reactive protein: 160 mg/dl, Prothrombin time: 18.5 s, Partial thromboplastin time: 70 s, INR: 1.6, fibrin degradation products: 15 mcg/ml (>40 mcg/ml indicates disseminated intravascular coagulopathy) and normal total and ionized serum calcium. Normal Protein C, S, anti-thrombin III assay. Initial blood culture and skin swabs showed no growth of organisms.

Differential diagnosis was considered; haemangioma, vascular malformation (kaposiform hemangioendothelioma or tufted angioma), hematoma, necrotizing fasciitis, purpura fulminans and subcutaneous fat necrosis.

CT brain was done for her and showed hyper dense material occupying brain sulci, basal cisterns and lateral and fourth ventricle mostly subarachnoid and intraventricular hemorrhage with dilatation in the ventricular system. Abdominal ultrasonography showed mild hepatomegaly with no focal lesions, other findings showed no abnormalities. Duplex ultrasonography detected no vascular lesions with no arterial, venous or mixed blood flow excluding the diagnosis of hemangioma or vascular anomaly. The presented lesion was likely to be cutaneous or subcutaneous tissue lesion which could be inflammatory.

Skin biopsy was consistent with subcutaneous fat necrosis with a

dense inflammatory infiltrate composed of lymphocytes, histiocytes and multinucleated giant cells. Some lipocytes were replaced by radially arranged needle-shaped clefts.

On the 13th day of life, the lesions of the back became fluctuant with a spontaneous drainage revealing a chalky non-purulent substance. Due to extensive involvement, excision with skin grafting was done. In addition, ventriculo-peritoneal shunt was inserted. That was the reason why serial follow up of the patient did not show hypercalcemia afterwards (Figure 3).



Figure 1: Firm purplish nodules on the back and back of left arm with areas of superficial necrosis.



Figure 2: Firm purplish nodules on the left buttock with areas of superficial necrosis.

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Figure 3: Two weeks after surgery.

Subcutaneous Fat Necrosis

Subcutaneous fat necrosis is a rare, temporary, self-limited pathology affecting adipose tissue of full-term or post-term neonates. It is a form of panniculitis [1]. Although exact pathogenesis is not clear, it is postulated that cold or stress-induced injury to immature fat cells results in the development of solidification and necrosis with formation of granulomatous tissue [2]. Suggested etiologies include fetal factors such as a primary defect in subcutaneous fat, perinatal complications (especially ischemic injury), local trauma, hypothermia or birth hypoxia, maternal disorders such as gestational diabetes, preeclampsia, or maternal exposure to cocaine or calcium antagonists [1]. For our patient the triggering factor could have been the decreased tissue perfusion from first days of hypoxia.

It is characterized by firm, mobile, discrete erythematous to purplish nodules and plaques over the trunk, arms, buttocks, thighs, and cheeks of full-term new-borns [3]. It is a benign condition that

does not require treatment. Skin lesions typically resolve over a period of weeks to several months. Patients should be followed for up to 6 months after the appearance of the skin lesions to detect manifestations of hypercalcemia [2]. In addition, patients should be evaluated and monitored for hypoglycemia, hypertriglyceridemia, anemia, thrombocytopenia [4]. The most important concern is hypercalcemia, which can lead to neurologic or cardiac problems, nephrocalcinosis and nephrolithiasis. Clinical signs of neonatal hypercalcemia include irritability, poor feeding and vomiting. Staining of biopsy specimens demonstrates increased levels of 1 alpha-hydroxylase within the granulomatous infiltrate. Alpha-hydroxylase promotes the conversion of 25 OH D3 to its active form 1,25 OH₂ D3. The treatment of hypercalcemia ranges from conservative measures such as hydration and restriction of vitamin D and calcium to more aggressive interventions such as furosemide, glucocorticoid or bisphosphonate therapy [5].

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