Occipital Scalp Nodule in a Newborn

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A 4-week-old male infant was referred to dermatology for evaluation of a nodule on the occipital protuberance of 2 weeks' duration. The patient was born at 36 weeks and 6 days' gestation via an emergency cesarean delivery due to fetal distress. He later was found to have hypoxic-ischemic encephalopathy, pulmonary hypertension, and hypertrophic cardiomyopathy. He underwent therapeutic hypothermia protocol treatment starting at less than 6 hours after birth. At the current presentation, physical examination showed a 2.5-cm, erythematous, firm, mobile nodule on the occipital scalp with some overlying crusting and minimal surrounding erythema. No other cutaneous features or lesions were present. Initial laboratory findings were remarkable for hypercalcemia at 11 mg/dL (reference range, 8.5-10.5 mg/dL). Magnetic resonance imaging showed a faint abnormality in the subcutaneous tissue in this region without a noted connection to the underlying brain/meningeal matter. A punch biopsy was performed.

WHAT'S THE **DIAGNOSIS?**

a. cephalohematoma
b. cold panniculitis
c. sclerema neonatorum
d. subcutaneous fat necrosis
e. tinea capitis

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The authors report no conflict of interest.

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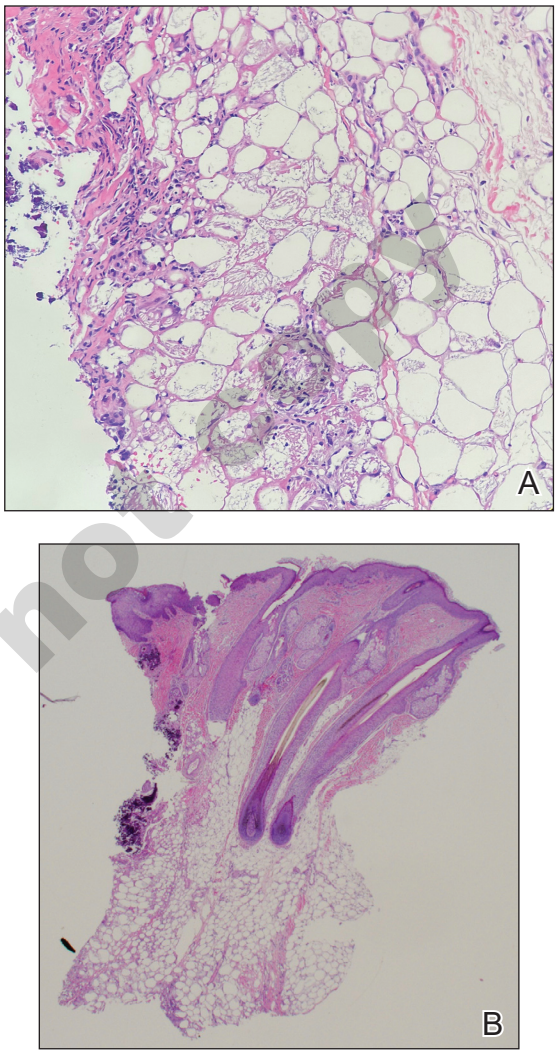
THE **DIAGNOSIS:** Subcutaneous Fat Necrosis

Histopathology revealed lobular panniculitis with lymphohistiocytic inflammation, lipid crystals, and calcifications in our patient (Figure). Subcutaneous fat necrosis (SCFN) was diagnosed based on these characteristic histopathologic findings. No further treatment was pursued.

Subcutaneous fat necrosis is a rare, self-limiting panniculitis that typically resolves within several weeks to months without scarring. It manifests as red or violaceous subcutaneous nodules or plaques most commonly on the buttocks, trunk, proximal arms and legs, and cheeks.[1] Histopathology reveals lobular panniculitis with dense granulomatous infiltrates of histiocytes, eosinophils, and multinucleated giant cells with needle-shaped crystals. Focal areas of fat necrosis with calcification also can be seen.[2]

The epidemiology of SCFN is unknown. Most cases occur in healthy full-term to postterm neonates who experience hypoxia, other prenatal stressors, or therapeutic hypothermia for the treatment of hypoxic-ischemic encephalopathy.[3] Although the etiology is unclear, certain inciting factors such as local tissue hypoxia, cold exposure, meconium aspiration, maternal diabetes, preeclampsia, and mechanical pressure have been proposed. Our patient underwent hypothermic cooling protocol, and it has been suggested that the increased saturated to unsaturated fat concentration in the skin of newborns increases the melting point, thus predisposing them to fat crystalization.[4] Cases of SCFN involving the scalp are rare; therefore, any newborns receiving hypothermic therapy for hypoxic-ischemic encephalopathy should have a thorough skin examination with possible biopsy of lesions that are characteristic of SCFN, such as red or violaceous subcutaneous nodules or plaques, for specific disease identification.

The main complication of SCFN is hypercalcemia, which occurs in approximately 50% of cases. Other serum abnormalities include hyperglycemia, hypertriglyceridemia, and thrombocytopenia, though these findings are not as well associated.[4] Patients with associated hypercalcemia may be asymptomatic, as in our patient, but other presentations include irritability, weakness, anorexia, vomiting, renal failure, failure to thrive, and encephalopathy. Nephrocalcinosis is a common complication of severe hypercalcemia; however, there is little evidence of associated major renal dysfunction.[5] The exact mechanism of hypercalcemia is poorly understood. A widely accepted theory postulates that a granulomatous inflammatory infiltrate upregulates 1-α-hydroxylase activity, which enzymatically converts 25-hydroxyvitamin D to its active form, 1,25-dihydroxycholecalciferol, which increases bone resorption and calcium absorption through the gastrointestinal tract and renal systems. Treatments for hypercalcemia include hyperhydration, calcium-wasting diuretics, and low calcium intake.[6] Furthermore, calcium levels should be obtained at the time of diagnosis and 30, 45, and 60 days after the lesions resolve.[4]



A and B, Histopathology revealed lobular panniculitis with lymphohistiocytic inflammation, lipid crystals, and calcifications characteristic of subcutaneous fat necrosis (H&E, original magnifications ×100 and ×20).

Subcutaneous fat necrosis needs to be differentiated from the more severe panniculitis, sclerema neonatorum (SN), which typically affects critically ill, preterm, and small-for-gestational-age newborns. It is associated with a high mortality rate and is characterized by skin and

subcutaneous hardening and fixation to underlying subadjacent tissue structures. The process typically begins in the thighs, buttocks, or trunk and spreads diffusely, sparing the fat-free palms, soles, and genitalia.[7] Although our patient was born preterm, the physical characteristics of the nodule and the lack of severe illness placed SN lower on our differential. Histopathologic differences between SCFN and SN involve the extent of tissue fibrosis and presence of inflammatory cells. Sclerema neonatorum typically manifests with thickened connective tissue with a sparse inflammatory infiltrate, including lymphocytes, histiocytes, and multinucleated giant cells.[7] Conversely, SCFN manifests with fat necrosis with an extensive inflammatory infiltrate. It is important to be able to distinguish between these 2 conditions, as both have vastly different prognoses.

Cold panniculitis, sometimes called "popsicle panniculitis," is a phenomenon in which cold contact with the skin causes eruption of firm, erythematous, indurated plaques at the site of exposure. This self-limiting condition typically appears hours to days after cold exposure and spontaneously resolves in a few weeks.[8] Therapeutic hypothermic protocol treatment involves using cooling devices to lower the body temperature for a short duration. The temperature typically is lowered to approximately 32 °C to 36 °C. These temperatures are not low enough to induce cold panniculitis, which is more commonly seen in facial ice applications when managing supraventricular tachycardia in neonates.

Cephalohematoma is a birthing injury that causes blood accumulation within the subperiosteal space. During parturition, the compressive and sheering forces on the calvarium rupture the vessels passing through the periosteum, causing blood to pool slowly into the subperiostium; thus, a cephalohematoma usually manifests later at 1 to 3 days of life as localized head swelling.[9] The bleeding typically does not cross suture lines and is primarily found in the occipital or parietal regions. The incidence has been reported to be 0.4% to 2.5% of all live births.[10] Although the location of the nodule in our patient was in the occipital region, imaging and biopsy results did not show hemorrhagic findings consistent with cephalohematoma. Management of cephalohematoma mainly is observational, as the mass slowly regresses and the accumulated blood gradually is reabsorbed.

Fungal scalp infections (tinea capitis) are common in the pediatric population. The peak incidence of this infection has been reported in children aged 3 to 7 years, with *Trichophyton tonsurans* and *Microsporum canis* as the usual causative organisms.[11] Clinical features of tinea capitis include scaly patches with hair loss, hair loss with black pigmented dots at the follicular openings, diffuse scalp scaling with subtle hair loss, and cervical lymphadenopathy.[12] Although less common, tinea capitis can progress to a more severe form known as a kerion, which is characterized by a tender plaque with pustules and crusting. A kerion can result in permanent scarring and alopecia if left untreated.[12] In our patient, a nodule with scaling and faint erythema was observed, but no black pigmented dots at the follicular orifices were present. Therefore, a potassium hydroxide wet mount preparation used to diagnose tinea capitis was unnecessary. Systemic oral antifungal therapy such as fluconazole or terbinafine is the standard treatment for tinea capitis.

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