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Cranial Masses in Sickle Cell Disease

A 17-year-old African male with homozygous sickle cell disease was admitted with a vaso-occlusive crisis of the lower limbs and lower back, further complicated with an acute thoracic syndrome. After 6 days of treatment with intravenous fluids, analgesics, and antibiotics, he developed a holocranial headache, nausea, and photophobia. Findings of the physical examination showed biparietal painful boggy masses (Figure 1). Neurologic examination was unremarkable, and there were no signs of intracranial hypertension. Ultrasound scan of the skull showed 2 heterogeneous fluid collections, compatible with subgaleal hematomas.

To exclude intracranial hemorrhage, computed tomography scan of the head was performed, which revealed an anterosuperior frontal epidural hematoma of 6 mm (Figures 2 and 3). Coagulopathy was excluded, and a conservative approach was decided. The masses were progressively reabsorbed, and he was discharged 22 days after admission, totally asymptomatic. One month later, cranial magnetic resonance imaging was performed, showing a reduction of the subgaleal hematomas, a complete resolution of the epidural hematoma, but also T2 hyperintense areas in the calvaria compatible with bone infarction (Figure 4). Nontraumatic spontaneous epidural hematoma is extremely rare, potentially fatal, and it may be concurrent with calvarial bone infarction and subgaleal hematoma.[1] The pathophysiology is still not fully understood and various explanations have been proposed: vaso-occlusion of the hematopoietically active skull bone with subsequent infarction and leaking of blood; rupture of epidural vessels next to the infarcted bone; acute expansion of hematopoiesis resulting in disruption of already thinner cortex skull bone, causing extravasation of blood to subgaleal or epidural spaces; and finally sludging of sickle cells causing insufficient venous drainage with subsequent congestion and hemorrhage.[1-4] Epidural hematoma is associated with high mortality; however, when concurrent with subgaleal hematoma and/or bone infarction, the survival rate seems to be 100%.[1] Nevertheless, as subgaleal hematomas can be associated with an underlying epidural hematoma, the threshold for requesting brain imaging should be low.[3] ■

Joana S. Gonçalves, MD
Pediatric Department
Hospital de São Francisco Xavier
Centro Hospitalar de Lisboa Ocidental
Lisbon, Portugal

Denil Tribovane, MD
Neuroradiology Department
Hospital Beatriz Ângelo
Lisbon, Portugal

Madalena Pires, MD
Pediatric Department
Hospital de Dona Estefânia
Centro Hospitalar Universitário de Lisboa Central
Lisbon, Portugal

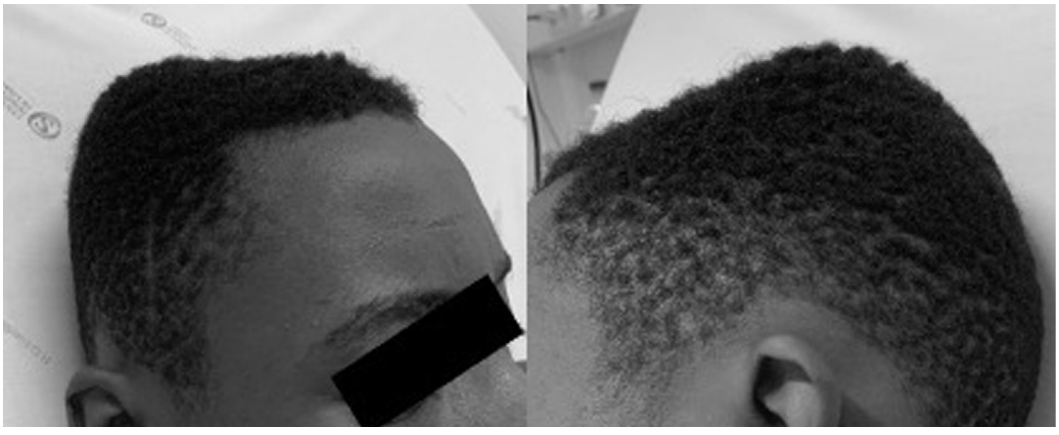

Figure 1. Bilateral parietal fluctuant masses.

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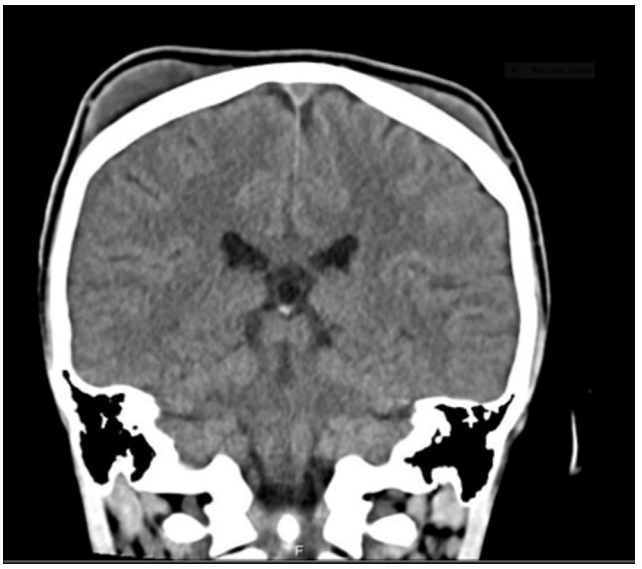

Figure 2. Coronal computed tomography scan image demonstrating both parietal subgaleal hematomas.

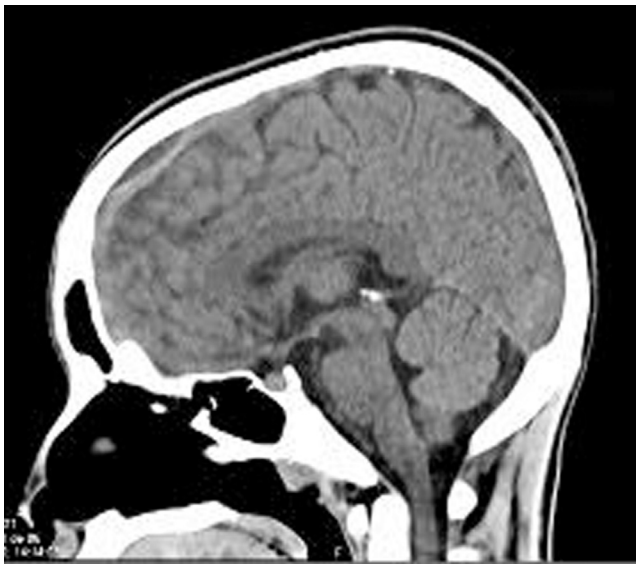

Figure 3. Sagittal computed tomography scan image showing the spontaneous epidural hematoma.

Rita Silva Lopes, MD
Pediatric Neurology Unit
Hospital de Dona Estefânia
Centro Hospitalar Universitário de Lisboa Central
Lisbon, Portugal

Marisa Raquel Inacio de Oliveira, MD
Hematology Unit
Hospital de Dona Estefânia
Centro Hospitalar Universitário de Lisboa Central
Lisbon, Portugal

Carla Conceição, MD
Neuroradiology Unit
Hospital de Dona Estefânia
Centro Hospitalar Universitário de Lisboa Central
Lisbon, Portugal

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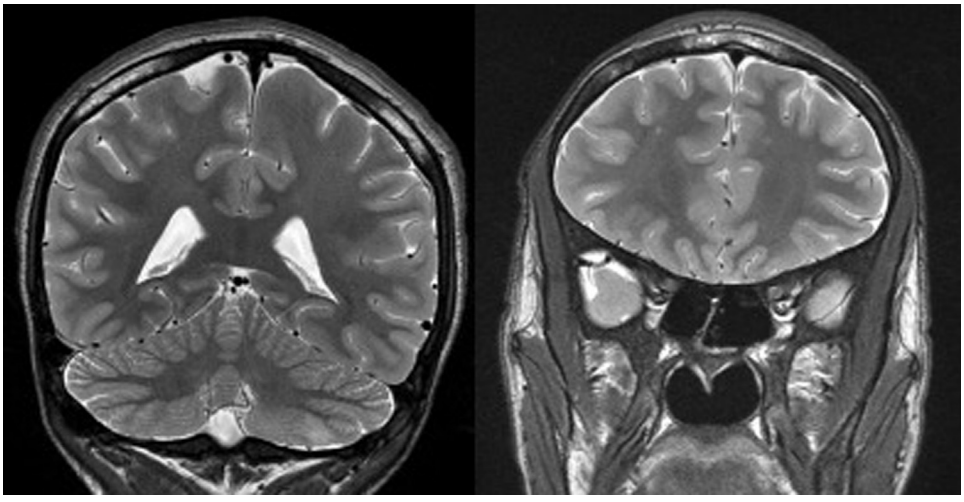

Figure 4. Coronal T2-weighted imaging depicting hyperintense areas in the calvaria, compatible with bone infarction, adjacent to the previous collections.

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Purulent Discharge from Stensen Duct in Neonatal Suppurative Parotitis

A 22-day-old male neonate presented to the emergency department with a 1-day history of fever associated with erythema on the left cheek (**Figure 1**). The pregnancy and vaginal delivery were uneventful. On admission, his body temperature was 38.4°, his heart rate was 77 bpm, and his blood pressure was 79/41 mm Hg. Findings of the physical examination revealed swelling and induration at the angle of the left mandible and a slightly protruding, brownish lesion with central redness at the opening of Stensen duct in the left buccal mucosa (**Figure 2**). Pressure to the left parotid gland expelled purulent exudate from the duct (**Figure 2**, **Video 1**). Methicillin-sensitive *Staphylococcus aureus* was detected in the pus. Laboratory tests demonstrated an elevated C-reactive protein level of 4.66 mg/dL, white blood cell count 20 130/μL, and normal serum amylase 6 U/L. Ultrasonography scan demonstrated swelling of the left parotid gland. Acute neonatal suppurative parotitis was diagnosed, and the patient was administered a 10-day course of intravenous antibiotic treatment.

Neonatal suppurative parotitis is diagnosed by the presence of parotid swelling, purulent discharge from the Stensen duct, and pathogenic bacterial growth in a culture of the pus.[1] Other symptoms include incessant crying, irritability, and erythema surrounding the orifice of Stensen duct (**Figure 1**).[2] Approximately one-half of patients with neonatal suppurative parotitis are afebrile, and the parotid swelling is bilateral in 10%-20% of cases despite bacterial involvement.[3-5] The orifice of Stensen duct is located in the buccal mucosa opposite the upper second molar, and the presence of pus is an important clue in the diagnosis and identification of the pathogen. The orifice should be closely observed while applying gentle, external pressure to the parotid gland. Laboratory findings are typically nonspecific. Elevated serum amylase occurs in only 10%-20% of cases because salivary isozyme activity is thought to be immature in newborns.[3-5] The known risk factors are preterm birth, breastfeeding, prolonged nasogastric feeding, and mechanical ventilation.[6] The differential diagnosis includes viral parotitis, cervical lymphadenitis, and cellulitis. *S aureus* accounts for approximately 60% of the causative pathogens, and gram-negative rods and anaerobic bacteria are found in 16% and 11% of cases, respectively.[4] Antibiotic treatment frequently consists of a first-generation cephalosporin in combination with an aminoglycoside or a third-generation cephalosporin.[3-5] The treatment duration is usually 7-14 days, which is relatively longer than for other conditions in the differential diagnosis. ■

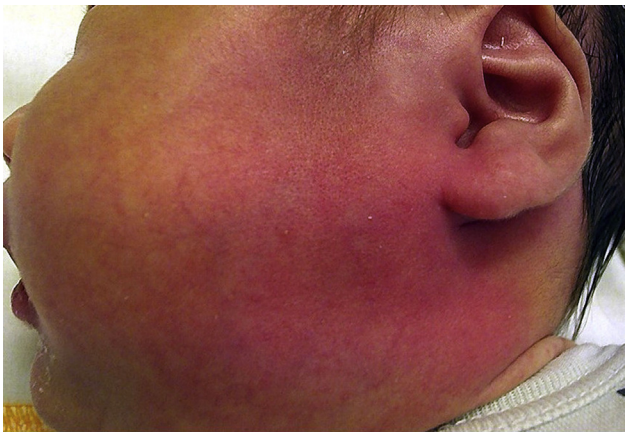

Figure 1. Erythema on the left cheek.

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Mami Ichinose, MD
Takahiro Matsushima, MD, PhD
Hiroshi Hataya, MD, PhD
Department of General Pediatrics
Tokyo Metropolitan Children's Medical Center
Tokyo, Japan

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