A
cute myeloid leukemia (AML) represents about 20% of acute leukemias. CNS hemorrhage is a complication that occurs more frequently in patients with AML than in those with acute lymphoblastic leukemia. It is caused by leukostasis in cerebral blood vessels, leading to leukothrombi, infarcts and hemorrhage and/or thrombocytopenia and coagulopathy.

A 2.5-year-old boy was admitted to our hospital with a progressive head enlargement three days after trivial head trauma due to falling on the ground while playing. He had no history of recent drug intake, fever or bleeding tendency. There was no family history of a bleeding disorder. On clinical examination, he was fully conscious and alert, but he appeared ill with severe pallor and a mild tinge of jaundice. There were no lymph node enlargements or other manifestations of bleeding tendency such as purpura or ecchymosis. The abdominal examination was normal. Head examination revealed a large soft swelling of the scalp demonstrated by pressure indentation with normal overlying skin (head circumference, 65 cm) (Figure 1). Neurological and fundus examinations were normal. Two days after admission, purpura and ecchymosis started to appear over the entire body.

Peripheral blood examination showed leucocytosis (total leucocytic count, 78×10⁹/L), severe normocytic normochromic anemia (hemoglobin, 4.5gm/dL) and thrombocytopenia (platelets, 12×10⁹/L). A peripheral blood film revealed the presence of 36% monoblasts. The bleeding profile was normal (aPTT=25 second, PT=12.5 second and INR: 1.1). Other laboratory investigations were a serum bilirubin of 1.3 mg/dL, alanine aminotransferase of 124 U/L, aspartate aminotransferase of 32 U/L and serum creatinine of 0.6 mg/dL. Bone marrow aspiration demonstrated 79% monoblasts, which confirmed the diagnosis of AML-M5 according to the FAB classification. X-ray of the skull revealed a radiolucent shadow surrounding the upper vault of the skull. CT scan of the head showed a large subgaleal hematoma with no intracranial hemorrhage, no shift of midline structures and no skull fractures (Figure 2).

The patient received four blood transfusions and a pressure bandage was applied to the scalp and forehead. The AML was treated by the MRC10 protocol² and the patient achieved complete remission with improvement of the scalp swelling and the general condition.

DISCUSSION
Most reported cases of subgaleal hematoma are neonates and the reported incidence of subgaleal hematoma ranges from 1.6-3/1000 live births.³ The risk fac-
images and diagnosis

Figure 2. A large subgaleal hematoma surrounding most of fronto-pari-temporal regions without intracranial hemorrhage or shift of midline structures, with normal skull base (CT image).

...tors are instrumental delivery, prolonged second stage of labor, precipitate labor, cougulopathy, prematurity, large infants, fetal dystocia and severe head moulding. Subgaleal hematoma occurring beyond the neonatal period is rare and is often associated with head trauma involving tangential or radial forces applied to the scalp causing emissary veins traversing the subgaleal space to be ruptured.

The diagnosis is generally made clinically, with a fluctuant bogy mass developing gradually over the scalp. The swelling frequently crosses suture lines. Imaging studies such as CT, magnetic resonance imaging, and ultrasound may assist in diagnosis. Detection of the cause of subgaleal hematoma is very important in its management. It had been reported in children with vitamin K deficiency, hemophilia, factor XIII deficiency and von Willebrand disease, which can be treated by adequate replacement therapy. It may also be due to hair combing, hair pulling or child abuse.

Hematological evaluation of a patient with a subgaleal hematoma associated with relatively mild or trivial trauma should include a complete blood cell count, prothrombin time (PT), and activated partial thromboplastin time (aPTT). Patients with a history of easy bruising or bleeding, or a positive family history, should also have an analysis of bleeding time, von Willebrand antigen, ristocetin cofactor, and factor VIII level to exclude von Willebrand disease. In our patient there was purpura and ecchymosis over the entire body, thrombocytopenia and leucocytosis detected by complete blood count, so a bone marrow aspiration was mandatory, which revealed AML-M5.

Most subgaleal hematomas do not require aspiration and drainage, as the risk of introducing infection outweighs the benefit of the procedure. The natural course is for spontaneous resolution in a few days or weeks without complication. Our patient was managed conservatively with frequent blood and platelet transfusions and a pressure bandage. No surgical aspiration was done to avoid infection and severe bleeding.

To the best of our knowledge, this is the first case to be reported with a large subgaleal hematoma in a child caused by a trivial trauma as an initial presentation of AML-M5.

REFERENCES