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A Rare Presentation: Intracranial Hemorrhage as a Symptom of Acute Leukemic Transformation in a 23-year old Male

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Abstract

This case highlights the urgency of considering acute leukemic transformation in young patients presenting with neurological deficits, emphasizing the importance of prompt evaluation and management to optimize patient outcomes. The case depicted is a tragic complication of Chronic Myeloid Leukemia (CML) and its acute blast crisis. Remarkably, the patient exhibited none of the typical constitutional symptoms associated with CML

Introduction:

Chronic myeloid leukemia (CML) is a myeloproliferative disorder where there is neoplastic proliferation of mature myeloid lineage cells.[1] CML accounts for between one and two cases per 100,000 adults, and the median age of diagnosis is 64 years.[1] There are three stages of CML. The chronic phase (CML-CP) represents 90–95% of all patients, the symptoms of which are due to bone marrow/hematological abnormalities [2] In the accelerated phase, blasts may increase to 10-19% with possible dysplastic changes, while in blast phase, blasts exceed 20% in peripheral smear or bone marrow, often with extramedullary involvement. [3] Blast phase may be myeloid (70%) or lymphoid (30%). Blast phase most commonly presents clinically as fever, splenomegaly, and bone pain.[4] CML is one of these acquired coagulopathies that can be rarely accompanied of ICH in acute blastic phase.[5]

Case Presentation:

A 23-year-old male with no significant past medical history presented to the emergency department (ED) with acute left-sided deficits. The patient, a new student on a visa, had complained of tooth pain approximately ten days earlier. Two days prior to presentation, he underwent dental cleaning without antibiotics or narcotics prescribed. Following the dental procedure, he experienced imbalance while walking and fell against a wall without head trauma or loss of consciousness. He had also complained of right arm pain, which was attributed to sleeping incorrectly.

On the morning of arrival to the emergency department, approximately three hours before arrival, he notified his friends of an inability to move or feel his left upper extremity, prompting transfer to the emergency department for evaluation. Approximately an hour prior to presentation, he called his friend reporting an inability to move his entire left arm. The patient's last known normal was at midnight. On arrival, the patient exhibited a National Institutes of Health Stroke Scale (NIHSS) score of 11, demonstrating an inability to move the left upper and lower extremity, speech impairment, and absence of movement in response to pain stimuli.

A stroke alert was activated, and imaging revealed an intracranial hemorrhage in the right temporoparietal region. The patient was subsequently admitted to the intensive care unit (ICU). However, upon ICU arrival, his Glasgow Coma Scale (GCS) deteriorated, necessitating a repeat CT scan that indicated progression of the intracranial bleed and showed increase size of the right front of Bridal prank on hemorrhage with new 4 mm right to left midline chef likely a component of cerebral edema resulting in effacement of the super cellular cistern. Consequently, the patient was intubated with ketamine and rocuronium for airway protection.

On arrival, the patients' vitals were blood pressure of 127/79, a rate of 91 beast per minute, a temperature 98.5°F, respiratory rate 24. SPO2 100% on room air. Her body Mass index was 27.06 kg/m².

Physical examination, the patient appeared ill and disoriented, with a Glasgow Coma Scale (GCS) score of 9/6, comprising an eye score of 4, a verbal score of 1, and a motor score of 4 on the right and 1 on the left. He was unable to speak and exhibited paralysis in both upper and lower extremities. Additionally, notable bruising was observed over the bilateral biceps of the arms. Otherwise, the examination was unremarkable.

Laboratory investigations revealed a relatively normal BMP, although there was a minor decrease in sodium levels at 132 and hyperglycemia with a glucose level of 141. The complete blood count (CBC) indicated leukocytosis with a white blood cell count of 89.5, while red blood cell count (RBC) was at 2.41, hemoglobin at 7.5, and hematocrit at 21.2. Platelet count was notably low at seven, with a mean platelet volume (MPV) of 11.4. Manual differential analysis of red blood cell morphology and platelet estimate showed one neutrophil, zero lymphocytes, zero monocytes, and 97 bands. The prothrombin time (PT) was 18.8 with an INR of 1.6. Urinalysis and urine drug screen (UDS) were negative. Troponin levels were within normal limits. CT scans of the chest and pelvis were negative for abnormalities. However, a CT scan of the brain revealed an anterior-posterior right frontal lobe hematoma, consistent with the patient's presentation following the fall.

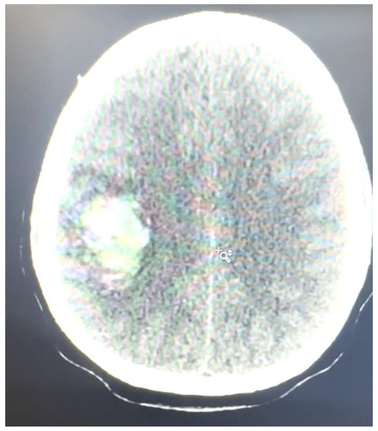

Image 1.- Initial CT Head- anterior-posterior right frontal lobe hematoma

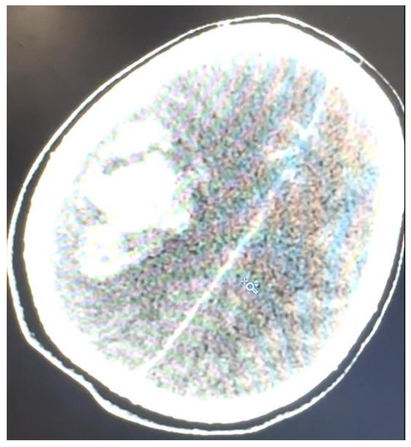

Image 2-Repeat CT Head two hours later after change of mental status

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Discussion:

The case above depicted a tragic complication of Chronic Myeloid Leukemia (CML) and its acute blast crisis. Remarkably, the patient exhibited none of the typical constitutional symptoms associated with CML, such as weakness, fatigue, night sweats, weight loss, fever, or bone pain [6]. The patient indeed presented with leukostatic symptoms, characterized by feelings of imbalance, and falling into a wall, which he initially dismissed. Some of the typical symptoms, include dyspnea, confusion and loss of coordination, are termed "leukostatic symptoms" as they arise from leukemia cells obstructing pulmonary or cerebral blood vessels [1].This unique presentation culminated in an acute blast crisis precipitating acute Intracranial Hemorrhage (ICH). The mechanism underlying this phenomenon involves the proliferating blasts, particularly of monoblastic lineage, which, due to their large size, contribute to increased blood viscosity, predisposing to stasis, tissue hypoxia, and endothelial injury, ultimately leading to thrombosis. Additionally, in Acute Myeloid Leukemia (AML) subtype M3, characterized by the French American British Classification, the presence of procoagulant granules may induce disseminated intravascular coagulation upon initiation of chemotherapy, resulting in consumption of clotting factors and platelets, leading to hemorrhage. Notably, hemorrhage in leukemia cases may also stem from qualitative or quantitative defects in platelets [4]. The prognosis at this stage is often fatal within the first 10 days. ICH is a grave prognosis indicator in either myelogenous or lymphoblastic leukemia and is second only to infection as the most common cause of death which is unresponsive to treatment.[7]

Conclusions:

Our clinical report contributes an additional case illustrating spontaneous acute hematoma and its correlation with Chronic Myeloid Leukemia (CML), underscoring several important conclusions.

Firstly, clinical judgment, along with the NIH Stroke Scale assessment and observed neurological deficits, should consistently prompt further investigation, including a potential head CT scan. Particularly in pediatric cases presenting with neurological deficits, consideration of intracranial hemorrhage and stroke should be paramount, despite the potential complicating factors of past medical history involving pseudo seizures or conversion disorder. However, this new case emphasizes the necessity of considering such conditions in the differential diagnosis. Secondly, the discovery of an acute hematoma in the absence of significant trauma warrants immediate additional investigation, including routine blood workup consisting of a complete blood count, peripheral blood film, a complete metabolic panel, and coagulation studies, which should be standard practice for all patients admitted to emergency medicine facilities.[5]