LETTER TO THE EDITOR
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Intracranial Hemorrhage as the Initial Presentation of Chronic-Phase Chronic Myeloid Leukemia in an Adolescent

Adölesan Bir Hastada Kronik Faz Kronik Miyeloid Löseminin İlk Klinik Bulgusu Olarak İntrakraniyal Kanama

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To the Editor,

Intracranial hemorrhage (ICH) is a rare initial manifestation of chronic-phase chronic myeloid leukemia (CML-CP), being far more often described in accelerated or blast phase, and few chronic-phase cases have been reported [1,2]. We describe an adolescent in whom multifocal ICH was the presenting feature of CML-CP despite a normal platelet count and normal clotting times.

A 17-year-old boy presented with a one-week history of headache, nausea, and vomiting. He was alert (Glasgow Coma Scale 15) without focal deficit, with hepatomegaly and marked splenomegaly (10 cm). The white blood cell (WBC) count was 479 × 10^9/L, hemoglobin 6.9 g/dL, and platelet count 247 × 10^9/L; prothrombin time, international normalized ratio, and activated partial thromboplastin time were normal, fibrinogen was 186 mg/dL, and D-dimer was mildly elevated (1.07 mg/L), without evidence of overt consumptive coagulopathy. Bone marrow examination confirmed CML-CP; conventional cytogenetics showed 46,XY,t(9;22)(q34;q11.2)[7]/46,XY[4], and real-time polymerase chain reaction detected the BCR::ABL1 p210 transcript at 24.3% on the International Scale. Cranial magnetic resonance imaging showed a 5 × 2.5 cm left frontobasal intraparenchymal hematoma with vasogenic edema, an 8-mm midline shift, and 4-mm caudal tonsillar displacement without clinical signs of herniation, plus a separate right frontoparietal hemorrhagic focus (Figure 1); the cerebral arteries and dural venous sinuses were patent, without ischemia on diffusion-weighted imaging.

Because he remained neurologically stable, he was managed without leukapheresis or neurosurgery, with supportive care, hydroxyurea (20 mg/kg/day) from day 2, low-dose cytarabine (30 mg/m^2 every 12 hours) on days 4–9, and imatinib 400 mg daily after molecular confirmation. Leukapheresis was avoided because the hemorrhage and edema were judged to raise the risk of neurological deterioration. The WBC count fell below 20 × 10^9/L by day 15, the hematoma resolved radiologically by month 2, and a deep molecular response (MR4.5) was achieved by month 12, without rebleeding.

A normal platelet count, normal clotting times without consumptive coagulopathy, and patent cerebral arteries and venous sinuses make thrombocytopenia, coagulopathy, and venous sinus thrombosis unlikely; in extreme hyperleukocytosis, the multifocal hemorrhage is most consistent with leukostasis, which we consider the presumed, though unproven, mechanism. Extreme leukocytosis raises blood viscosity and leukocyte aggregates obstruct the microcirculation, causing hypoxia and endothelial injury, compounded by cytokine and protease release from activated leukocytes; the resulting microvascular disruption can culminate in hemorrhage. Because circulating cells in CML-CP are mainly maturing granulocytes, less rigid and adhesive than blasts, leukostasis and ICH are rarer than

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in blast phase or acute leukemia, although risk rises with extreme counts and marked splenomegaly [3,4]. Central nervous system leukostasis may present with headache, dizziness, blurred vision, tinnitus, or somnolence and can progress to focal deficits, seizures, or ICH; such symptoms with extreme leukocytosis warrant urgent neuroimaging and leukoreduction [5].

Rapid leukoreduction had to be balanced against neurological safety: hydroxyurea and tyrosine kinase inhibitors lower the WBC count over 1–2 weeks, whereas leukapheresis acts within hours [5] but is less efficient in CML-CP and was considered hazardous here [6]. Imatinib was started after the hemorrhage; unlike dasatinib, it does not appear to impair platelet function [7], and no recurrent bleeding occurred. Acquired von Willebrand syndrome, which can occur in CML irrespective of platelet count, was not assessed because von Willebrand factor activity was not measured and remains an unexcluded possible contributor [8].

This case suggests that closely monitored pharmacologic cytoreduction, followed by early BCR::ABL1-directed therapy, can achieve complete radiological and deep molecular remission in a carefully selected adolescent. In our patient, this approach was feasible despite substantial mass effect and radiological midline shift, as he remained neurologically stable without clinical signs of herniation. A single case cannot define when leukapheresis or neurosurgery should be withheld; such decisions must be individualized through multidisciplinary collaboration, with serial neurological examination and neuroimaging.

Keywords: Leukemia, myeloid, chronic-phase, Intracranial hemorrhages, Hyperleukocytosis, Leukostasis, Imatinib mesylate, Adolescent

Anahtar Kelimeler: Kronik miyeloid lösemi, kronik faz, İntrakraniyal kanama, Hiperlökositoz, Lökostaz, İmatinib mesilat, Adölesan

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Conflict of Interest

The authors declare that they have no conflict of interest to disclose.

Authorship Contributions

Surgical and Medical Practices: V.K., F.T.Y., Z.Ö., E.E., A.F.; Concept: V.K., A.F.; Design: V.K., A.F.; Data Collection or Processing: V.K., F.T.Y., Z.Ö., E.E.; Analysis or Interpretation: V.K., F.T.Y., A.F.; Literature Search: V.K., F.T.Y.; Writing: V.K.

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The authors declare that they have no conflict of interest to disclose.

Declaration Regarding the Use of AI and AI-Assisted Technologies

During the preparation of this work, the authors utilized a generative artificial intelligence large language model to improve the language and readability of the manuscript. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication. No AI tools were used to generate scientific content, analyze data, interpret results, or draw conclusions.

Written informed consent for publication of this report and the accompanying image was obtained from the patient's parent/legal guardian.

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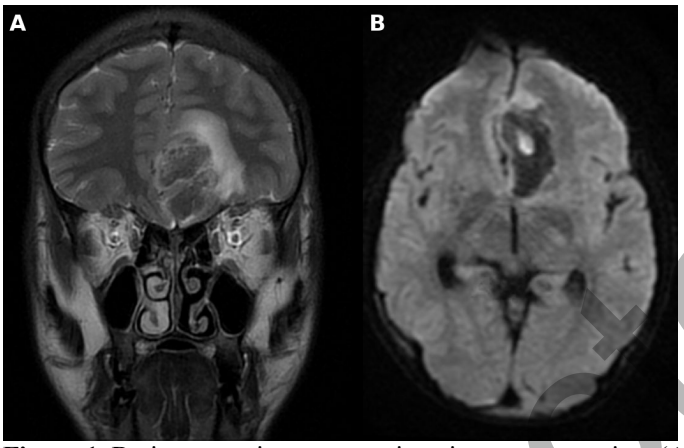


Figure 1. Brain magnetic resonance imaging at presentation. (A) Coronal fat-suppressed T2-weighted image showing the left frontobasal intraparenchymal hematoma with vasogenic edema and rightward midline shift. (B) Axial diffusion-weighted image showing the frontal hemorrhagic focus without acute ischemia.

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