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LETTER TO THE EDITOR

Cardiac tamponade as a symptom of the blast crisis of chronic myeloid leukemia

Dear Editor

We report the clinical findings of a study on a 29-year-old woman diagnosed with chronic myeloid leukemia (CML) blast crisis with an initial presentation of cardiac tamponade. She first visited our hospital because of dyspnea experienced for 1 month. Other associated symptoms were abdominal fullness and bilateral lower leg pitting edema. She denied having any underlying disease. At the time of admission, her blood pressure was 82/50 mmHg and pulse rate was 120/min, with a marked paradoxical pulse. Initial laboratory findings revealed a white blood cell count of $614 \times 10^3/\mu\text{L}$, hemoglobin level of 4.6 g/dL, and platelet counts of 198,000/ μL . The differential blood count showed 45% blasts, 10% promyelocytes, 4% myelocytes, 1% basophils, and 1% eosinophils, with normocytic normochromic erythrocytes (Figure 1B). Renal and liver functions were normal. An abdominal computed tomography scan revealed massive splenomegaly (Figure 1A), and bone marrow examination revealed hypercellularity, with a myeloid/erythroid ratio of 13:1 and 65% blasts. Moreover, the Philadelphia chromosome was observed in all analyzed metaphases, and polymerase chain reaction revealed a bcr-abl rearrangement on *P210 (b3a2)*. Flow cytometry revealed 96% CD33, 93% CD13, and an aberrant expression of CD7 and CD15. The diagnosis was Philadelphia-positive CML in blast crisis and a change in acute myeloid leukemia. Echocardiography showed a massive pericardial effusion with cardiac tamponade (Figure 1C). Furthermore, pericardial fluid cytology revealed leukemic cell infiltration (Figure 1D). No bacteria, fungi, or acid-fast bacilli were observed on repeated examinations. The patient was administered hydroxyurea, normal saline hydration (2500 mL/d), low-dose cytarabine (20 mg/m²/d) for 5 days, and dasatinib (100 mg/d). Pericardiocentesis drainage was also performed, and initially 1200 mL of yellowish

pericardial fluid was removed. Two weeks later, the patient's symptoms improved considerably, and she was discharged.

Fifty percent of patients with CML are asymptomatic at initial presentation, and CML is often diagnosed during a routine general health blood test or an abnormal hemogram. The most common symptoms of CML are fatigue, abdominal fullness, and left upper quadrant fullness. Pericardial effusion is a rare complication of CML and typically occurs in the chronic phase of CML [1]. Cardiac tamponade as the initial presentation in CML blast crisis is extremely rare; to date, only four cases have been reported [2–5], which occurred before the year 2000.

In a patient with acute leukemia harboring Philadelphia/BCR-ABL, the bcr-abl rearrangement often revealed *P190*, and a hemogram usually revealed thrombocytopenia. In our case, the bcr-abl rearrangement revealed *P210 (b3a2)*, the most common rearrangement area of CML. The hemogram revealed normal platelet counts with eosinophilia and basophilia, and physical examination revealed splenomegaly. The final diagnosis was blast crisis of CML.

The causes of pericardial effusion may be associated with leukemic cell infiltration, extramedullary hematopoiesis, infection, and bleeding in patients with CML. In our case, the mechanism of cardiac tamponade was leukemic cell infiltration (Figure 1D). The National Comprehensive Cancer Network guideline for treatment with CML blast crisis includes induction chemotherapy with a tyrosine kinase inhibitor (TKI) followed by hematopoietic cell transplantation (HCT) or TKI followed by HCT. In our article, the treatment was hydroxyurea, normal saline hydration (2500 mL/d), low-dose cytarabine (20 mg/m²/d) for 5 days, and TKI (dasatinib; 100 mg/d). Two years later, we checked the molecular response of BCR-ABL which revealed 3.2 log reduction (a major molecular response). We will arrange hematopoietic cell transplantation in the future. The outcome of cardiac tamponade as the initial presentation in CML blast crisis cited in the literature [2–5] was invariably fatal. Because in previous cases treatment was just with

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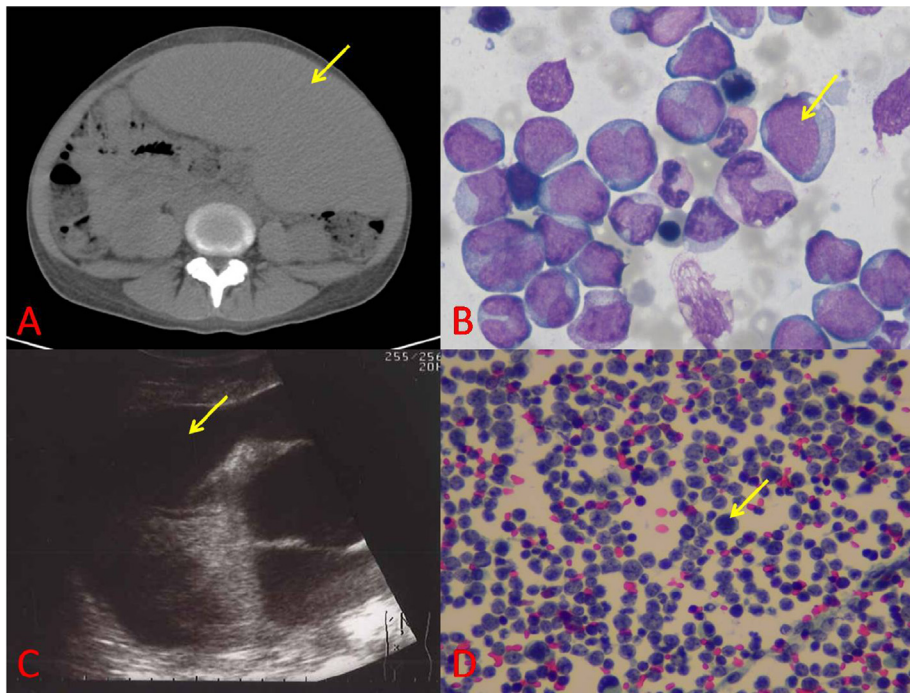


Figure 1. (A) An abdominal computed tomography scan revealed massive splenomegaly (arrow). (B) Initial laboratory findings revealed a white blood cell count of $614 \times 10^3/\mu\text{L}$ with 45% blasts (arrow; original magnification, $1000\times$). (C) Echocardiography showed a massive pericardial effusion with cardiac tamponade (arrow). (D) Pericardial fluid cytology revealed leukemic cell infiltration (arrow; original magnification, $400\times$).

hydroxyurea and pericardiocentesis, there was no treatment with TKI or HCT.

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