Vol. 5 No. 2 Year 2024
jurnal.umj.ac.id/index.php/MMJ
mmjfkk@umj.ac.id
e-issn: 2721–317X
Muhammadiyah Medical Journal

Case Report

A Patient with Suspect Chronic Myeloid Leukemia: A Case Report

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ABSTRACT

Chronic myeloid leukemia (CML) is a type of cancer that starts in the blood-forming cells of the bone marrow. In contrast to other types of leukemia, chronic myeloid leukemia (CML) progresses slowly. This report covers a 52-year-old male patient who came to the emergency room at Wangaya Regional Hospital with weakness, nausea, and pallor. His weight had drastically decreased over the last few months. The clinical condition of the patient was that both eyes appeared anemic, and the abdominal examination revealed hepatomegaly. Complete blood count examination results showed the following results: leukocytes 222.73x103/µl, hemoglobin 7.3 g/dL, platelets 679x103/µl, neutrophil 87.5%, neutrophil/lymphocyte ratio 16.66%. The results of peripheral blood smear analysis showed that hyperleukocytosis, with the maturation stage of myelocyte cells with a percentage of myeloblasts 6%, promyelocytes 7%, myelocytes 6%, metamyelocytes 8%, neutrophils 10%, neutrophil segments 60%, other cells 3%. These examination results suggest chronic myeloid leukemia. However, to confirm the diagnosis, the BCR-ABL fusion protein must be expressed, which can be used to identify the presence or absence of the Philadelphia chromosome in patients diagnosed with chronic myeloid leukemia (CML).

Keywords: chronic myeloid leukemia, myeloblast, neutrophil segments

INTRODUCTION

The blood malignancy known as chronic myeloid leukemia (CML), sometimes referred to as "chronic myelogenous leukemia", begins in the bone marrow (1). Most cases of CML occur in adults. Unlike other forms of leukemia, CML is a slow-growing disease that does not entirely interfere with developing erythrocytes, leukocytes, and platelets. Several factors can increase the risk of CML, such as gender, age, radiation exposure, and immunocompromised (2).

CML patients are found to have an abnormal chromosome called the Philadelphia chromosome. The Philadelphia chromosome is specific to CML and can also be found in acute leukemia patients (2). The ABL gene on chromosome 9 and the BCR gene on chromosome 22 are reciprocally translocated on the Philadelphia chromosome (2). ABL plays a role in signal transduction from adhesion receptors and cell surface growth factors to regulate the cytoskeleton structure of chromosome 9. The ABL gene mutation moves to chromosome 22 and fuses with the remaining part of the BCR gene. This gene fusion results in the formation of the BCR-ABL gene that causes leukemia. Additionally, transcription factors play a role in BCR-ABL signaling. In CML, STAT1 and STAT5 (signal transducer and activator of transcription) are transcription factors that are always active (2).

The clinical manifestations of CML constantly change according to the phase of the disease, namely the chronic phase (CP), accelerated phase (AP), and blastic crisis (BP). The chronic phase is found in most patients (90%-95%) (3). Common signs and symptoms of CP-CML due to anemia and splenomegaly include fatigue, weight loss, malaise, fullness, and a feeling of fullness in the left upper quadrant. In the accelerated phase, the disease progresses, characterized by increased leukocytes and enlarged spleen. The blastic crisis phase looks like acute leukemia. Patients are found to have a fever, malaise, splenomegaly, and weight loss.

The diagnostic approach in CML patients can be established by examining a peripheral blood smear from a patient with chronic-phase chronic myelogenous leukemia. In the chronic phase, blasts in the marrow typically make up less than 5%; greater levels (>10%) indicate a shift to a more aggressive course. Blast crisis of CML is defined as having 30% or more blasts in the blood or margin; however, more recent ideas may reduce the threshold to 20%, as has been suggested for acute leukemia (3).

CASE DESCRIPTION

A male patient, 52 years old came to the emergency room of Wangaya Hospital with complaints of nausea, pale and weak. Complaints of weakness and nausea were complained of since 1 month ago, the patient's complaint was accompanied by weakness, decreased appetite, easily tired when doing activities, there was a weight loss of >10% since 1 month ago. The patient had no previous history of disease, and the patient's family history of chronic disease was denied. The patient works as a private employee, history of radiation exposure is denied. The patient has a history of smoking since a young age, and the patient's diet is irregular**.** He often consumes food processed by burning and rarely consumes water.

On physical examination of the patient at the emergency room, he was found to be compos mentis with Glasgow Coma Scale (E4V5M6) vital sign results, blood pressure 120/80 mmHg, pulse 95x/min, respiratory rate 20x/min, temperature 37.2°celcius, oxygen saturation 96%. General examination obtained anemic conjunctiva in both eyes of the patient, on abdominal examination obtained tenderness in the left upper quadrant and enlargement of the liver and spleen scuffner IV organs. The patient underwent an ultrasound examination in July 2024 and was found to have hepatospleenomegaly. The results of complete blood support examination on day 1 MRS, obtained the results of leukocytes 222.73 103 / ul, hemoglobin 7.3 g / dL, platelets 679 103 / ul, neutrophils% 87.5%, lymphocytes% 5.3%, monocytes% 3.2%, eosinophils% 1.6%, basophils% 2.4%, neutrophil / lymphocyte ratio 16.66. Electrolyte examination with the results of blood sodium 131 mmol/L, blood potassium 3.8 mmol/L, blood chloride 9.3 mmol/L. Blood chemistry examination with the results of SGPT 18 U/L, SGOT 18 U/L, glucose at 173 mg/dL, urea 46 mg/dL, blood creatinine 1.0 mg/dL. The results of peripheral blood smear examination (Figure.1) with the impression of normochromic normocytic erythrocytes, leukocytes impression of greatly increased numbers, obtained all stages of maturation of myelocytic series with the presentation of myeloblasts (6%), promyelocytes (7%), myelocytes (6%), metamielocytes (8%), neutrophil stabs (10%), neutrophil segments (60%), other cells (3%), platelets impression of increased numbers (Figure.2), giant platelets (+), platelet clumps (-). The impression of peripheral blood smear results obtained peripheral blood cell morphology with a picture suspicious of chronic

myelocytic leukemia. The patient was treated for 8 days, during the treatment period the patient received prc (packed red cell) transfusion therapy as many as 4 bags.

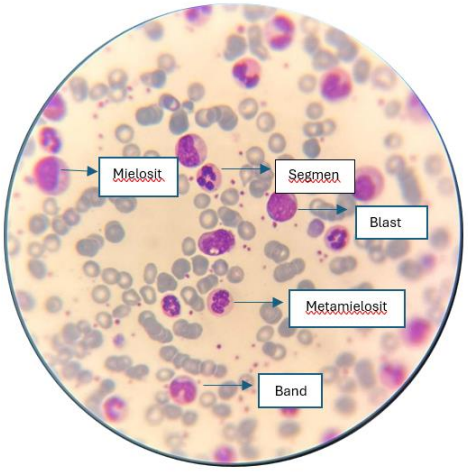

Figure 1. Peripheral blood smear result

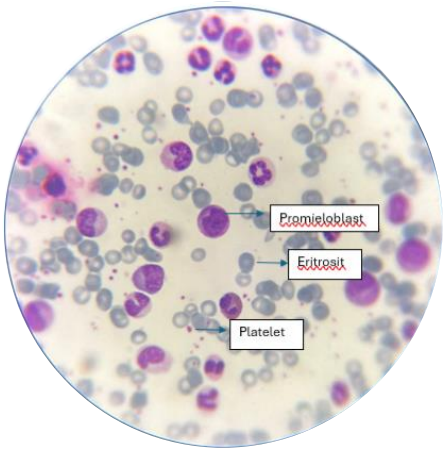

Figure 2. Peripheral blood smear result

DISCUSSION

CML is caused by an acquired genetic disorder or genetic damage to the DNA of a single bone marrow cell (2,5). The cause of CML is still unknown, but genetic and environmental factors, such as exposure to radiation and so on, play an important role. Mutated cells multiply into many cells (6). In fact, investigations conducted in vitro have demonstrated that high-dose radiation exposure to myeloid cell lines results in the development of BCR-ABL1 (7). The number of CML cells in the blood rises as a result of the unchecked proliferation of CML cells in the marrow. Erythrocyte, leukocyte, and platelet cell formation is not entirely disrupted by CML. Because of this, the chronic stage of myeloid leukemia is typically not as severe as the acute phase, and many people with it do not exhibit any symptoms at the time of diagnosis. Most cases of CML occur in adults. In the course of the disease, clinical manifestations that

are often found in patients diagnosed with CML are splenomegaly, weight loss, lethargy, and anemia. The clinical manifestations of CML patients always change according to the phase of the disease (8). The majority of CML patients receive a diagnosis during the chronic stage (90%-95%). Common signs and symptoms of chronic phase CML due to anemia and splenomegaly include fatigue, weight loss, malaise, fullness, and a feeling of fullness in the left upper quadrant. A rare clinical manifestation is bleeding associated with low platelet count (1). Peripheral blood smear examination in this chronic phase bone marrow contains less than 5% young (blast) cells. This phase often lasts for several years. Accelerated phase, in this phase the disease is progressive, with clinical manifestations of an enlarged spleen (9). This phase clinically shows a decreased hematologic response or molecular response and tends towards a blastic crisis (10). According to the criteria of the M.D Anderson Center, the accelerated phase is said when: blasts in the periphery ≥ 15%, promyelocytes in the periphery ≥ 30%, and basophils in the periphery ≥ 20%, thrombocytopenia (<10x109/L not due to side effects with therapy). The blastic crisis phase of the patient's clinical manifestations was found to resemble acute leukemia, the patient complained of frequent fever, malaise (feeling unwell), enlarged spleen, weight loss (11). This phase according to ELN is characterized by young cells ≥ 30% either in peripheral blood or bone marrow, while according to WHO criteria either in peripheral blood or bone marrow young cells ≥ 30%. One characteristic of CML is the BCR-ABL1 fusion gene. The expression patterns of distinct BCR-ABL1 transcripts change as CML advances from the chronic phase to the accelerated phase and finally to the blast phase (12). Molecular study also identifies the BCR-ABL gene. The most sensitive molecular diagnostic technique available is a quantitative polymerase chain reaction (13). Cells from bone marrow or blood can be used for this test. In essence, the PCR test "amplifies" or increases minute amounts of particular RNA or DNA fragments to facilitate their detection and measurement (14). Therefore, even when the BCR-ABL gene aberration is present in a relatively small percentage of cells, it can still be detected by PCR (15).

In this case, the patient was male, 52 years old. By the theory that says factors that increase the risk of CML are male gender and age> 50 years. On physical examination, it was found that the conjunctiva in both eyes of the patient looked anemic, abdominal examination found hepatosplenomegaly, this is by the theory that describes the current clinical condition of the patient leading to chronic phase CML. The bone marrow may develop aberrant white blood cells termed blasts as a result of CML, The spleen may grow as a result of CML cells building up there. Red blood cell deterioration may result from this, which may exacerbate anemia (1).

On complete blood examination, leukocytes were found to be 222.73 $x10^3$/µl, hemoglobin 7.3 g/dL, platelets 679 $x10^3$/µl, neutrophils 87.5%, neutrophil/lymphocyte ratio 16.66%. The results of peripheral blood smear analysis showed hyperleukocytosis, with all stages of myelocyte cells found with a percentage of myeloblasts 6%, promyelocytes 7%, myelocytes 6%, metamyelocytes 8%, neutrophils 10%, neutrophil segments 60%, other cells 3%. These supporting results are by the theory of CML in the chronic phase.

CONCLUSION

The patient's examination results suggest chronic myeloid leukemia. However, to confirm the diagnosis, BCR-ABL fusion protein expression must be performed, which can be used to identify the presence or absence of the Philadelphia chromosome.

ACKNOWLEDGMENTS

The author would like to thank to Laboratorium Installation of General Hospital of Wangaya Denpasar, Indonesia

CONFLICT OF INTEREST

None declared.

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