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Case Report OMICS International

Acute Renal and Hepatic Failure and Abnormal Blood Cell Count in Acute Leukemia: A Report of Four Cases and Review of the Literature

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Abstract

Leukemia is white blood cells cancer, which characterized with production of immature blood cells. Overcrowding of bone marrow with this abnormal immature cells, which interference with normal blood cells production, this may be lead to hyperleukocytosis, cytopenias, liver and kidney failure. In this study reports a case series of four patients with acute leukemia were studied. From this study, we concluded that hyperleukocytosis and cytopenias, disturbance in liver and kidney function were correlated with ALL than in AML.

Keywords: Acute leukemia; Renal failure; Hepatic failure; Cytopenias; Hyperleukocytosis

Introduction

Acute leukemia patients, Acute Lymphoblastic Leukemia (ALL) and Acute Myeloid Leukemia (AML), the clinical features are very vague and variable. However, certain patients with acute leukemia present with hyperleukocytosis and cytopenias [1].

Most of acute leukemia patients have organs enlargement as splenomegaly and hepatomegaly, this might be caused by infiltration of leukemic cells, which are seen in some cases of leukemia [2]. Acute liver failure considers as the initial manifestation of acute leukemia, also it is extremely rare, it is difficult to diagnose due to the rapid progression [3]. It has been described in acute lymphoblastic leukemia far more than in acute myeloid leukemia this due to blasts infiltration in ALL more than in AML [4].

In addition, acute renal failure has been reported as complicated feature in many of leukemia cases [5]. Multiplication and overgrowth of malignant cells increase rate of turn over nucleic acid and this can cause acute renal failure due to the release of urate salts, which may deposited in renal tubule and cause blocking it [6]. Also, high cell turnover may be causes elevated lactate dehydrogenase. However, leukemia patients characterized with infection and inflammation this may be reason for elevated the rate of erythrocyte sedimentation [1].

The present study presents four patients with acute leukemia. Cases 1 and 2 were ALL with abnormal hematological and clinical parameters, while cases 3 and 4 were AML with abnormal hematological and clinical parameters. Patients provided written informed consent.

Cases Report

Case 1: In January 2014, a 19-year-old man was admitted to Department of Hematology following one month bone pain and abdominal discomfort. During the previous week, this was accompanied by mild fever, fatigue, loss of appetite, and dark urine color in addition to enlargement in liver and spleen. He reported dyspnea, nausea, vomiting. He had paleness and bleeding. The patient's history included hyperglycemia. He had no risk factors for viral hepatitis or a history of liver disease. Bone marrow smear showed ALL (L2), and immunophenotyping studies showed acute B-lymphocytic leukemia. The bone marrow biopsy showed highly positively for B-cell lineage.

Physical examination upon admission revealed mild skin jaundice. There was slight tenderness over his right upper quadrant. There were splenomegaly and hepatomegaly. The rest of the examination was normal. Laboratory results showed a hemoglobin level of 8 g/dL, white blood cell count of 225.000/μL with 65% peripheral blood blasts, 75% bone marrow blasts and platelet count of 18,000/μL. The serum bilirubin level was 3.9 mg/ dL, aspartate aminotransferase 178 U/L, and alanine aminotransferase 74 U/L, INR 2.3, albumin 3.3 g/dL. Lactate dehydrogenase was 4272 U/L. Serum creatinine 5.4 mg/L and serum uric acid 7 mg/L. Erythrocyte sedimentation rate level was 65 mm/h and glucose level was 520 mg%. The patient succumbed to the disease 24 h later.

Case 2: In December 2012, a 60-year-old man was admitted to Department of Hematology due to decreased appetite and weight loss. Laboratory tests revealed that white blood cells count was 3.7×10^9/L with 21% peripheral blood blasts, 23% bone marrow blasts, hemoglobin count of 8.7 mg/L and platelet count of 4×10^9/L. The serum bilirubin level was 1.2 mg/dL, aspartate aminotransferase 29 U/L, and alanine aminotransferase 37 U/L, INR 2.3, albumin 3.2 g/dL. Lactate dehydrogenase was 765 U/L. Serum creatinine 1.1 mg/L and serum uric acid 8.8 mg/L. Erythrocyte sedimentation rate level was 13 mm/h and glucose level was 172 mg%. Bone marrow smear showed ALL, and immunophenotyping studies showed acute Tlymphocytic leukemia. The bone marrow biopsy showed highly positively for T-cell lineage. Dexamethasone (15 mg per day) was administered by intravenous infusion for five days to induce leukoreduction. This was then followed by combination chemotherapy including vincristine (2 mg intravenous injection on days 1, 8, 15, and 22), daunorubicin (40 mg/m^2 intravenous infusion on days 1 to 3, and days 15 and 16). The patient achieved complete remission after 45 days.

Case 3: In May 2014, a 43-year-old woman was admitted to Department of Gastroenterology, suffers from mild fatigue in addition to enlargement in liver and spleen. She reported no fever, bleeding or weight loss. Laboratory results showed a hemoglobin level of 9.5 g/dL, white blood cell count of 175.000/μL with 36% peripheral blood blasts, 45% bone marrow blasts and platelet count of 102,000/μL. The serum bilirubin level was 1.0 mg/dL, aspartate aminotransferase 58 U/L, and alanine aminotransferase 63 U/L, INR 1.0, albumin 4.3 g/dL. Lactate dehydrogenase was 1086 U/L. Serum creatinine 1.1 mg/L and serum uric acid 6.2 mg/L. Erythrocyte sedimentation rate level was 62 mm/h and glucose level was 209 mg%. Bone marrow smear and immunophenotyping studies showed AML [Acute Myeloblastic Leukemia, with granulocytic maturation subtype (M2)], according to the FrenchAmericanBritish classification on the basis of morphological features, and histochemical staining of cells with peroxidase and esterase [7].

Case 4: In November 2013, a 45-year-old male was admitted to the Department of Hematology due to fever for the past two days. The patient's WBC, hemoglobin and platelet counts were 8.2×10^9/L, 13 g/dL and 125×10^9/L respectively. Also, patient's serum bilirubin level was 1.0 mg/dL, aspartate aminotransferase 26 U/L, and alanine aminotransferase 101 U/L, INR 1.5, albumin 1.8 g/dL. Lactate dehydrogenase was 1200 U/L. Serum creatinine 0.8 mg/L and serum uric acid 6.4 mg/L. Erythrocyte sedimentation rate level was 60 mm/h and glucose level was 126 mg%. Bone marrow smear and immunophenotyping studies showed AML [Acute erythroid leukaemia's subtype (M6)], according to the FrenchAmericanBritish classification on the basis of morphological features, and histochemical staining of cells with peroxidase and esterase [7]. The patient achieved complete remission after 10 days.

Discussion and Conclusion

Acute leukemia is a heterogeneous disease with distinct manifestations and disturbance in different metabolic process occur in acute leukemia patients [8]. At the cellular level, acute leukemia's are rapidly progressing diseases, which characterized with over growth of immature malignant cell and this lead to decrease synthesis of mature normal blood cell due to suppression of hematopoiesis and this lead to anemia, thrombocytopenia, and hyperleukocytosis, all of this lead to fever, fatigue, infection, and bleeding [9]. Continuously divided and overproduced of immature cells in the bone marrow caused damage in some organs due to spreading (infiltrating) of blasts cells to other organs [10].

In this study, all cases were subjected to hematological and clinical studies. Two cases from four cases (Cases 1 and 2) were classified according to bone marrow film and immunophenotyping to ALL, one case was B-ALL and the other T-ALL, while the other cases (case 3 and 4) were classified as AML, and according to FAB classification divided in to one case (AML-M2) and the other (AML-M6).

Hyperleukocytosis and cytopenias have been seen in all acute leukemia case except case 2 and case 4. In case 2, there was leucopenia while in case 4 there was normal white blood cells count. In AML, most of studies have found that hyperleukocytosis is unfavourable prognostic factor [1]. AML patients with hyperleukocytosis have demonstrated low rate of complete remission, disease free survival and overall survival as well as high rates of early mortality. In ALL, there are consistent data regarding the poor prognosis of hyperleukocytosis, so hyperleukocytosis correlated with high rate of death as compare to patients with low or normal white blood count.

In this study, all cases were shown that there were highly elevated in liver enzymes, increase in INR and level of bilirubin, decrease in albumin synthesis. Malnutrition and inflammation suppress albumin synthesis. As part of the systemic inflammatory response to the tumor, proinflammatory cytokines and growth factors are released and have a profound catabolic effect on host metabolism. Interleukin-6 produced by the tumor or surrounding cells, stimulates liver production of acute-phase reaction proteins (such as C-reactive Protein (CRP) and fibrinogen) in both the fasted and fed states. This increases the demand for certain amino acids, which if limited in the diet may be obtained from breakdown of skeletal muscle. The lower serum albumin concentration may be due to the production of cytokines such as IL-6, which modulate the production of albumin by hepatocytes. Alternatively, tumor necrosis factor may increase the permeability of the microvasculature, thus allowing an increased transcapillary passage of albumin. Various authors have reported that acute leukemia associated with abnormal liver function [11,12].

Our study showed that mean LDH levels were elevated in patients with leukemia. This in coincide with Pujari's study, who reported that high lactate dehydrogenase activity correlated with aggressive malignancy in acute leukemia [1].

Hyperglycemia was shown in some acute leukemia cases, this indicate there was defect in metabolic process due to the overcrowding of malignant up normal less function cells. Association between the presence of hyperglycemia and the development of complicated infections and death in adult patients during induction therapy for acute leukemia [13].

As regard Erythrocyte Sedimentation Rate (ESR) level there was significant increase in all acute leukemia cases. Elevated ESR this may be due to anemia, increased fibrinogen and gamma globulins as well as low albumin are common finding in infected patients [14].

Some cases have been shown disturbance in renal function as increase in uric acid levels and creatinine levels. Increase of uric acid in acute leukemia may be due to the increase Tumor Lysis Syndrome (TLS), which is an oncologic emergency that is caused by massive tumor cell lysis with the release of large amounts of potassium, phosphate, and nucleic acids into the systemic circulation [15]. Catabolism of the nucleic acids to uric acid leads to hyperuricemia; the marked increase in uric acid excretion can result in the precipitation of uric acid in the renal tubules and renal vasoconstriction, impaired autoregulation decreased renal flow, oxidation, and inflammation, resulting in acute kidney injury [16].

Also, increase of creatinine was associated with increase the rate of catabolism and blocking of renal tubules, all of this lead to accumulation of creatinine in blood stream and finally lead to elevation in creatinine level, this agreed with other studies [17,18].

In conclusion, leukemia is heterogeneous aggressive disease in which different clinical manifestation are shown. Complete blood count and blood film smear are very necessary for early diagnosis of acute leukemia. In addition to any disturbance in liver function tests or kidney function test may be act as prognostic alarm for the presence of organ damage and become useless. Elevation of lactate dehydrogenase and erythrocyte sedimentation rate may act as diagnostic tool for malignant tumor.

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