
| RESEARCH ARTICLE

Acute Myeloid Leukemia: A Neurological Presentation of a Blood Pathology

Dr. Jasim Umeed Ali Mohammed Aziz, Accident & Emergency Medicine, Senior Resident, Salmaniya Medical Complex, Kingdom of Bahrain.

Dr. Rahul Sam Mathew, Medical Intern, Salmaniya Medical Complex, Kingdom of Bahrain

Soumya Sunil Nair, Medical Student, Royal College of Surgeons – Ireland, Kingdom of Bahrain

Dr. Ali Haider Ali, Accident & Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain

Dr. Sara Abdulla, Accident & Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain

Dr. Abdulla Ebrahim Salman, Accident & Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain

Dr. Husain Jehad Ahmed Madan, Accident & Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain

Dr. Ahmed Taher Ahmed Alqayem, Accident & Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain

Corresponding Author: Ali Haider Ali, **E-mail:** Alihaiderali97@outlook.com

| ABSTRACT

Acute Myeloid Leukemia (AML) is a rapidly progressing hematological malignancy often complicated by severe, life-threatening bleeding events, such as intracranial hemorrhage (ICH). We present a case of a 31-year-old male who presented with acute neurological deficits and was subsequently diagnosed with AML complicated by spontaneous ICH. This case highlights the challenges in the management of AML patients with concurrent hemorrhagic complications and the importance of multidisciplinary care in an intensive care setting.

| KEYWORDS

“Malignancy”, “Oncology”, “AML”, “Leukemia”, and “Neurology”

| ARTICLE INFORMATION

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1. Introduction

AML is characterized by the clonal proliferation of immature myeloid cells, leading to bone marrow failure and a subsequent reduction in the production of normal blood cells. This dysfunction increases the risk of bleeding, infections, and anemia. ICH is a common fatal complication in patients with AML, often secondary to thrombocytopenia and dysfunctional platelets. This case highlights the need for clinicians to maintain a high index of suspicion for ICH in patients presenting with new-onset AML, especially those with neurological symptoms.

2. Case Report

A 31-year-old Indian male with no significant past medical history presented to the Emergency Room with slurred speech, left-sided weakness in both upper and lower limbs, fever, and vomiting. These symptoms began abruptly in the morning around 7 a.m. His mental status was altered, exhibiting agitation and confusion, with disorientation regarding time and place. The vomiting was food content, clear, with no blood or mucus. He was accompanied by a colleague and was transferred from a private hospital. The patient denied any chronic illnesses, smoking habits, or significant trauma. Vital signs showed a heart rate of 110 beats per minute, blood pressure of 145/95 mmHg, temperature of 39 degrees Celsius, respiratory rate of 16 breaths per minute, and an oxygen saturation of 100% on room air.

Examination showed an agitated and delirious patient, unaware of time or place, with a Glasgow Coma Scale (GCS) of 14/15. No rashes, bruises, or obvious signs of trauma were noted. Power in limbs was 5/5 on the right side and 3/5 on the left

side (although examination was limited due to agitation), with no signs of meningismus, neck stiffness, or focal neurological deficits except for the left-sided weakness. The NIH Stroke Scale (NIHSS) Score was 8 (indicating moderate neurological impairment). The rest of the examination was unremarkable. Laboratory investigation can be seen in Table 1.

Table 1: Serum Laboratory Investigation on Presentations.

Investigation	Value	Interpretation
White Blood Cell count (WBC)	6.02 x 10 ³ /μL	Normal
Hemoglobin (Hb)	10.6 g/dL	Low (Anemic)
Platelets	20 x 10 ³ /μL	Low (Severe Thrombocytopenia)
Manual Blast Cells	97%	Suggestive of Acute Leukemia
Reticulocytes	1.2%	-
Potassium	2.2 mmol/L	Low (Hypokalemia)
Calcium	2.48 mmol/L	Low (Hypocalcemia)

Furthermore, immunology & Serology showed the following due to the high blast cell noted, this included:

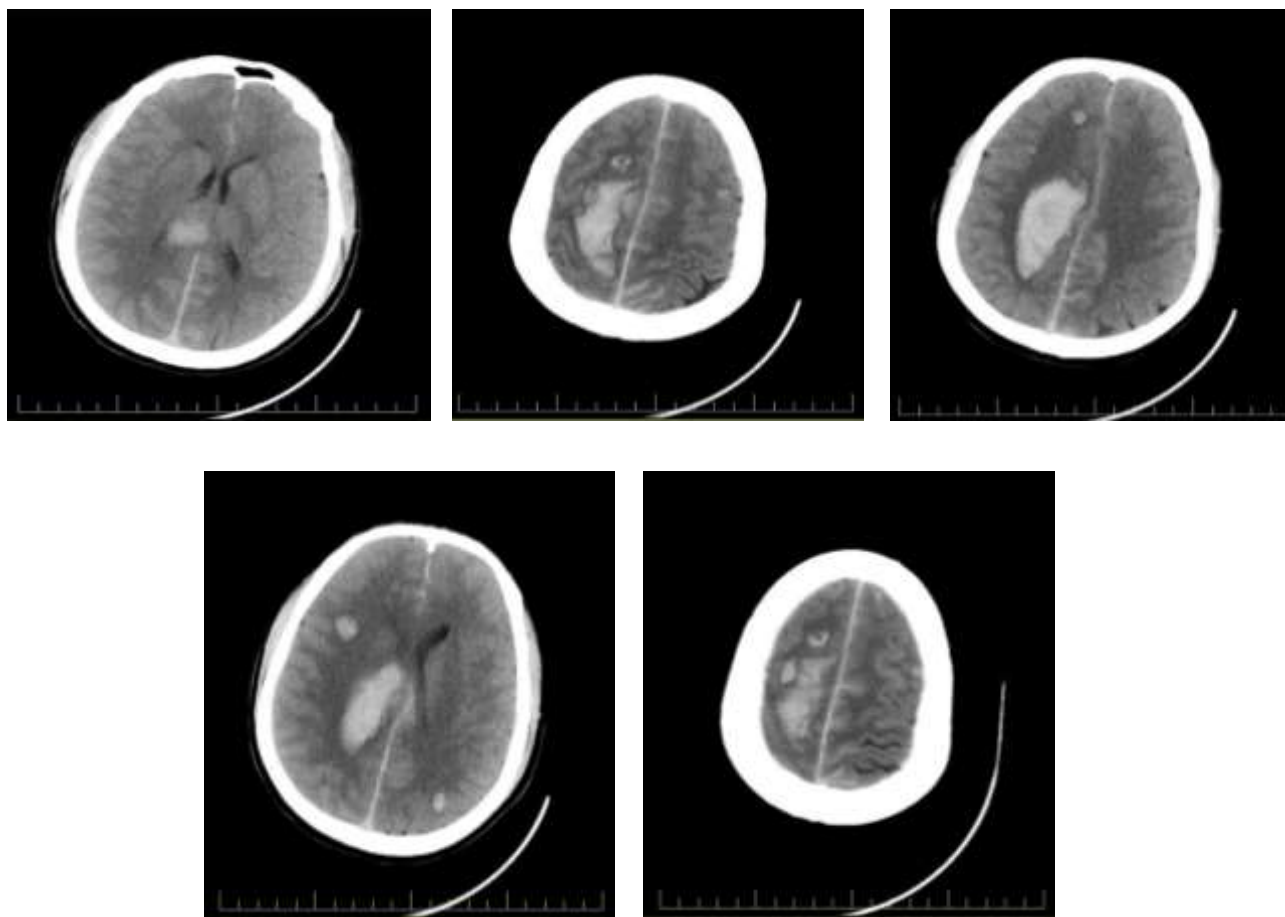
- CD33: 30.6%
- HLA-DR: 81.7%
- CD117: 0.1%
- CD19: 81.4%Flow Cytometry Immunophenotyping
- On dim CD45: 92% blast cells, brightly expressing HLA-DR, CD-19, CD-38, and dim CD33, consistent with acute leukemia.

Moreover, a computed tomography (CT) of the brain was done and showed the following:

- Right cerebral hemorrhages with multifocal areas of bleeding.
- Suggestive of acute intracranial hemorrhage in the frontal/parietal regions.

Which led to an angiogram and venogram to be done and revealed two foci of contrast blush within the right posterior frontal/parietal hematoma, with no evidence of a pseudoaneurysm or vascular malformation. There was poor filling of the venous circulation, likely due to raised intracranial pressure from the hemorrhage. The CT images are found in Image 1. Neurology and neurosurgery teams were consulted, and further management included starting labetalol infusion (max 300mg/24hrs) and codiovan 80mg OD to manage blood pressure, keeping it below 140/85 mmHg. Due to the patient's rapid deterioration and poor prognosis, the case was referred to palliative care on the third day of ICU admission. The palliative care team recommended reducing intravenous fluids to 20 mL/hour and discontinuing all oral medications. In the event of further bleeding or a drop in platelet count below 20, platelet transfusions were advised. Dexamethasone was not initiated due to the unfavorable risk-to-benefit ratio. On the fourth day, the patient's condition rapidly worsened, with a sudden drop in blood pressure to 30/23 mmHg, heart rate of 38 bpm, and oxygen saturation of 34%. Despite 20 Minutes of high-quality CPR according to ACLS protocol, the patient did not respond and was declared deceased.

Image 1: Images from the CT Brain



3. Discussion

Acute myeloid leukemia is an extremely aggressive hematologic malignancy resulting from clonal immature myeloid cell proliferation in the bone marrow and disrupts normal hematopoiesis, leading to lethal complications like anemia, infections, and thrombocytopenia, all of which predispose AML patients to very severe life-threatening conditions. This case emphasizes the importance of considering intracranial hemorrhage (ICH), one of the leading causes of morbidity and mortality is complications in patients with AML presenting with neurological deficits (2). This complexity in management is illustrated by the case of a 31-year-old male patient admitted with neurological deficits due to ICH. Symptoms noticed included slurring of speech, left-sided weakness, fever, and vomiting. A CT scan showed multifocal hemorrhages in the brain (3). ICH needs to be identified rapidly as a potential complication of AML patients, particularly those with severe thrombocytopenia and platelet dysfunction, since it is induced by thrombocytopenia. An event that happens within a few hours would require a multiprofessional approach to managing the patient. Management of patients with acute myeloid leukaemia with spontaneous intracranial hemorrhage primarily involves dealing with the challenges of leukemia and hemorrhagic complications (1,4). Initial management included platelet transfusion to correct the extreme severity of thrombocytopenia; hydroxyurea to control leukocytosis, and allopurinol to prevent tumor lysis syndrome. Blood pressure control, most critical to ICH management, was achieved using antihypertensives like labetalol. Despite all these interventions, the likelihood of survival remains very slim (4). This indication highlights the present limitations of therapeutic options while calling for closer multidisciplinary involvement in the management of the highly complicated pathophysiology of ICH associated with AML by hematology, neurology, and critical care.

The prognosis for AML patients with complications like ICH continues to be poor due to the aggressive nature of the disease and limited treatment options available. After assessing the prospects, the doctors have concerned themselves with palliation for the patient's full comfort hereafter, including the management of his symptoms, in terms of moderation of intravenous fluid intake and withdrawing unnecessary medications, ensuring dignity and relieve themselves of symptoms in the dying days of life. The goal that has come out clearly in the strategy is that palliative care should be fully integrated into the management of these patients with AML who have the least possibility of recovery. Clear communication with the family and clinical teams is essential to fulfilling the patient's preferences and needs for end-of-life care (5).

It is important to state that early detection and treatment are mandated for this group of patients because they may present at a critical time when they prove life-threatening, such as with an ICH. Supportive care, while the backbone of the treatment, cannot fulfill the urgent need for innovative therapies designed to target processes underlying this disease and its sequelae. Advances in targeted therapies such as FLT3 inhibitors and hypomethylating agents hold good promise for better outcomes. In addition, risk stratification tools may identify patients at greater risk for complications such as ICH and thus facilitate preventive actions such as close monitoring and early intervention.

Ultimately, this case brings out the need for a holistic approach in the management of AML, which entails the aggressive treatment of leukemia, effective management of the complications, and then palliative care in the last stages of the disease. Multidisciplinary collaboration and sustained research efforts are highly needed to combat the enormity of challenges that have constantly presented themselves by this devastating malignancy. More studies must be carried out on the complex interplay of this leukemia with its complications, like ICH, in conjunction with innovation.

4. Conclusion

AML remains a highly aggressive hematologic malignancy, and its complications, particularly spontaneous intracranial hemorrhage, are often fatal. Early detection of ICH and its management in the context of AML requires a multidisciplinary approach involving hematology, neurology, and intensive care specialists. Palliative care plays a crucial role in providing supportive care and ensuring patient comfort in the terminal stages of the disease.

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