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# Management of Acute Myeloblastic Leukaemia (AML) Treated with Intensive Chemotherapy: Experience in a Single Centre

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## Abstract

**Introduction:** Acute myeloblastic leukaemia (AML) is a haematological malignancy with a poor prognosis, despite significant therapeutic progress. This study presents the results of AML management in Mali according to GFAOP recommendations. **Methodology:** This was a retrospective, cross-sectional study. It included patients aged 0 - 15 years treated in the paediatric oncology unit for AML and followed up between January 2016 and December 2020. **Results:** During the study period, 85 cases of acute leukaemia were diagnosed in the paediatric oncology unit (including 51 cases of ALL), of which 34 cases of AML were included in this study. The majority were boys (59%). The mean age was 8 years, with extremes of 18 months and 15 years. The mean time to diagnosis was 68 days. In 79% of cases, patients were referred by 1st or 2nd level hospitals. Anaemia was observed in 91% of cases, an infectious syndrome in 68%, haemorrhage in 56% and a tumour syndrome in 85%. The haemogram showed hyperleukocytosis in 15% of cases, thrombocytosis in 22% and severe anaemia in 73%. Death occurred in 85% of cases, most often in the context of sepsis or haemorrhage. **Conclusion:** AML is probably underestimated in Mali and diagnosis delayed, which may be explained by patient-related factors (lack of knowledge, financial constraints) and a cumbersome referral system. These results suggest the need to implement an appropriate diagnostic and therapeutic strategy, with strong involvement of the political authorities.

## Keywords

Acute Myeloblastic Leukaemia, Children, Mali

## 1. Introduction

Acute myeloblastic leukaemia (AML) is a haematological malignancy characterised by the proliferation of abnormal myeloid precursors, called blasts, leading to bone marrow failure [1] [2]. Its origins are often poorly understood and it is thought to result from an interaction between environmental, genetic and infectious factors [3] [4]. Precise diagnosis is based on a range of factors (clinical, cytological, cytochemical, immunophenotypic, cytogenetic and molecular) [5].

Acute myeloblastic leukaemia (AML) is an uncommon disease in children, with an incidence of 7 cases per million children under the age of 15 [6]. It accounts for 15% - 20% of acute leukaemias, which make up a third of childhood cancers [7]. Little is known about its incidence in our country due to diagnostic difficulties and the absence of multicentre prospective studies [8] [9].

The prognosis of children with AML has improved considerably thanks to the development of risk classification and advances in therapeutic strategies combining intensive myelosuppressive chemotherapy and haematopoietic stem cell transplantation (HSCT) [10]. However, the survival rate in developing countries such as Mali is very low. This poor prognosis has been attributed to a number of factors, including limited access to healthcare, delayed diagnosis, co-morbidities (malnutrition, malaria), unavailability or intermittent availability of chemotherapy, antibiotics and transfusion products, lack of qualified personnel, as well as high rates of treatment drop-out, premature death and mortality due to treatment side-effects [11].

In order to improve the long-term survival rate of children with AML in poor countries, it is first necessary to evaluate and publish the results of management in these countries. The aim of this study was to describe the outcome of children treated for AML and the impact of epidemiological, clinical, biological, cytological and therapeutic characteristics on the management of these patients.

## 2. Methodology

### 2.1. Setting and Location of the Study

This study took place in the paediatric oncology unit of the paediatrics department of CHU Gabriel Touré, located in the city centre. The CHU Gabriel Touré receives patients from all over Mali.

The paediatric oncology unit (UOP) comprises ten (10) individual hospitalisation rooms, two (02) doctors' offices, one (1) office for the unit's supervisor, one (01) treatment room, one (1) slide reading room and one (01) shop.

The staff consists of four (04) paediatric oncologists, four (04) nurses trained in oncology care, a general practitioner assistant in clinical research, doctors undergoing specialisation and student trainees from the Faculty of Medicine.

### 2.2. Type and Period of Study

The present study was a retrospective, monocentric, cross-sectional survey. It

included 34 patients diagnosed with acute myeloblastic leukaemia between January 2016 and December 2020.

### 2.3. Study Population

All children aged [0 - 15] years admitted to a paediatric oncology unit with myelogram-confirmed AML.

### 2.4. Inclusion Criteria

The study was carried out on a population of patients aged 0 - 15 years with AML, defined by the WHO as the presence of more than 20% blasts on the bone marrow smear and the presence of cytoplasmic granulations.

Other methods such as immunophenotyping, karyotyping and cytochemistry were not feasible (Table A1).

### 2.5. Non-Inclusion Criteria

The following cases were not included in the study:

- Age > 15 years.
- Patients at the end of treatment coming for follow-up.
- Patients previously treated with chemotherapy.
- Incomplete or unusable files.

### 2.6. Data Entry and Analysis

For this study, we used the departmental register to obtain the total number of patients with AML. The rest of the data was collected from

- medical records
- interviews with relatives of patients included in the study.
- The questionnaire included the following elements
- epidemiological characteristics
- clinical characteristics
- biological examinations carried out: haemogram, myelogram and categorisation of patients according to the Franco-American-British (FAB) classification.
- Metabolic work-up
- Extension assessment (lumbar puncture), immunohistochemical study when the myelogram is inconclusive.
- Pre-treatment work-up (assessment of general condition and cardiac, hepatic and renal functions; infectious disease work-up (viral status).

Statistical analysis was performed using statistical processing software (SPSS version 20.0).

### 2.7. Ethical Considerations

The study was authorised and approved by the university and hospital authorities prior to data collection. Participation in the study was voluntary and data

confidentiality was strictly respected. The information recorded was transcribed anonymously. Documents relating to the interviews were coded anonymously and kept in a safe place.

### 2.8. Treatment Protocol

The treatment regimen was in accordance with the GFAOP guidelines for the treatment of AML.

1) Proposal No. 1

a) Induction treatment: (two courses)

- Induction Cure 1:
  - Doxorubicin: 60 mg/m$^2$ as a single IV injection over 30 min on D1 or Daunorubicin: 40 mg/m$^2$/D × 3 times daily IV over 30 min on D1; D2; D3.
  - Aracytine: 50 mg/m$^2$ morning and evening by subcutaneous injection for 7 days (*i.e.* 14 injections in total).
  - Allopurinol (Zyloric): 15 to 20 mg/kg/d for 10 days.
- Induction 2:

Identical to the first, to be carried out between D28 and D35 of the first course, as soon as the PNN are greater than 500/mm$^3$ and the platelets greater than 50,000/mm$^3$ (without Allopurinol).

NB: check for complete remission of the marrow between D28 and D35 of the second course of chemotherapy as soon as the PNN are greater than 1000/mm$^3$ and the platelets greater than 100,000/mm$^3$.

- Consolidation treatment: (two treatments)

In the event of complete remission, repeat the two previous courses at 4 - 5 week intervals depending on haematological recovery (without exceeding 360 mg/m$^2$ of Daunorubicin).

b) Neuromeningeal treatment:

An intrathecal injection of Aracytine during the first course of chemotherapy if haematological status permits.

- NB: In hyper leukocytic forms with more than 50,000 white blood cells/mm$^3$, M4 or M5 forms and in cases of clinical meningeal involvement and/or presence of blast cells in the CSF, 3 additional intrathecal injections are required, *i.e.* one with treatments No. 2 - 4.

2) Proposal No. 2

- Induction cures: (two cures)
  - Induction course 1:
    - Aracytine: 10 mg/m$^2$ morning and evening as a subcutaneous injection for 14 days (*i.e.* 28 injections in total).
    - Depakine (valproate de sodium) 40 mg/kg/d per os as a single dose for 14 days.
    - Allopurinol: 15 to 20 mg/kg/d for 14 days.
- Induction treatment 2:
  - Identical to the first, to be carried out between D28 and D35 of the first

course, as soon as the PNN are greater than 500/mm$^3$ and the platelets greater than 50,000/mm$^3$ (without Allopurinol).

- *NB: check bone marrow for complete remission between D*28 *and D*35 *of the second course of chemotherapy.*

- Consolidation course: (4 courses)

- In the event of complete remission, carry out 4 new courses of chemotherapy identical to the previous 2 at 4 - 5 week intervals depending on haematological recovery.

### 2.9. Operational Definitions

1) Leukaemia is classified as acute if the bone marrow contains more than 20% immature leukaemic blasts.

2) Blasts are immature, undifferentiated cells.

3) Induction refers to the start of chemotherapy treatment and includes a phase of treatment intensification.

4) Time to diagnosis is defined as the time between admission to the primary care unit and confirmation of the diagnosis.

5) Relapse is defined as the presence of more than 5% myeloblasts in the bone marrow aspirate or the reappearance of blasts in the peripheral blood, and the development of extramedullary disease after an initial complete remission.

6) Discontinuation of treatment was defined as failure to initiate or continue curative treatment for at least four consecutive weeks.

7) Lost to follow-up: any person included in the protocol who has not been heard from for 6 months.

8) Complete remission is defined as the presence of <5% myeloblasts in the bone marrow aspirate with normal haematopoietic elements, a normal blood count and the absence of clinical signs of disease.

## 3. Results

During the study period, from 2016 to 2020, 85 cases of acute leukaemia were diagnosed in the paediatric oncology unit, (including 51 cases of ALL) of which 34 cases of AML were included in this study. Patients were referred in 71% of cases. In 79% of cases, they were referred by first- or second-level health centres. Of these, 59% were from the city of Bamako. The average age at diagnosis was 8 years, with extremes of 18 months and 15 years. The 6 - 10 age group was the most represented. Of the patients recruited, 20 (59%) were male, giving a sex ratio of 1.42. Socioeconomic status was unfavourable in 68% of patients. Fathers were farmers in 35% of cases. Mothers were housewives in 82% of cases. The mothers were aged over 35 at the time of diagnosis.

The average time to consultation was 80 days, and 38% of patients consulted more than 2 months after the onset of symptoms. Clinical examination revealed an anaemic syndrome in 91% of patients, an infectious syndrome in 68%, a haemorrhagic syndrome in 56% and a tumour syndrome in 85%. Undernutri-

tion marked by muscle wasting and weight loss was observed in 44% of patients.

The mean white blood cell count was 26,255/mm$^3$. Hyperleukocytosis (>50,000) was observed in 15% of patients and leukopenia (<4000) in 18%. The mean platelet count was 113,868/mm$^3$ and thrombocytopenia (platelets < 150,000) was observed in 76% of patients. Severe anaemia (Hb ≤ 7) was observed in 73% of patients. The mean haemoglobin level was 6.18 g/dl, with extremes of 2.46 and 12.6 g/dl. The type of AML was not identified in 85% of cases, and in 12% of cases it was AML2. Malaria (presence of plasmodium) was associated in 50% of cases (Table 1).

The average duration of treatment was 79 days, with extremes of 7 and 42 days. All patients received antibiotic therapy, 79% received platelet concentrates and 85% erythrocyte concentrates (Table 2). Of the 34 patients, 29 had started treatment according to the protocol established by the Groupe Franco-Africain d'Oncologie Pédiatrique (GFAOP). The n˚1 proposal protocol was followed by 79% of our patients (see methodology).

Table 1. Socio-demographic and clinical characteristics of patients.

| | Variables | **Effective** | **%** |
|---|---|---|---|
| Residence | Bamako | **20** | **59** |
| | Ségou | 4 | 11 |
| | Kayes | 3 | 9 |
| | Sikasso | 3 | 9 |
| | Koulikoro | 3 | 9 |
| | Mopti | 1 | 3 |
| Age | 1 - 5 years old | 9 | 27 |
| | 6 - 10 years | **14** | **41** |
| | >10 years | 11 | 32 |
| Sex | Women | 14 | 41 |
| | Male | **20** | **59** |
| Signs | Undernutrition | 15 | 44 |
| | Anaemic syndrome | **31** | **91** |
| | - Pallor | 31 | 91 |
| | - dyspnoea | 4 | 12 |
| | Infectious syndrome: | **23** | **68** |
| | Haemorrhagic syndrome | **19** | **56** |
| | - Cutaneous | 6 | 18 |
| | - Mucosal | 15 | 44 |
| | Tumour syndrome | 29 | 85 |
| | - Polyadenopathy | 29 | 85 |
| | - Hepatomegaly | 10 | 29 |

Among the 17 patients who completed induction therapy, complete remission was observed in 38% and chemoresistance in 6%. The survival rate was 12% (Table 3); death occurred in 85% of cases. Haematological toxicity (side effects of treatment) was the cause of death in 79% of cases. Death occurred in hospital in 83% of cases.

## 4. Discussion

Paediatric cancer is a health problem that is underestimated by the country's political and health authorities. It is difficult to determine the true incidence of AML because of inadequate diagnostic facilities.

This study is one of the few in sub-Saharan Africa to report the clinical, biological and cytological features, as well as the results of treatment, of childhood AML. The study identified 34 new cases of AML over a 5-year period. Most patients were male, and similar results have been reported in previous studies [12]. For some authors, this male preponderance may be explained by better access to care for boys [13]. Acute myeloblastic leukaemia (AML) is a rare childhood cancer, accounting for 15% - 20% of childhood leukaemia cases. In this study, its prevalence was higher in children aged 6 to 10 years (41%), which corroborates the studies by Williams C K and Ngamai Bele Oli Carine, aged 5 to 9 years and 8

Table 2. Breakdown of support care received by patients.

| Support care for patients | **Effective** | **%** |
|---|---|---|
| Transfusion of platelet concentrate | 27 | 79 |
| Transfusion of packed red blood cells | 29 | 85 |
| Transfusion of whole blood | 3 | 9 |
| Antibiotic | 34 | 100 |
| Antifungal | 3 | 9 |
| Antimalarial | 9 | 26 |
| Furosemide | 4 | 12 |
| Antipyretic/analgesic | 22 | 65 |
| Infusion | 23 | 68 |
| Deworming | 3 | 9 |
| Therapeutic milk | 2 | 6 |
| Allopurinol | 19 | 56 |

Table 3. Overall results after the end of induction.

| Results | **Effective (n = 17)** | **%** |
|---|---|---|
| Complete remission | 13 | 38 |
| Failed induction (refractory disease) | 2 | 6 |
| Loss to follow-up | 1 | 3 |
| Treatment interrupted | 1 | 3 |

to 10 years respectively [12] [14]. According to other sources, the highest incidence is seen in patients aged zero to four years, with a higher incidence in neonates, reflecting the congenital nature of AML [13].

Patients were generally referred by other hospitals. Most of them had already received treatment based on blood transfusions and antimicrobials. Many cancer patients resort to traditional "unconventional" care. In this study, the average time to diagnosis was 68 days. Only one patient was diagnosed with AML within one month of the onset of symptoms. This long delay before diagnosis was multifactorial, linked to the patient and the doctor. Training primary care providers in acute leukaemia could reduce the delay in diagnosis. A delay in diagnosis would have a clear impact on early mortality. The majority of patients arrived very seriously ill, with signs of sepsis, severe anaemia or haemorrhage. Co-morbidities were frequent, most often infectious pathologies or deficiency malnutrition. These vital emergencies had consumed their meagre resources.

In the present study, hyperleukocytosis was found in 15% of cases. According to the literature, a very high WBC count at diagnosis is a poor prognostic factor and is associated with a lower rate of complete remission [15] [16]. Similarly, the need for platelet transfusion was general, with only 22% of patients having a platelet count greater than 150,000/mm$^3$ on admission, but apheresis platelets were not available. Not surprisingly, the majority (85%) of deaths occurred in the context of haemorrhage and severe sepsis. This is an urgent problem that needs to be resolved to ensure better therapeutic outcomes.

This study, like the majority of previously published studies, showed that the most common morphological subtype was AML2 [17]. Cytogenetic abnormalities, somatic mutations and response to induction therapy are considered important information for risk classification and appropriate treatment [17]. The therapeutic approach used in this study was systemic multidrug therapy. In accordance with the guidelines for the treatment of AML (GFAOP study), treatment is generally divided into two phases: induction (to induce complete remission) and consolidation/intensification after remission (to reduce the risk of relapse). In this study, the patients were at an advanced stage of the disease. Early mortality (within 28 days of diagnosis) was high. The result was lower than that reported in the Western literature. In a Tanzanian study, all children died within one year of diagnosis [14] [18].

The majority of deaths were due to sepsis or haemorrhage linked to intensive chemotherapy and complications of the disease. Visits were irregular, making it difficult to monitor patients. This problem could be solved to some extent by improving parents' confidence in treatment protocols and their level of understanding of the disease. Given the high cost of biological and radiological tests and the difficulties observed in monitoring patients, financial or infrastructural support from the state or NGOs would be necessary.

The literature tells us that reducing drop-out rates is the best way to achieve better outcomes in the treatment of childhood AML. Drop-out rates can be suc-

cessfully reduced by improving adherence to treatment and adopting a comprehensive global strategy including financial support, education and free accommodation close to the hospital for patients or families in need [18]. This advanced strategy could significantly reduce the incidence of treatment discontinuation and attrition. Further research is needed on how to effectively treat paediatric cancers in resource-limited settings. Descriptive studies on the causes of treatment failure in the context of poverty should be encouraged and supported.

The main limitations of this study are its retrospective design and relatively small sample size. Data collection from medical records was often hampered by the poor condition of the records and the lack of information on certain clinical, cytological and therapeutic data.

## 5. Conclusions

This study revealed major shortcomings in the management of paediatric AML. Diagnosis was most often delayed, due to problems associated with financial difficulties, the lack of a multidisciplinary approach and the absence of adequate infrastructure and equipment for diagnosis, follow-up and treatment. Delays in consultation and diagnosis led to the discovery of advanced forms of the disease.

Strategies to improve survival must be put in place, in particular by improving supportive care, optimising the treatment protocol and reducing the drop-out rate and delay in diagnosis through early initiation of treatment.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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## Appendix

**Table A1.** Some clinical and biological characteristics of AML at the pediatric oncology unit of Bamako.

| Patients | Age (M = month, Y= year) | Sex | Duration of symptoms (weeks) | FAB classification (not classified = NC) | Number of WB cells/mm$^3$ | % of blasts | Number of platelets/ mm$^3$ | Tissue invasion | Transfusion of blood products | Quality of chemotherapy (not done, adequate, insufficient) | Survival in days |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. | 06A | M | 04 | NC | 3700 | | 44,000 | S | | adequate | 135 |
| 2. | 10A | F | 04 | NC | 8700 | | 109,000 | A | | Not done | 5 |
| 3. | 12A | F | 08 | NC | 4150 | | 84,000 | A H | CG, CP | adequate | 93 |
| 4. | 16M | F | 16 | NC | 39,500 | | 85,000 | S H | CG, CP | inadequate | 30 |
| 5. | 09A | M | 04 | NC | 10,500 | | 37,000 | H A | CG, CP | Not done | 10 |
| 6. | 26M | M | 16 | NC | 63,500 | | 257,000 | A S | CG, ST, CP | adequate | 16 |
| 7. | 14A | M | 04 | NC | 17,000 | | 453,000 | A | CG, CP | adequate | 38 |
| 8. | 12A | M | 04 | LAM2 | 18,000 | 56 | 28,000 | A | CG, CP, ST | adequate | 97 |
| 9. | 20M | M | 36 | NC | 5300 | | 205,000 | A | CG, CP | adequate | 180 |
| 10 | 08A | M | 08 | NC | 17,200 | 94 | 43,000 | A | CG, CP | inadequate | 69 |
| 11 | 10A | F | 08 | LAM2 | 2950 | | 33,000 | A | CG, CP | inadequate | 19 |
| 12 | 03A | M | 12 | LAM2 | 139,300 | 100 | 32,000 | A S H | CG, CP | adequate | 217 |
| 13 | 12A | M | 04 | LAM2 | 18,000 | 56 | 28,000 | A | CG, CP | adequate | 97 |
| 14 | 12A | M | 08 | NC | 4900 | | 300,000 | A | CG | adequate | 26 |
| 15 | 06A | M | 08 | NC | 60,700 | | 9000 | A | CG, CP | inadequate | 19 |
| 16 | 09A | F | 24 | NC | 106,600 | 49 | 57,000 | A H | CG, CP | adequate | 260 |
| 17 | 13A | F | 04 | NC | 94,200 | | 65,000 | H | CG, CP | adequate | 7 |
| 18 | 10A | M | 04 | LAM4 | 48,000 | 55 | 118,000 | A H | CG, CP | adequate | 47 |
| 19 | 06A | F | 12 | NC | 4600 | | 324,000 | A S H | CG | adequate | 480 |
| 20 | 07A | M | 02 | NC | 43,200 | | 31,200 | A S H | CP, CG, ST | adequate | 33 |
| 21 | 05A | M | 12 | NC | 15,050 | | 134,000 | A | CG, CP | adequate | 146 |
| 22 | 08A | M | 04 | NC | 6600 | | 2000 | | CP | adequate | 11 |
| 23. | 14A | F | 04 | NC | 139,300 | | 598,000 | A S H | CG | adequate | 150 |
| 24 | 8A | F | 12 | NC | 6900 | | 12,000 | A | CP, CG | adequate | 14 |
| 25 | 7A | M | 08 | NC | 47,200 | | 11,000 | A, H, S | CP, CG | adequate | 10 |
| 26 | 3A | M | 12 | NC | 5620 | | 45,000 | | CP, CG | Not done | 11 |
| 27 | 11A | F | 16 | NC | 3200 | | 264,000 | | CP, CG | adequate | 02 |
| 28 | 08A | M | 08 | NC | 7800 | | 277,000 | S, A | CP, CG | Not done | 02 |
| 29 | 11A | F | 4 | NC | 13,900 | | 15,000 | S, A | CP, CG | Not done | 20 |
| 30 | 3A | M | 3 | NC | 47,100 | | 17,000 | A | CP, CG | adequate | 32 |
| 31 | 12A | M | 12 | NC | 23,270 | | 9300 | A | CG | adequate | 7 |
| 32 | 5A | M | 20 | NC | 2020 | | 61,000 | A | | adequate | 21 |
| 33 | 13A | F | 8 | NC | 3800 | | 258,000 | A | CP, CG | adequate | 33 |
| 34 | 12A | M | 16 | NC | 2400 | 45 | 73,000 | | CP, CG | adequate | 10 |

PS: Blood products; CG: Red blood cell concentrate; CP: Platelet concentrate; A: Adenopathy; S: Splenomegaly; H: Hepatomegaly.