



A case of platelet refractoriness in a patient with acute myelogenous leukaemia and paraplegia: management in a low resource setting

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ABSTRACT:

Background: Acute myelogenous leukaemia is a type of acute leukaemia more commonly seen in adults than in children and usually presents with features of anaemia, neutropoenia and thrombocytopenia (pancytopenia). Red cell transfusions and the use of granulocyte colony stimulating factor usually correct the anaemia and neutropoenia respectively while platelet concentrates are required for correction of thrombocytopenia. However, some patients develop platelet refractoriness where they fail to achieve expected increment in platelet count following platelet transfusions which may be fatal because severe thrombocytopenia may lead to bleeding into vital organs including the brain. The aim of this report is to document the management of platelet refractoriness in a patient with acute myelogenous leukaemia and paraplegia, with correction of thrombocytopenia in a low resource setting.

Methods: Data was obtained from the case notes of a 14 year old male with acute myelogenous leukaemia and paraplegia who developed platelet refractoriness. A review of literature was done by searching on Google and PubMed.

Results: A 14 year old male who presented with pancytopenia and paraplegia was diagnosed with acute myelogenous leukaemia. He had multiple transfusions and developed platelet refractoriness. Despite severe thrombocytopenia and platelet refractoriness, he was commenced on chemotherapy and achieved remission.

Conclusion: Paraplegia is an uncommon presentation of central nervous system involvement in acute myelogenous leukaemia. Platelet refractoriness is a feared complication occurring in haematological malignancies. The acute myelogenous leukaemia complicated by platelet refractoriness was treated successfully in a low resource setting.

Key words: Platelet refractoriness; Acute Myelogenous Leukaemia; Acute myeloid leukaemia; paraplegia; AML; Nigeria.

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Introduction:

The acute leukaemias are clonal haematological malignancies of myeloid or lymphoid origin. In both cases they are characterized by the proliferation of immature haemopoietic cells known as blast cells which accumulate in the bone marrow and circulate in the peripheral blood. This



accumulation may also occur in other organs such as the spleen, liver, testes, skin or central nervous system.

In 2008, the revised World Health Organization diagnostic criteria for acute leukaemias required the demonstration of $\geq 20\%$ blast cells in the peripheral blood or bone marrow.¹ Acute lymphoblastic leukaemia (ALL) is the commonest malignancy in children^{2,3} while acute myelogenous leukaemia (AML) occurs more commonly in adults.^{4,5} The French American British classification of AML uses morphology to classify AML into 8 subtypes, from M0 to M7. The M0 subtype is undifferentiated, M1 and M2 are myeloblasts without or with maturation respectively. Acute promyelocytic leukaemia is the M3 subtype while M4 and M5 show monocytic differentiation. M6 is erythroleukaemia while M7 is acute megakaryoblastic leukaemia.⁶

Extramedullary leukaemia occurs when leukaemic cells are found in other organs outside the bone marrow. Central nervous system (CNS) involvement in acute leukaemias is more common in ALL, however it may occur in AML.⁷ Patients with AML may have symptoms and signs of CNS disease at presentation, or may develop it later while on chemotherapy or even after chemotherapy. CNS disease is diagnosed when blasts are present in the cerebrospinal fluid, if there is cranial nerve palsy or meningeal involvement, or the presence of a non-haemorrhagic CNS chloroma (solid mass of leukaemic cells) on CT scan. Central nervous system disease in AML although unfavourable (because it is difficult to completely eradicate it) does not affect survival in AML.^{8,9}

Patients with AML commonly present with clinical and laboratory features of anaemia, neutropenia and thrombocytopenia (pancytopenia). Haemoglobin levels generally vary for different age groups and sex but normal values range from 11.5 – 15.5g/dL in pre-teen and early teen years.¹⁰ The normal white cell count for this age group is from 5-13 X 10⁹/L with absolute neutrophil counts ranging from 2-8 X10⁹/L.¹⁰ Normal platelet counts in Africans have been reported to be 10 -20% lower (95 – 322 X 10⁹/L) compared to Caucasian values (150 – 445 X 10⁹/L)^{10, 11, 12, 13} Cytopenias occur when a patient presents with full blood count parameters below expected values for their age group, sex or ethnicity.

Platelets are the smallest sized of all the blood cells and have an average lifespan of 7-10 days. They are anucleated cells produced by megakaryocytes in the bone marrow. Normally, about a third of platelets are stored in the spleen. Platelets are essential for haemostasis and achieve this by forming a mechanical plug when there is vascular injury. In the cell based theory of coagulation, platelets provide a rich phospholipid framework for coagulation to occur, they also release some stored coagulation factors when activated.¹⁴ Thrombocytopenia is a reduction in the platelet count



below the normal reference value. Since platelets are required for haemostasis, thrombocytopenia results in bleeding which may be fatal especially at counts $<10 \times 10^9/L$ because it can result in intracranial haemorrhage. Thrombocytopenia in AML is both due to reduced platelet production (due to bone marrow infiltration by the blast cells) and shortened lifespan of the platelets.¹⁵ Platelets also have impaired function in AML patients due to reduced platelet activation.¹⁶ About 20-32% of patients with AML and thrombocytopenia have haemorrhage.¹⁷

The cytopenias in AML require correction; red cell transfusions and the use of granulocyte colony stimulating factor usually correct the anaemia and neutropenia respectively while platelet concentrates are required for correction of thrombocytopenia. Platelet concentrates may be harvested from a single donor (apheresis platelets) or pooled from about 4 – 6 donors' whole blood by the use of a cold centrifuge. One unit of apheresis platelets is estimated to increase the platelet count by $30 \times 10^9/L$ – $50 \times 10^9/L$, while a single donor unit (before pooling) can increase the platelet count by $5 \times 10^9/L$.^{18, 19} However, about 30% of patients develop platelet refractoriness (PR) where they fail to achieve expected increment in platelet count following platelet transfusions.²⁰ This poses a great challenge in managing patients with acute myelogenous leukaemia as severe thrombocytopenia may lead to bleeding into vital organs including the brain. Causes of PR may be immune or non-immune mediated. The immune mediated causes may be due to development of alloantibodies against the ABO blood group antigens, human leukocyte antigens (HLA) and or human platelet antigen (HPA) systems on the transfused donor platelets which destroys them. Autoimmune causes such as immune thrombocytopenia or thrombotic thrombocytopenic purpura may also cause PR. Up to 80% of PR arise from non-immune causes which include fever, sepsis, splenomegaly, hepatomegaly, disseminated intravascular coagulopathy (DIC), active bleeding and drugs such as heparin, quinidine, penicillin or aspirin.^{18, 20, 21}

There are several ways of assessing response to transfused platelets. The corrected count increment (CCI) is cumbersome to calculate and uses the formula: – $[(\text{post platelet count} - \text{pre platelet count}) \times \text{Body Surface Area}] / \text{platelet dose}$. With the CCI, a platelet increment of $<7.5 \times 10^9/L$ is regarded as suboptimal. The percentage of platelet recovery (PPR) is the second method which uses the formula: – $(\text{number of platelets transfused} \times 0.67 \times 10E3) / \text{blood volume in mL}$, where 0.67 is the factor for splenic pooling. A PPR of $<30\%$ is suboptimal. The third method is the post-transfusion platelet increment and is the easiest and most practical to calculate. It is calculated simply by subtracting the pre-transfusion platelet count from the post-transfusion platelet count. An increment of $<11 \times 10^9/L$ is regarded as suboptimal and if it occurs on 2 consecutive occasions



then there is platelet refractoriness. For the PPR and post-transfusion platelet increment, platelet count is assessed at one hour after transfusion of platelets but for the CCI, it is assessed both at one hour and twenty-four hours post transfusion.^{15, 22}

The risk for developing PR is increased in multiply transfused patients, the use of non-leukoreduced blood and blood components, pregnancy, in the transfusion of ABO-mismatched platelet concentrates or stored platelets of more than three days.¹⁵ Leukoreduction involves removal of white cells from blood components, this reduces the risk of developing PR or the need to use HLA matched platelets.^{23, 24, 25} Treatment of PR involves treating the underlying cause in cases of non-immune PR, or the use of HLA or HPA matched platelets for transfusions. In immune mediated cases, the use of intravenous immunoglobulins, Rituximab and Cyclosporine A have all been proposed.¹⁸

In a low resource centre, there may not be facilities for blood component preparation. Presently, the University of Port Harcourt Teaching Hospital in Rivers, Nigeria is one of the handful of centres that can provide this service in the country. This is the first case of PR recorded in our centre. The aim of this report is to document the PR which occurred in a patient with AML and CNS disease that was successfully treated in a low resource setting.

Case Report

M.T. was a 14 year old male referred to the University of Port Harcourt Teaching Hospital in Rivers, Nigeria with recurrent fever, bone pains and tiredness of three months duration. He also complained of a two week history of pallor of his palms and soles; and inability to walk for a few days. He had received two units of blood at a peripheral centre because he had a haematocrit of 12%. On examination at presentation at our centre he was severely pale, anicteric, febrile with a temperature of 40.5°C. There was no peripheral lymphadenopathy and no pedal oedema. Examination of the abdomen revealed hepatomegaly of 7cm below the costal margin. The CNS examination showed he was oriented in time, place and person. The power was five in the upper limbs but zero in both lower limbs.

Full blood count done revealed pancytopenia; there was severe anaemia with a haemoglobin concentration of 7.3g/dl. Although the white blood cell (WBC) count was normal ($8 \times 10^9/L$), the differential WBC count was abnormal and showed presence of 95% blast cells with severe neutropenia; the absolute neutrophil count was $0.32 \times 10^9/L$. The platelet count was also severely reduced at $5 \times 10^9/L$ (*Table 1*). The peripheral blood film showed blasts that were moderately large in size with lacy or loose nuclear chromatin. The nuclei of the blasts generally had about 1-3



nucleoli. Their cytoplasm were basophilic, several of them had granules and a few of them possessed Auer rods, in keeping with myeloid blast cells- myeloblasts (*Fig. 1*). The patient's bone marrow aspirate showed a hypercellular pattern with depressed erythropoiesis and megakaryopoiesis. There was proliferation of myeloid blasts with the bone marrow differential count giving a value of 91% myeloblasts. The blasts were large sized cells with deeply basophilic cytoplasm containing Auer rods and granules in some of them. The blasts had heterogeneous nuclear shapes with nuclear indentations in some of them. Nuclear chromatin pattern of the myeloblasts was loose in nature, with prominent multiple nucleoli (*Fig. 2*). There were no foreign cells in the bone marrow and there were normal iron stores. Other tests done included uric acid which was markedly elevated at 721 μ mol/L; the urinalysis, renal and liver functions tests were essentially normal. Brain CT-scan requested for was not done by the patient. Lumbar puncture was withheld until the time of intrathecal chemotherapy.

With the above clinical and laboratory features (including a blast count of 95% in the peripheral blood and 91% in the bone marrow), a diagnosis of AML of the M1 subtype with CNS involvement was made. He was optimized for chemotherapy with fresh whole blood (FWB) and four units of freshly prepared ABO-blood-group-specific apheresis platelets (AP) harvested from donors with a minimum platelet count of 250 X 10⁹/L. He also had granulocyte- colony stimulating factor (G-CSF) given subcutaneously at the dose of 300 μ g per day. The anaemia was corrected following the transfusions however, the patient developed platelet refractoriness (PR) with lack of increment in the post transfusion platelet 1 hour and 24 hours after each platelet transfusion. By the 21st day he had received a total of 9 units of FWB and 6 units of AP but his platelet count was now 02 X 10⁹/L. He received prophylactic antimicrobials (Ciprofloxacin, Fluconazole and Acyclovir) for four weeks with reverse barrier nursing care for neutropoemia. The fever subsided a few days after admission. Following transfusion of AP, the patient had platelet counts done routinely within an hour of transfusion. It was noticed that he failed to achieve platelet increments >11 X 10⁹/L after AP transfusions, therefore patient was diagnosed as having PR (*Table 2*). A decision to commence chemotherapy despite the PR was taken due to increasing blast count in the peripheral blood. Chemotherapy with Cyclophosphamide, Vincristine, Cytosine Arabinoside and Prednisolone (COAP) was commenced at 75% of the calculated dose (due to severe thrombocytopenia), while withholding intrathecal chemotherapy (ITC). He had further daily alternating transfusions of FWB or AP. By day 12 of the first cycle of chemotherapy, his platelet count started rising and at Day 21 of chemotherapy his platelet count was 207 X 10⁹/L (*Table 1, Fig 3*). He achieved haematological



remission during the first cycle of 28 days (from the 21st day of the cycle) with regression of the hepatomegaly, a normal full blood count and clearing of blast cells from the peripheral blood film (*Fig. 4*). The blast count was 4% in the bone marrow aspirate done on Day 28 of the first cycle. From the second cycle, chemotherapy dose was at 100%, platelet counts ranged from 109 - 397 $\times 10^9/L$ at intervals and ITC commenced. During the first lumbar puncture for the ITC, cerebrospinal fluid (CSF) sample was collected. Blast cells were absent and there were <5 WBC on CSF.

All through his hospital stay, the patient had no bleeding episode. Due to multiple red cell transfusions, he was commenced on oral iron chelators (Deferasirox) given daily. He started rehabilitation for paraplegia and power in the lower limbs improved. His clinical and laboratory parameters improved remarkably; however his parents had to relocate to another city so he was referred after the fourth cycle of chemotherapy.

The child was apparently stable and on regular follow-up at the referral centre until he had a relapse 15 months after diagnosis with severe anaemia, he died soon after. The cause of death was not known.

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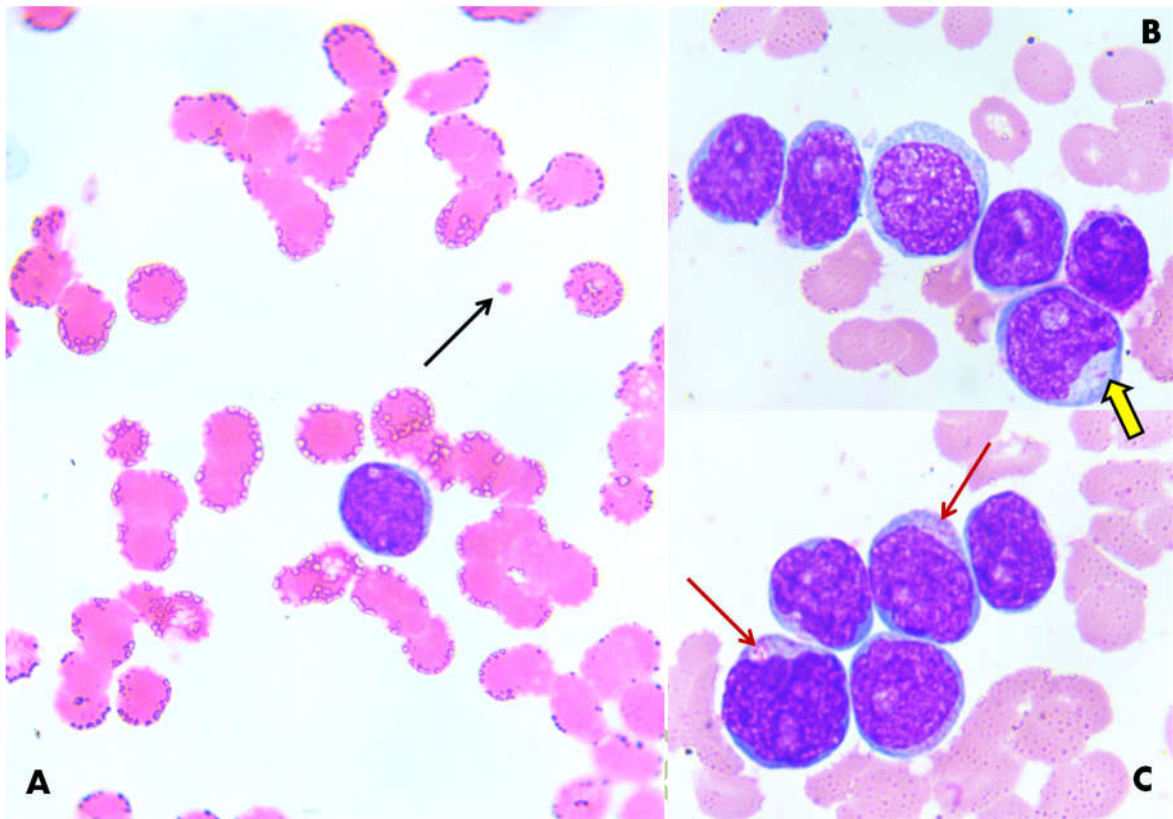


Fig. 1- Peripheral Blood Film, High Power View X100 objective: showing myeloblasts with prominent nucleoli and thrombocytopenia in the peripheral blood. **(A)** There is a single myeloblast with severe thrombocytopenia on film- only one platelet is seen (black arrow). **(B)** Several large myeloblasts, one with an Auer rod (yellow arrow). **(C)** Red arrows point to granules in the myeloblasts.

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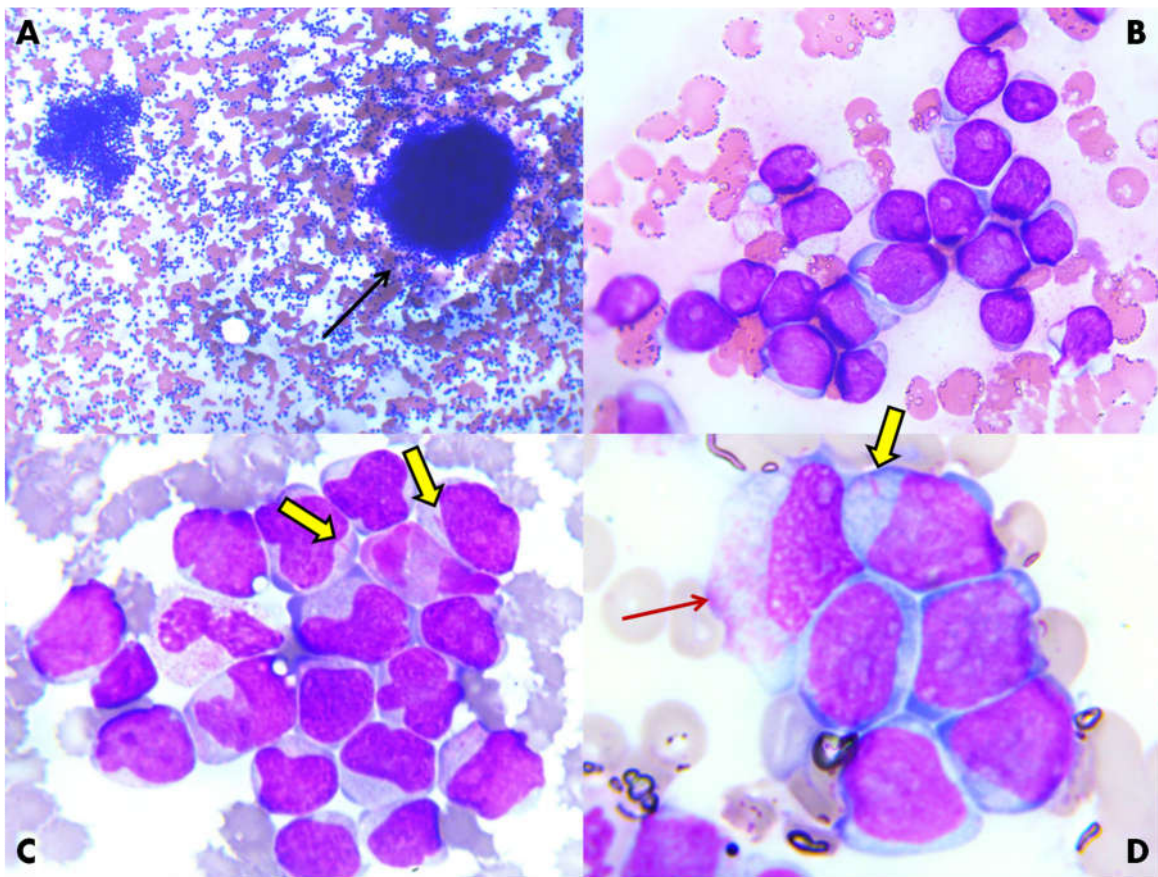


Fig. 2- Bone Marrow Aspirate, showing marrow infiltration by myeloblasts. **(A)** Low power view at X10 objective: markedly hypercellular marrow made up of mononuclear cells. **(B)** High power view x 100 objective: Several large myeloblasts in a cluster, some with granules. They generally have nuclei with prominent nucleoli. **(C) & (D)** Yellow arrows point to Auer rods in cytoplasm of the myeloblasts. A few blasts also have prominent granulation (red arrow in picture **D**).

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Table 1: Post-transfusion haemograms prior to and during the 1st cycle of chemotherapy

Day Post Adm.	Normal Values	Day 1	Day 2	Day 4	Day 8	Day 14	Day 15	Day 17	Day 19	Day 21	Day 23	Day 25	Day 27	Day 29	Day 31	Day 33	Day 37	Day 42
Day of Chemo.	N/A	-	-	-	-	-	-	-	-	-	Day 2 1 st Cy.	Day 4 1 st Cy.	Day 6 1 st Cy.	Day 8 1 st Cy.	Day 10 1 st Cy.	Day 12 1 st Cy.	Day 16 1 st Cy.	Day 21 1 st Cy.
Type of Transf.	N/A	None	FWB	FWB	AP	FWB	AP	AP	AP	AP	FWB	FWB	FWB	AP	AP	AP	AP	None
Hb (g/dL)	11.5 - 15.5	7.3	8.7	9.6	8.5	13	12	12	11.1	10.8	15	16.3	18.2	17.7	16	14.4	15	13.2
WBC (x10 ⁹ /L)	5 - 13	8.0	9.6	9.8	8.0	3.8	6.2	12.8	13.0	13.5	2.0	2.0	1.9	2.0	1.9	1.9	0.9	5.9
ANC (x10 ⁹ /L)	2 - 8	0.32	0.3	0.42	0.4	0.22	1.01	1.1	0.89	0.8	0.22	0.19	0.34	0.45	0.45	0.43	0.78	1.71
Blast Count (%)	0	73	79	65	82	88	95	96	93	97	97	81	66	51	32	26	17	0
Platelet (x10 ⁹ /L)	170 - 450	05	05	22	05	03	08	08	03	02	13	10	09	04	08	15	27	207

Transf.- Transfusion; Adm.- Admission; Chemo- Chemotherapy; N/A- Not applicable; Hb- Haemoglobin concentration; WBC- white blood cell; ANC- absolute neutrophil count; FWB- Fresh whole blood; AP- Apheresis platelet; Cy.- Cycle.

Note- Patient received only ABO blood group specific units of transfused blood.

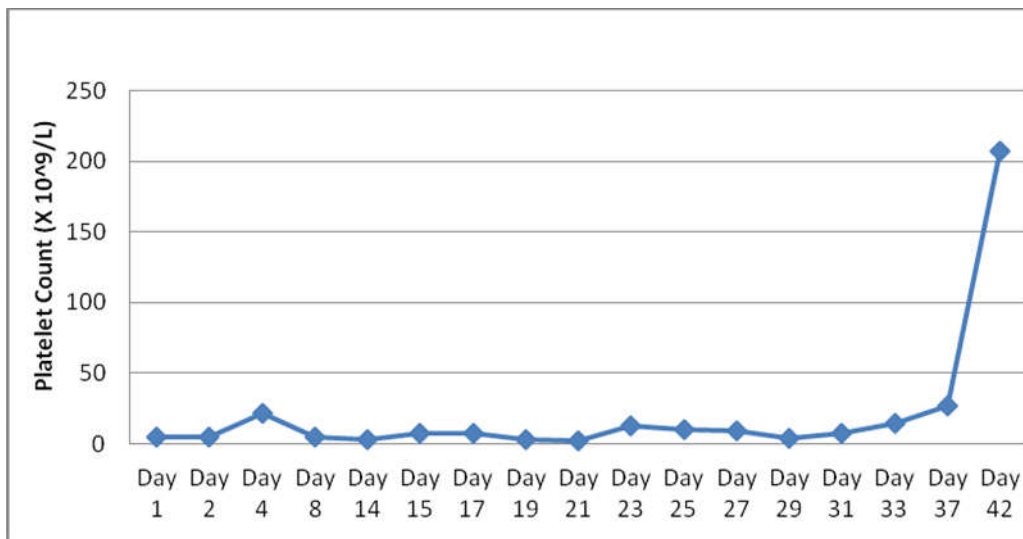


Fig. 3: Platelet count on specific days, showing a sustained rising platelet count from Day 31 of admission (10th day of the first cycle of chemotherapy).

Table 2: Pre & Post-Transfusion platelet counts† after transfusion of apheresis platelets

Day post Admission	Day of Chemo.	Component Transfused	Pre-Transfusion PC (X 10 ⁹ /L)	Post-Transfusion PC (X 10 ⁹ /L)	Expected Platelet Increment post AP transfusion (X 10 ⁹ /L)	Actual Platelet Increment 1 hour post AP transfusion (X 10 ⁹ /L)
8	-	AP	5	18	35 - 55	13
15	-	AP	8	16	38 - 58	8*
17	-	AP	8	15	38 - 58	7*
19	-	AP	3	8	33 - 53	5*
21	-	AP	2	10	32 - 52	8*
29	8	AP	4	10	34 - 54	6*
31	10	AP	8	19	38 - 58	11
33	12	AP	15	31	45 - 65	16
37	16	AP	27	55	57 - 77	28

†Post-transfusion platelet count was done one hour after transfusion of platelet concentrates.

*Denotes platelet increments <11 X 10⁹/L signifying platelet refractoriness

PC- platelet count, AP- apheresis platelets

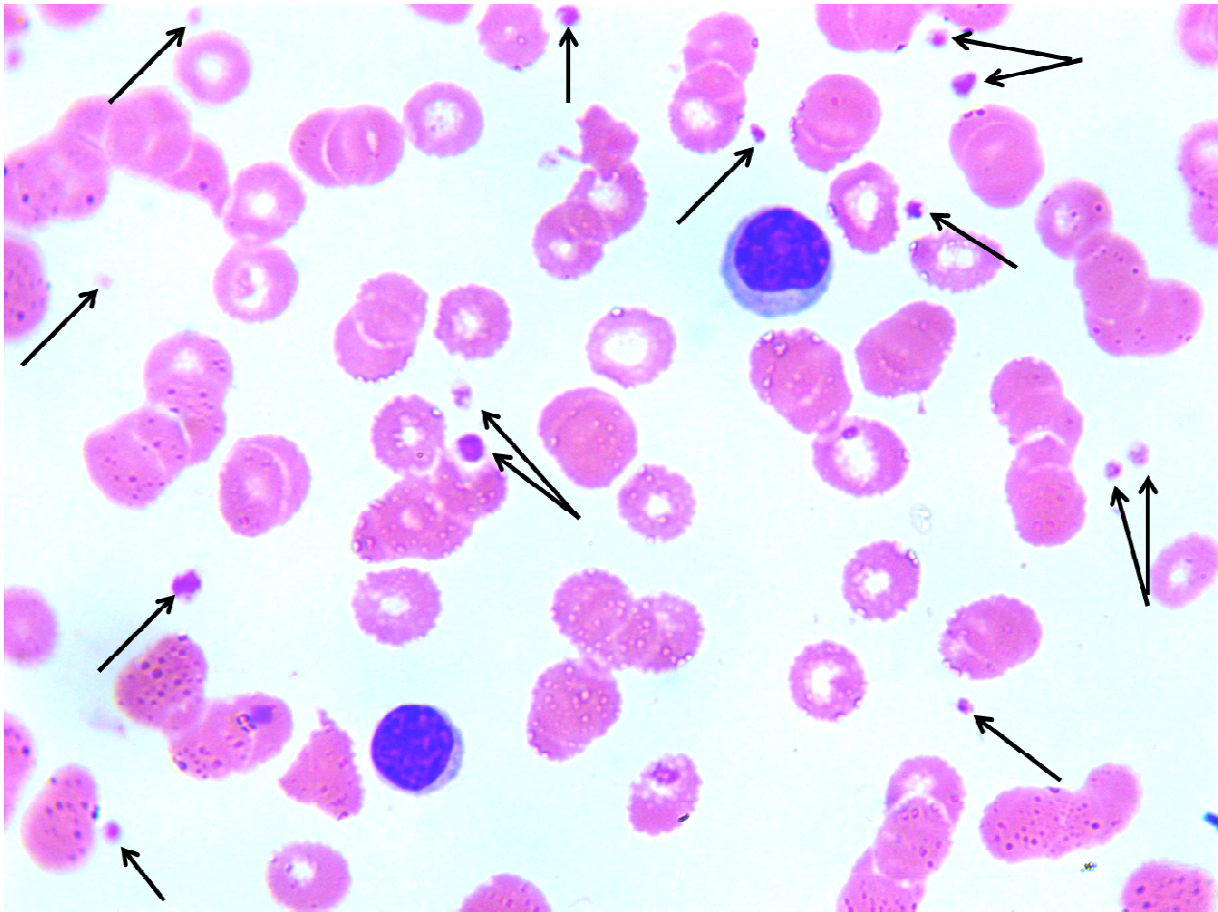


Fig 4: Peripheral blood film smear done on Day 42 post admission (21st day of the 1st cycle of chemotherapy): High power view (X100 Objective) showing 2 normal mature lymphocytes with several platelets (black arrows).

Discussion

Acute myelogenous leukaemia is a clonal haematological malignancy resulting in uncontrolled proliferation of myeloid blast cells in the bone marrow and other organs. The blast cells also circulate in the peripheral blood and almost any tissue. A diagnosis of acute leukaemia requires $\geq 20\%$ myeloblasts in the peripheral blood and/or bone marrow.^{1, 14, 15} At presentation and before remission was induced by the chemotherapy; our patient had peripheral blood blast counts persistently above 20% (73% at initial FBC and 95% on initial manual differential count of the peripheral blood film. He also had a bone marrow blast count of 91%), therefore confirming the



diagnosis of an acute leukaemia. He specifically had AML because the morphology of the blasts showed Auer rods and granules, which are consistent with a diagnosis of AML.

AML is more of an adult disease; however AML may occur in children as was the case in our patient. In both the acute myeloid and lymphoid leukaemias, there can be CNS disease. The CNS involvement occurs less frequently in AML compared to ALL and therefore patients with AML do not require prophylactic intrathecal therapy unlike those who have ALL.¹⁴ Across all age groups, AML may present with extramedullary leukaemia in 9% of cases but in paediatric AML, this increases to about 40%.⁷ CNS disease is a form of extramedullary leukaemia and in up to 29% of paediatric AML cases have CNS disease at presentation. Diagnosis of CNS leukaemia requires the presence of blasts in the CSF; presence of cranial nerve palsy or meningeal involvement; or the demonstration of a non-haemorrhagic CNS chloroma mass on CT scan. CNS disease at presentation can be graded into CNS1= <5 WBC and no blasts; CNS2= <5 WBC with blasts; CNS3= ≥5 WBC with blasts in the CSF.^{8,9} Our patient did not have blasts in CSF and the CT scan requested for was not done; however because of the paraplegia of a few days duration, he had signs of CNS disease at presentation. Some cases of CNS disease do not have blasts in the CSF (CNS1), as was seen in our patient.^{8,9} Paraplegia in AML is an uncommon finding, but when present it is usually seen in young males.²⁶ Granulocytic sarcomas or chloromas cause spinal cord compression leading to paraplegia in AML patients.^{7,26,27} These granulocytic sarcomas occur more frequently in children and young adults.^{7,26} Unfortunately our patient was unable to have a CT scan done for financial reasons but the paraplegia may have been as a result of cord compression. Also, when treatment including intrathecal therapy and physiotherapy was given, the patient responded and power improved in the lower limbs.

Patients with AML usually have cytopenias, especially anaemia and thrombocytopenia. The index case had depression of all cell lines (pancytopenia) with severe thrombocytopenia of $5 \times 10^9/L$ due to bone marrow suppression by the leukaemic blasts. Thrombocytopenia increases the risk for spontaneous haemorrhage with counts less than $50 \times 10^9/L$ and life threatening haemorrhage such as intracranial haemorrhage can occur at counts $<15 \times 10^9/L$.¹⁵ Thrombocytopenia in AML is usually treated with platelet concentrate transfusions. Platelet concentrate can be prepared by cold centrifuge or apheresis. Cold centrifuge platelets are prepared by pooling 4-5 units of blood from random donor; while AP which are obtained from a single donor, have a higher platelet yield and also have the added advantage of exposing the recipient to only one donor's platelet antigens at a



time thereby reducing the risk for PR. For these reasons, AP are preferred to cold centrifuge platelets.¹⁸ Other factors shown to improve response to platelet transfusions include transfusion of ABO-matched platelets and the use of fresh platelets which have been stored for less than 3 days.¹⁸ Our patient received single donor apheresis platelets and not cold centrifuge platelets pooled from random donors. The AP he received were ABO matched platelets and they were freshly prepared on the same day of transfusion (therefore they were not stored platelets), yet he developed PR.

Platelet refractoriness is said to occur if 2 sequential one-hour post-transfusion platelet increments are $\leq 11 \times 10^9/L$. In the index case, apart from the first AP that was transfused on Day 8 of admission, the subsequent post transfusion platelet increments prior to commencement of chemotherapy were all $< 11 \times 10^9/L$. This confirmed that our patient had PR. Platelet refractoriness is a feared complication that arises in haematological malignancies, especially AML.²⁸ Patients with AML have a 10 – 15% risk of developing anti-HLA antibodies and therefore are at an increased risk for PR.²⁹ In a study by Ferreira *et al* to identify platelet refractoriness in 16 patients with haematological malignancies who received multiple platelet transfusions; 2 out of the 3 confirmed cases of PR had AML.²⁰ Our patient did not have an identifiable non-immune cause of PR because the usual causes such as fever, DIC, active bleeding, drugs and sepsis were absent. The fever had subsided by the time platelet transfusions commenced; he did not have splenomegaly or DIC. Identification of immune mediated causes of PR poses a diagnostic challenge especially in low resource settings.²⁰ In well-developed centres, the risk for development of PR due to alloantibodies in multiple transfused persons can be determined using microbead flow cytometry.³⁰ Diagnosis of PR may be by lymphocytotoxicity test, enzyme linked immunosorbent assay (ELISA), antigen capture ELISA, platelet immunofluorescence test (PIFT) or the use of panel reactive antibodies against human leukocyte antigen class I (PRA-HLA).²⁰ In a low-medium income country hospital like ours we were not able to ascertain that our patient developed immune mediated alloantibodies to the ABO, HLA or HPA antigens on the platelets.³¹ It is worthy to note that many patients may concurrently have PR secondary to both immune and non-immune mediated mechanisms.¹⁵ Therefore, although he had hepatomegaly which likely caused the PR, we could not identify if there was also co-existing immune mediated PR.

Although leukoreduction can reduce the risk for PR, it is not commonly done in our environment. Our patient received AP, but he also had several FWB which were not leukoreduced. This may have



predisposed him to PR. Management of PR depends on the cause. In non-immune cases, the underlying cause should be treated. In our patient the hepatomegaly which he had at presentation was the most probable cause of hepatomegaly³², especially as the liver regressed in size as chemotherapy commenced. Treatment options in PR secondary to immune causes include transfusion of crossmatch compatible platelets (which increases transfused platelet survival), giving HLA matched platelets or identifying the offending alloantibody and transfusing only donor platelets that lack the corresponding antigen.^{21, 33} Due to practice in a resource poor setting, we were unable to determine the presence of HLA or platelet specific alloantibodies. For this reason also, transfusion of HLA/ HPA matched platelets or transfusion of crossmatched platelets were not options for use in his management. Although intravenous immunoglobulin (IVIg) and Rituximab can be used in immune-mediated PR,³⁴ the cost of these drugs posed a challenge in his management.

The decision to go ahead with chemotherapy despite profound thrombocytopenia and PR had to be made because of the proliferative nature of AML and increasing blast count in peripheral blood film. He was on admission for 21 days before chemotherapy was commenced. The PR eventually resolved with chemotherapy and continued transfusion of FWB or AP which resulted in a gradual rise by the 10th day of chemotherapy and a normal platelet count was achieved by 21st day of chemotherapy. Since our patient subsequently responded to transfused platelets while on chemotherapy, he may not have had an immune-mediated PR. This is because if alloantibodies were present, with further transfusion during the chemotherapy he would still have had destruction of platelets by the alloantibodies thereby causing thrombocytopenia; however, this was not the case. As management for AML was instituted with continued transfusions, the PR was corrected with rising platelet count. Remission is achieved when there is normalization of the full blood count, no blasts in the peripheral blood film, bone marrow blast count is <5% and a normal clinical state.¹⁴ Our patient went into remission during the first cycle of chemotherapy and he achieved remission for AML. In this report, a patient with AML complicated by paraplegia and PR was successfully managed with subsequent correction of thrombocytopenia and attainment of remission in a low resource setting.

Limitations of the study: The patient was not able to do brain or spinal cord imaging as requested, due to financial constraints.



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