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Mitigation of Myelosuppression by *Musa acuminata* and Aminopyrimidine in Doxorubicin Treated Rats

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Abstract **Original Research Article**

Doxorubicin is a widely used antineoplastic drug, but its clinical efficacy is limited by dose-dependent haematotoxicity predominantly related to oxidative stress and subsequent cellular damage. The present study examined the protective effects of *Musa acuminata* sap (MAS) and aminopyrimidine in doxorubicin-induced haematological toxicity in Wistar rats. Thirty-five male Wistar rats were randomly divided into five groups, with 7 rats in each group: Group A (normal control); Group B (doxorubicin alone); Group C, D, and E (doxorubicin plus MAS at 50, 100, and 200 mg/kg, respectively, and aminopyrimidine at 10 mg/kg). Treatments were orally given for 42 days and doxorubicin was administered intraperitoneally at 3 mg/kg once weekly according to the study protocol. MAS was subjected to phytochemical screening and gas chromatography-mass spectrometry (GC-MS) analysis. Haematological indices and splenic histology were assessed, and data were analysed by one-way analysis of variance followed by appropriate post hoc comparisons, with $p < 0.05$ considered statistically significant. Phytochemical screening showed the presence of flavonoids, phenols, tannins, alkaloids, saponins, terpenoids and glycosides while GC-MS analysis identified aminopyrimidine as one of the major constituents. Doxorubicin treatment resulted in significant haematological derangements such as decrease in red blood cell count, haemoglobin concentration, packed cell volume and mean corpuscular haemoglobin concentration with leukopenia, neutrophilia, increased platelet counts and splenic histological damage. Co-administration of MAS and aminopyrimidine significantly reduced these changes in a dose-dependent manner, the group administered 100 mg/kg MAS plus aminopyrimidine exhibited the most significant recovery of haematological parameters and better preservation of splenic architecture. The combination therapy exhibited more protective effect than MAS alone. These findings indicate that MAS in combination with aminopyrimidine ameliorates the doxorubicin-induced haematological toxicity in rats through the antioxidant and cytoprotective effects.

Keywords: *Musa acuminata*, Aminopyrimidine, Doxorubicin, Haematotoxicity, Antioxidant.

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INTRODUCTION

Chemotherapy is still one of the great pillars of cancer management. However, its clinical utility is often hampered by toxic effects on normal tissues. Doxorubicin, an anthracycline widely used in oncology, is particularly effective against several malignancies, but its use is constrained by dose-related adverse reactions that include myelosuppression and damage to the hematopoietic microenvironment [1, 2]. Because the bone marrow is highly sensitive to oxidative injury and apoptotic signaling, exposure to doxorubicin can disrupt the production and survival of blood cells, leading to clinically relevant haematological alterations. The haematological consequences of doxorubicin toxicity are important not only because they reflect marrow injury, but also because they compromise host defense and oxygen transport [3].

Reductions in red cell indices may contribute to fatigue and reduced tissue oxygenation, while leucopenia and neutropenia increase the risk of infection; thrombocytopenia may further predispose to bleeding complications. These changes have practical implications in preclinical and clinical settings where they may require treatment delay, dose reduction or use of supportive care. Therefore, it remains a worthwhile biomedical goal to identify agents that can limit doxorubicin-induced blood toxicity [4].

The interest in plant-derived compounds as protective adjuncts to conventional chemotherapy has increased significantly in recent years. Many medicinal plants contain flavonoids, phenols, alkaloids and other secondary metabolites that can counter oxidative stress and modulate inflammatory responses. Such bioactive constituents are attractive because they may reduce tissue injury without completely opposing the therapeutic action of anticancer drugs [5].

In this context, *Musa acuminata* has received increasing attention, not merely as a food crop, but as a source of biologically active fractions with potential medicinal value.

Available evidence suggests that *Musa acuminata* possesses activities that may be relevant to toxicity mitigation [6]. A report on *Musa acuminata* sap-derived flavonoid indicated anti-inflammatory effects accompanied by reduced leukocyte response and no obvious adverse haematological effect in the model used. Additional experimental work on *Musa acuminata* stem and other plant parts has also described favourable effects on hematological indices, supporting the view that the plant may exert hemomodulatory properties. These findings do not yet establish a protective role against doxorubicin, but they provide a credible rationale for further investigation in a chemotherapy-induced toxicity model [7].

Aminopyrimidine is another compound of interest in experimental pharmacology because members of this chemical class are associated with diverse biological activities. The structure and substituents of aminopyrimidine derivatives may modulate different cellular signaling pathways related to inflammation, oxidative balance and survival responses. This makes the class relevant to studies aimed at reducing drug-induced tissue injury [8]. In a model of doxorubicin exposure, aminopyrimidine may therefore serve as a useful comparator or potent protective agent, especially where the outcome of interest is preservation of haematological parameters.

The present study was designed to evaluate the mitigation of doxorubicin-induced haematological alterations by *Musa acuminata* sap and aminopyrimidine in Wistar rats. The study aims to determine whether either intervention can maintain erythrocytic, leucocytic and platelet-related parameters by measuring important blood indices following toxic challenge. The findings might aid in a better understanding of inexpensive, biologically plausible approaches for mitigating chemotherapy-associated hematotoxicity and may also support the future development of adjunctive therapies in oncology.

MATERIALS AND METHODS

Collection of Plant and Authentication.

The plant samples were identified and authenticated by Dr. Dilibe Clifford a plant taxonomist in the Department of Plant sciences and biotechnology, University of Nigeria Nsukka. The voucher specimen was prepared and deposited in the herbarium (*Musa acuminata*: UNN 3019 NSUKKA) of the Department.

Ethical Approval

All experimental procedure were conducted in accordance with National Institute of Health Guide for care and use of Laboratory Animal Science as stated in the guide to care and use of Laboratory Animal Resources. Ethical clearance for this study was obtain from Faculty of Basic Medical Sciences Research and Ethical Committee, of Enugu State University of Science and Technology, College of Medicine, Enugu, With Ethical Right Permission Number ESUCOM/FBMS/ETR/2025/009.

Preparation and Extraction of Musa acuminata Extract.

The freshly cut stems were washed with clean tap water after the rotten parts of the stems had be removed. The pseudo stems were unfolded and washed twice with clean tap water. The stems will then chopped into smaller pieces (5-10cm) using a cutlass and crush them into a paste and the banana pseudo sap will be extracted immediately using a juicer extractors and strainer machine in order to get its sap which is the main component of the product. About 1.2 L of *Musa acuminate.*

Phytochemical Analysis

The plant extracts were subjected to phytochemistry and GC-MS analysis using a gas chromatography apparatus connected to an MS (5977B). A 0.25 µm film DB-5 fused-silica capillary column (J&W Scientific, Folsom, CA) was employed for the separation process. The Mass Hunter library was used to identify peaks in the mass spectrum, which was scanned from m/z 50 to 650 at a rate of 1.5 scans per second. The compounds were identified using retention time [9-11].

Lethal dose (LD_{50}) of the extract.

With a few minor modifications, the extract's lethality was assessed using Lorke's technique [12]. This approach consisted of two steps.

First Step

Nine (9) male rats were employed in this phase. The rats were divided into 3 groups of 3 rats each. The leaf extract was administered at 500, 1000, and 1500 mg/kg to each animal group. The animals were kept under observation for a whole day to track both mortality rate and behaviour.

Second Step

Four (4) male rats were divided into four (4) groups of one (1) rat each throughout this phase. After administering 2000, 3000, 4000, and 5000 mg/kg of the

leaf extract, respectively, to the rats, they were monitored for 24 hours post-treatment for behavioural changes (dullness,
restlessness, sedation, agitation), signs of toxicity, and mortality.

The following formula was used to determine the extract's LD_{50}:

: $LD50 = \sqrt{D_0 \times D_{100}}$

Where;

D_0 = Highest dose that gave no mortality

D_{100} = Lowest dose that produced mortality.

Experimental Animal Groupings

A total of thirty-five (35) Wistar rats were used for this study and were assigned into five groups of seven (7) rats per group. The animal grouping are as follows:

Group 1 : (Normal control) and received feed and water *ad libitum* only

Group 2: Received 3mg/kg of doxorubicin only (DOX)

Group 3: Received 50mg/kg of the MAS extract and 10mg/kg Aminopyrimidine (ADP)

Group 4: Received 100mg/kg MAS extract and 10mg/kg Aminopyrimidine (ADP)

Group 5: Received 200mg/kg of the MAS extract and 10mg/kg Aminopyrimidine (ADP).

Induction of Haematotoxicity

Haematological damage was induced in all the animals in the treatment groups except the control group intraperitoneally with 3 mg/kg doxorubicin once weekly for six weeks according to the study protocol [1].

Blood Samples Collection

After forty two days of oral administration of the extract, forty eight hours after the last dose were administered to each of the groups, the animals were anesthetized with chloroform, dissected to exposed the cardiac cavity of the heart, blood was obtained by orbital puncture using heparinized capillary tube inserted into the medial canthus of the eye (30 degrees to the eye) and carefully discharge into container tubes coated with Ethylene diamine tetra acetic acid (EDTA) for hematological analysis by running it down the side of the bottle, covered and rolled gently to mix with EDTA so as to avoid clotting. The sample bottles were labelled accordingly for all the 5 groups [13-19].

Hematological Analysis

The methods of Charles *et al.,* [20] was adopted to determine the Packed cell volume (PCV), haemoglobin concentration (Hb conc.), red blood cell count (RBC count), white blood cell count (WBC count), platelet count (PLT count), mean corpuscular haemoglobin concentration (MCHC), mean corpuscular volume (MCV), absolute count of lymphocytes, absolute count of neutrophils, platelet distribution width (PDW), mean platelet volume (MPV), reticulocyte count (Retics. count) and mean corpuscular haemoglobin (MCH). All the haematological parameters were analysed using Mindary 5 Path Differential BC 5300 Autoanalyser at Enugu University of Science and Technology Teaching Hospital (ESUTH) Parklane GRA.

Histological Analysis

For histologic analysis, the spleen was quickly fixed for 24 to 48 hours in 10% buffered formalin. Each tissue sample was routinely processed and embedded in paraffin following fixation. Tissue samples fixed in paraffin were sliced into 5-µm-thick slices for standard histologic analysis. Sections were stained with haematoxylin-eosin following deparaffinisation and rehydration. A light photomicroscope was used to examine and take pictures of each region [21-24].

Statistical Analysis

Version 29 of the Statistical Package for the Social Sciences (SPSS) was used to analyse the data from the study. Both post hoc LSD and ANOVA were used to analyse the data. At $p < 0.05$, the data were deemed statistically significant.

RESULTS

Table 1: Results of Qualitative Phytochemical Analysis

S/N	Constituents	Results
1	SAPONINS	-
2	ALKALOIDS	-
3	TANNINS	+
4	FLAVONOIDS	+
5	TERPENOIDS	-
6	STEROIDS	-
7	GLYCOSIDES	+

KEY: - = absent; + = present

Table 2: GC-MS analysis of *M. acuminate* sap extract

S/N	Name of the Compound	RT (min)	Molecular Formula	Extract mass (g/mol)
1	Polycyclic Hydrocarbon	0.934	C8H10	106
2	Pyridine	1.060	C6H6N2O3	154

3	1,2-Dimethyl cycloprop-1-ene	5.387	C5H8	68
4	Dihydrolansterol acetate	15.108	C33H54O4	514
5	Amine	15.423	C10H40N12Si4	440
6	Acetoxy-24-methylene	15.960	C33H54O4	514
7	5- Nitroguaiacol/5-hydroxy-2-nitrophenol	16.320	C21H22N4O3S	410
8	Ethynyl acetate	16.663	C20H22N4O10S	510
9	Propanoic acid	16.800	C28H28Si	392
10	Aminopyrimidines	16.869	C27H37Cl2N7O	545
11	Steroid	17.017	C31H44O5	496
12	Ergo sterol	17.143	C30H48O2	440
13	Fatty acid	17.240	C27H52O2	408
14	N-Phenylacetamide	17.417	C19H7Cl2F6NO3	481
15	Chorostyrene	17.549	C26H21Cl2N3O2	477
16	Isopropenyl	17.577	C30H52	412
17	Indole acetic acid	17.726	C19H18BrNO3	387
18	steroid	17.863	C21H31BrO2	394
19	Ethyl	17.926	C21H18N2O5	378
20	Steroid	17.989	C30H48O2	440
21	Zinc dipentylcarbamate	18.480	C22H44N2S4Zn	528
22	Coprostanone	18.612	C28H47N3O	441
23	Steroid	18.875	C28H34O8	498

Table 3: Values of body weight of the study animals

GROUPS	WEEK 1	WEEK 2	WEEK 3	WEEK 4	WEEK 5	WEEK 6
A (control)	160.43±8.74	171.71±9.42	156.00±2.92	161.50±5.67	175.00±7.79	208.33±2.40
B (DOX)	137.29±1.85	149.85±3.72	130.00±5.57$^{\alpha}$	152.83±6.42	167.50±5.59	201.17±5.23
C (LDEG + DOX)	149.57±7.17	158.14±8.16	136.29±6.94	152.00±9.18$^{\beta}$	167.85±10.19	178.57±11.64
D (MDEG + DOX)	131.14±3.61	139.86±4.30 $^{\alpha}$	125.00±3.61 $^{\alpha}$	157.71±8.78 $^{\beta}$	169.14±10.10	177.85±10.45
E (HDEG + DOX)	149.71±5.42	159.14±5.31	142.14±4.97	144.00±5.17 $^{\beta}$	154.14±6.78	169.28±9.44
P-VALUES	0.0035	0.0160	0.0014	0.0012	0.2505	0.0222

Table 4: Values of RBC and its Indices of the study animals

GROUPS	RBC (X10^{6}/L	Hb (g/dl)	PCV (%)	MCHC (g/dL)	MCV (fL)	MCH (pg)
A (Control)	9.76±0.23	19.05±0.15	50.00±0.15	377.50±14.50	54.14±2.75	19.50±0.70
B (DOX)	6.69±0.62	12.75±0.80	38.00±3.00$^{\alpha}$	328.50±3.50$^{\alpha}$	57.45±0.55	19.00±0.50
C (LDEG + DOX)	6.23±0.40	12.85±0.65	38.50±2.50	306.00±44.0$^{\alpha}$	69.55±7.35$^{\alpha}$	20.45±0.25
D (MDEG + DOX)	8.79±0.93	17.25±1.55	50.50±3.50$^{\alpha}$	370.00±22.00$^{\alpha}$	60.25±3.75	20.30±0.40
E (HDEG + DOX)	8.24±0.87	14.65±0.65	43.50±2.50	338.00±14 .00$^{\alpha}$	82.75±19.25$^{\alpha}$	20.00±0.10

Table 5: Values of Platelets and WBC of the study animals

GROUPS	Platelets (x10^{9}/L)	Neutrophils	Lymphocytes	WBC
A (CONTROL)	324.00±114.00	13.00±5.00	70.50±0.50	9.25±0.15
B(Dox Only)	877.00±102.00.	40.00±8.00	91.50±0.50	5.90±0.20
C(LDEG + DOX)	456.50±324.50	46.00±5.00	53.00±4.00	5.60±0.23
D(MDEG + DOX)	35O.50±120.50 $^{\alpha}$	27.50±0.50	59.00±8.00	8.00±0.41 $^{\alpha}$
E(HDEG + DOX	549.00±272.00	44.00±19.00	52.50±18.50	6.17±0.01
P-VALUE	0.0753	0.2662	0.4214	0.0167

Histological Examination

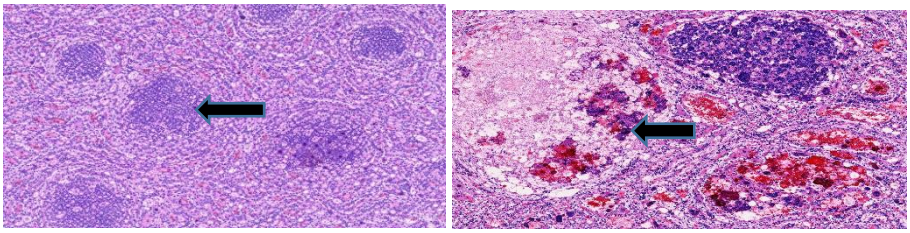

Plate 1: Photomicrograph Control group Plate 2: Photomicrograph Positive Control

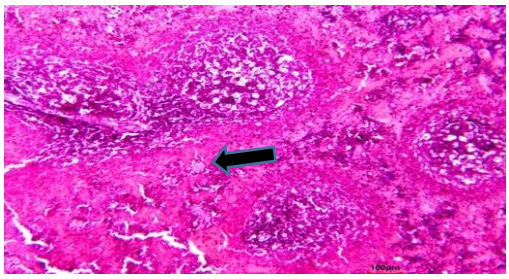
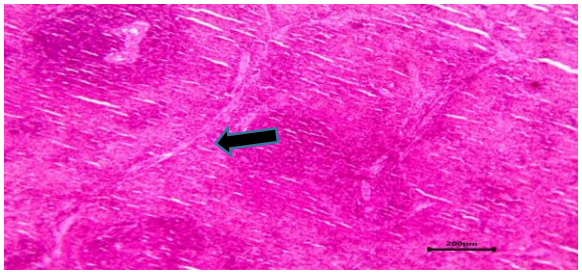

Plate 3: Photomicrograph Low dose 50mg/kg. Plate 4. Median –dose group 100mg/kg

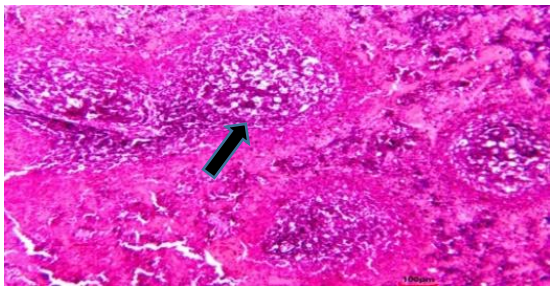

Plate 5: Photomicrograph High dose 200mg/kg

DISCUSSION

This study investigated whether *Musa acuminata* sap (MAS), administered with aminopyrimidine, mitigates doxorubicin (DOX) induced haematological toxicity in Wistar rats. Three principal findings emerge. First, qualitative phytochemistry and GC–MS profiling identified antioxidant relevant classes (tannins, flavonoids, glycosides) alongside sterol and steroidal derivatives and an aminopyrimidine related peak. Second, DOX produced the expected myelotoxic and haemotoxic phenotype—marked reductions in RBC, Hb, PCV and MCHC, leukopenia, and histological splenic damage. Third, co administration of MAS plus aminopyrimidine, particularly at the medium dose, substantially attenuated these haematological deficits and ameliorated bone marrow lesions.

Phytochemical composition and mechanistic plausibility

Table 1 showed a significant Phytochemical analysis of *M. acuminate.* The sap's content of tannins, flavonoids and glycosides offers a plausible biochemical basis for protection. These polyphenols scavenge reactive oxygen species (ROS), chelate transition metals, and stabilize cell membranes—mechanisms directly relevant to DOX toxicity, which is mediated largely by oxidative stress and lipid peroxidation. Table 2 showed a significant GCMS analysis of *M. acu minata*. The GC–MS profile further revealed sterol/sterone derivatives and fatty acids that can support membrane integrity and modulate inflammatory signalling. Detection of aminopyrimidine related compounds in the extract or as a co administered agent provides an additional mechanistic element: certain pyrimidine derivatives have been reported to influence redox-sensitive pathways and may preserve bone marrow function under chemotherapeutic stress. The present finding is concordant with the study of Akinmoladun *et al.,* (2022). [25]

Systemic effects and body weight

Table 3 showed the body weights of the Rat control group and the treated groups. DOX caused transient but significant weight loss compared with controls, consistent with chemotherapy associated anorexia and catabolism. Medium and high dose MAS groups showed partial recovery of body weight trends relative to the DOX group, suggesting systemic attenuation of toxicity. While the weight data are supportive, variability and intermittent significance across weeks indicate modest systemic benefits that warrant confirmation with larger cohorts and measures of food intake and metabolic markers.

Haematological protection and dose response

DOX produced classical myelosuppression: decreased RBC, Hb, PCV and MCHC (Table 4). The medium dose group (100 mg/kg) restored these indices toward control values more effectively than the low (50 mg/kg) or high (200 mg/kg) doses. Low dose was largely ineffective and associated with elevated MCV, whereas high dose produced partial recovery but also marked macrocytosis. This inverted U dose–response suggests an optimal therapeutic window in which antioxidant and marrow supporting effects are maximized without inducing compensatory or off target responses (e.g., excessive reticulocytosis or nutrient perturbation).

White cells and platelets

In table 5, total WBC declined with DOX and showed partial normalization with medium dose group,

supporting marrow preservation beyond erythropoiesis. Differential data were variable; because only percentages were reported, interpretation is limited without absolute counts. Platelet results showed high variability and a paradoxical elevation in the DOX group—an atypical finding given DOX's thrombocytopenic potential. The large standard deviations suggest sampling variability or reactive thrombocytosis; confirmatory studies with larger sample sizes and bone marrow cytology are needed.

Mechanistic interpretation

The protective effects likely involve a combination of mechanisms such as antioxidant activity of flavonoids/tannins, scavenging ROS, preventing oxidative damage to erythroid precursors and mature erythrocytes, membrane stabilization by sterols and fatty acids, preventing hemolysis and preserving PCV and Hb, improved marrow microenvironment by anti-inflammatory effect, leading to erythropoiesis and leukopoiesis, potential for aminopyrimidine to pharmacologically modulate redox and repair pathways to promote marrow recovery post DOX insult.

These mechanisms align with observed improved total WBC counts and erythrocyte indices in the medium dose group, although definitive assignment requires direct markers of oxidative stress, marrow histology and functional assays.

Spleen histopathology

Plates 1-5 showed the photomicrographs of control and treated groups. DOX produced spleenocytolysis, fiber disarray, cytoplasmic vacuolation and focal necrosis—findings concordant with ROS mediated haematotoxicity described in the literature. MAS plus aminopyrimidine attenuated these lesions in a dose dependent manner, with the medium dose showing the most consistent histological protection. This spleen protective signal supports systemic antioxidant action and suggests the combination may protect both haematopoietic and non haematopoietic tissues from DOX induced oxidative injury. The findings from this study is consistent with the studies of [25,26,27,28]

CONCLUSION

Musa acuminata sap combined with aminopyrimidine attenuated DOX induced anaemia, preserved leukocyte counts and reduced spleen histopathology in rats, with a medium dose yielding the most robust protection. The data support a protective role mediated plausibly by antioxidant, membrane stabilizing and marrow supporting activities. Validation with quantitative phytochemical analysis, mechanistic biomarkers and expanded experimental designs is warranted before considering translational development.

Conflict of interest

No conflicts of interest are disclosed by the authors.

Authors' Declaration

The authors hereby attest to the originality of the work and agree to bear all liability for claims about its content.

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