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Detection MN1 Gene Expression in Sudanese Patients Diagnosed with Acute Myeloid Leukemia (AML)

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Abstract:

- ***Background:***

The unchecked growth of immature blood cells in the bone marrow is the hallmark of acute leukemia, a potentially fatal hematologic cancer. The burden of acute leukemia is exacerbated in low-resource environments like Sudan by a lack of thorough cancer registries, regional healthcare inequities, and inadequate diagnostic skills. Meningioma 1 (MN1) overexpression (OE) is associated with the progression from myelodysplastic syndrome to acute myeloid leukemia (AML) and correlates with poor survival in AML patients. So this study conducted to detect the expression of MN1 among Sudanese patients diagnosed with AML.

- ***Methodology:***

100 AML patients recruited in this study, they were diagnosed with basic steps of diagnosis, which involved bone marrow, flow cytometer and complete blood count also conducted.MN1 detection was conducted through application of real time polymerase chain reaction (RT-PCR). Data analysis conducted by SPSS version 20.

- ***Result:***

Anemia found among 60% of patients, high white blood cell count (WBC) and low platelet count also observed. The overexpression of Meningioma-1 (MN1) is common in AML. High expression levels of MN1 have been shown to correlate with poor prognosis. MN1 remains poorly characterized both functionally and structurally. Some evidence suggests that MN1 can act as transcriptional activator. Forced expression of MN1 in murine bone marrow causes the development of an aggressive leukemia.

Keywords: *Meningioma 1, Real Time Polymerase Chain Reaction.*

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I. INTRODUCTİON

One of the most prevalent cancerous conditions impacting people worldwide is leukemia. With 437,033 cases and 309,006 deaths, leukemia was the fifteenth most prevalent cancer diagnosed worldwide in 2018, making it the eleventh leading cause of death from malignant diseases. Leukemia is geographically widespread, with more

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developed nations having higher frequency and overall fatality rates. However, the death rate is higher in underdeveloped nations [1].

Certain types of leukemia and lymphomas are regarded as distinct manifestations of the same disease because the World Health Organization (WHO) classifies hematological neoplasms according to the cell of origin. Acute lymphocytic leukemia and acute precursor B- and T-cell lymphoblastic leukemia/lymphoma (ALL/LBL) and chronic lymphocytic leukemia and small cell lymphoma (a kind of adult B-cell lymphoma; CLL/SLL) are two examples. Clinically, CLL/SLL is regarded as a kind of lymphoma. However, depending on whether the cancer cells were discovered in the bone marrow, blood, or lymph nodes, population-based cancer monitoring programs usually classify CLL/SLL patients as either leukemias or lymphomas.[2].

The accumulation of somatic genetic changes in myeloid precursors that cause their clonal proliferation and maturation arrest is the hallmark of acute myeloid leukemia (AML), a cytogenetically and molecularly varied disease. It is generally known that one of the most significant independent prognostic markers for AML patients, both adults and children, is cytogenetic findings at the time of diagnosis. At the time of diagnosis, 40–50% of adult AML patients had normal cytogenetics. They are referred to as CN-AML, or cytogenetically normal AML. In terms of genetic alterations and treatment results, this group is diverse. Over the years, numerous organizations have attempted to dissect this diverse group using a variety of strategies. [3—6]. cancer cases in the US. With an estimated 5-year OS of 32% (up to 50% in young people and fewer than 10% in patients over 60), AML is primarily detected in elderly adults (median age at diagnosis: 68 years). However, given that 12 new medications or combination regimens have been approved since 2017, these results do not adequately reflect the significant shift in the treatment landscape [7-8]. The International Consensus Classification of AML expanded the range of classification determined by cytogenetic and mutational profiles by updating the previous revised fourth edition World AML is caused by the unchecked growth of clonal hematopoietic cells. It accounts for 1% of all newly diagnosed Health Organization (WHO) classification of AML with new genetic entities and modifications to the blast thresholds. Genetic abnormalities are prioritized in defining AML disease classification due to their overwhelming influence on disease phenotype and outcome. Other predisposing factors (therapy-related, prior myelodysplastic syndrome (MDS) or MDS/myeloproliferative neoplasm (MPN) [9-10].

The human chromosome contains the meningioma (disrupted in balanced translocation) 1 (MN1) gene. band 22q12 and produces a protein that joins the nuclear receptor RAR-RXR or the vitamin D receptor in a gene transcription regulator complex. This gene's role in human neoplasia was first identified in a case of meningioma with t(4;22), and it was also identified in myeloid malignancies with t(12;22). In a mouse model, high levels of MN1 expression have been linked to inv(16) AML and have been demonstrated to collaborate with CBFB-MYH11 gene fusion in the formation of AML [9-11]. According to recent reports, MN1 overexpression was associated with a worse prognosis for patients with CN-AML. However, these findings have not yet been independently verified or examined in relation to a number of other recognized prognostic indicators in CN-AML. Therefore, we assessed the MN1 expression in diagnostic bone marrow (BM) samples from younger adult CN-AML patients that were also thoroughly characterized for other molecular markers associated with prognosis in order to validate the prognostic significance of MN1 expression in CN-AML. Additionally, we identified gene and microRNA expression profiles linked to variations in MN1 expression levels in order to obtain understanding of MN1-mediated leukemogenesis [12-13].

II. MATERIAL AND METHOD

This study was conducted as cross sectional one, included 100 patients diagnosed with AML. Peripheral blood samples were collected as well as the samples of blood bone marrow specimens were collected in Ethylene diamine tetra acetic acid (EDTA) containers for each patient. Bone marrow samples (1ml) from patients were collected on EDTA from patients with AML. Bone marrow was treated with erythrocyte lysis solution; Leukocytes were collected and stored in buffer RLT (1x107leukocytes) at -80 ℃ till use for complete RNA extraction. Total RNA was extracted from bone marrow cells using QIAamp RNA extraction blood Mini kit (QIAGEN) following the standard procedures according to the manufacturer's instructions. RNA was converted to complementary DNA (cDNA) using Applied Biosystems High Capacity cDNA Reverse Transcription Kit (Life Technologies) according to the manufacturer's instructions and stored at –20 ℃ till use [14].

Real-time RT-PCR: Quantitative assessment of gene expression levels was performed by TaqMan gene expression assay (Applied Biosystem, Foster City, CA, USA) as recommended by the manufacturer. The Step One Real-Time PCR System (Applied Biosystems, Foster City, CA, USA) was used for real-time analysis. Relative expression of MN1gene was analyzed by the comparative Ct method ($2^{-\Delta\Delta Ct}$) (Livak and Schmittgen, 2001), using glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as the endogenous control. Data were expressed as the fold change in MN1 gene expression [15].

III. RESULT

Samples were 100 AML patients with age between 16-67 years old, they were 40 male (40 %) and 60 female(60%) as in figure 1, their mean age of (49.4+11.1) years old. Parameters measured included hemoglobin concentration, white blood cell and platelet counts, beside peripheral blast cell (52.8+2.89%) and blast in bone marrow (63.2+2.1%) as in table 1.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26sep240

Participants AML patients sorted according to sub-types of AML according to FAB characteristics of blood picture, M4 was the most frequent, then M5, then M3, M1 and M2 then M7 and lower frequency was for M0 as in table 2 and figure 2.

Table 1 Descriptive Statistics of Hematological Parameters and Blast Percentages Among Study Participants (N = 100)

Variable	Mean ± SD
Age (years)	49.35 ± 11.10
WBC (×10⁹/L)	35.36 ± 7.10
Hemoglobin (g/dL)	9.01 ± 0.23
Platelet count (×10⁹/L)	55.40 ± 8.89
Peripheral blood blasts (%)	52.80 ± 2.89
Bone marrow blasts (%)	63.20 ± 2.10

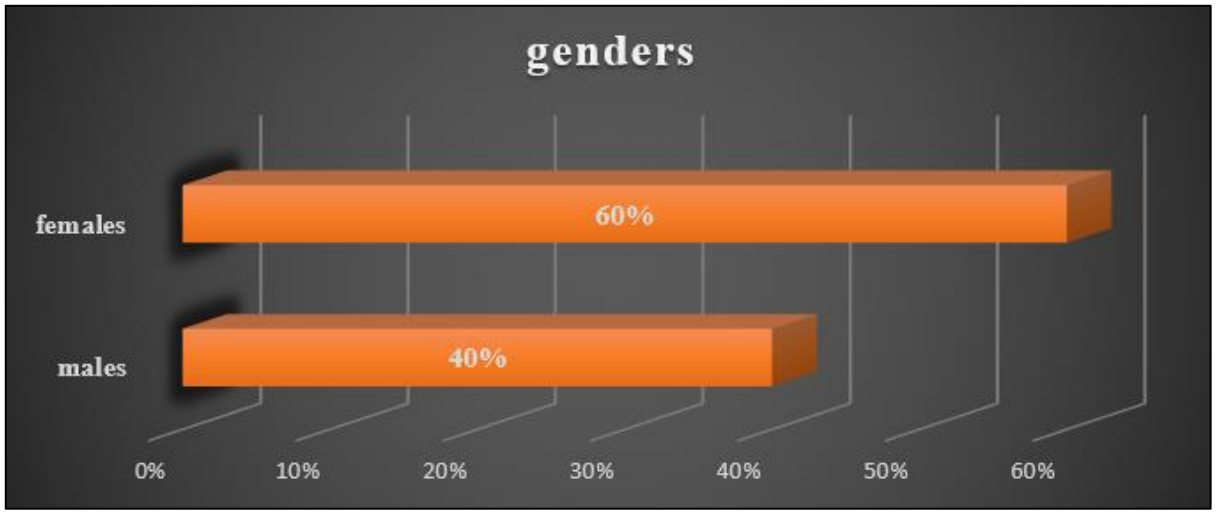

Fig 1 Gender Distribution Among AML Patients

Table 2 Overall Frequency Distribution of FAB Subtypes

FAB subtype	Frequency, n	Percent, %
M0	1	1.0
M1	12	12.0
M2	12	12.0
M3	16	16.0
M4	24	24.0
M5	19	19.0
M6	7	7.0
M7	9	9.0
Total	**100**	**100.0**

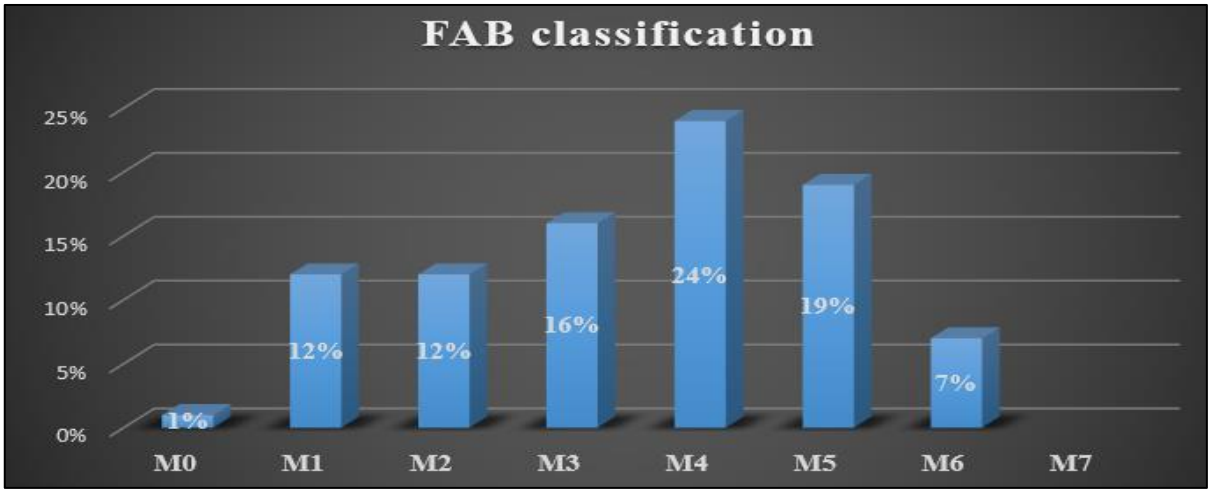

Fig 2 Classification of Study Population According to FAB Classification

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Presence of complications among AML patients involved extra medullary infiltration showed it occurred among 60% of patients as positive result and among the 40% negative or not occurred. Lymphadenopathy as in table 3 and figure 3.

Table 3 Clinical and Hematological Complications Among AML Patients

Complication	Status	Frequency, n	Percent, %
Extra-medullary infiltration	Positive	60	60.0
	Negative	40	40.0
Bone marrow cellularity	Normocellular	20	20.0
	Hypercellular	80	80.0
Lymphadenopathy	Present	27	27.0
	Absent	73	73.0
Splenomegaly	Present	28	28.0
	Absent	72	72.0
Hepatomegaly	Present	23	23.0
	Absent	77	77.0

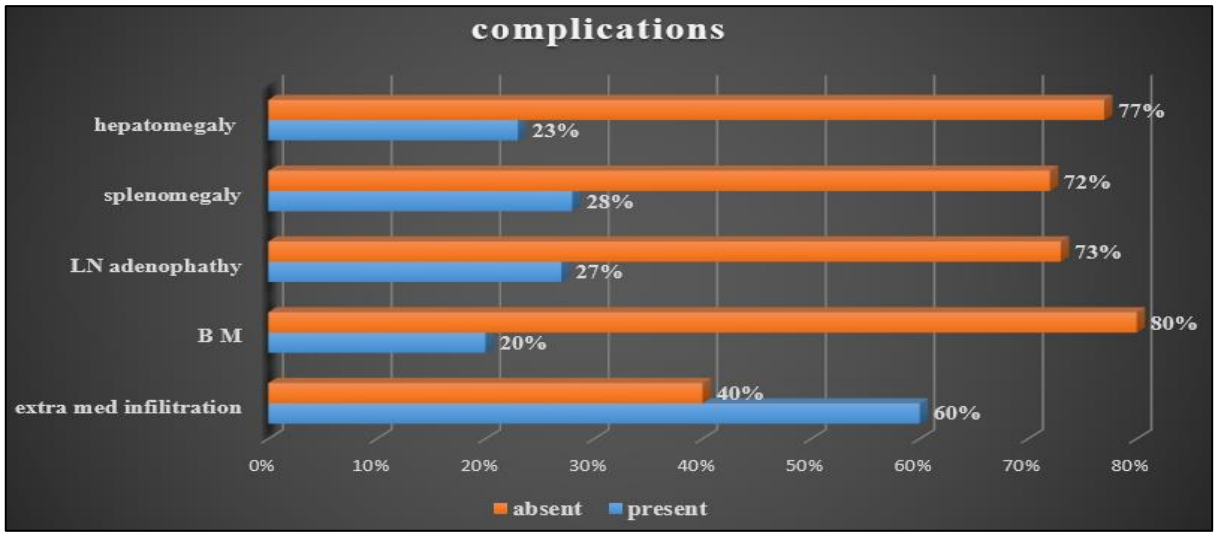

Fig 3 Complications Among AML Patients

Detection of MN1 gene among the 100 AML patients showed that 24% were under expression, 76% presented with over expression as in table 4 and figure 5.

Table 4 Distribution of MN1 Mutation Expression Among the Study Participants

MN1 mutation	Frequency	%
Under expression	24	24%
Over expression	76	76%
Total	100	100%

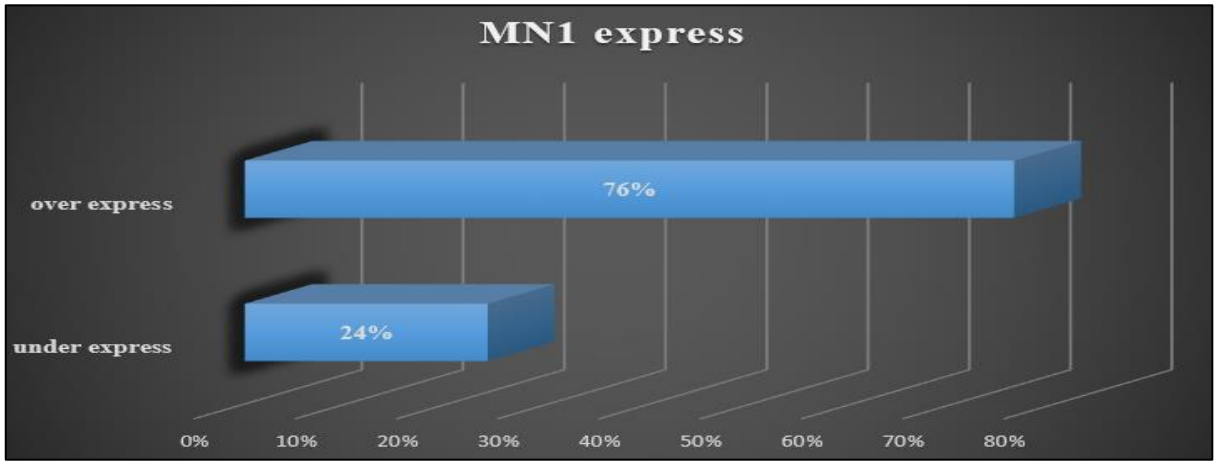

Fig 4 The Expression of MN1 Gene Among the Study Population

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Considering MN1, distribution of it occurrence among FAB classifications of AML participant patients showed that, over expression occurred more among M4, then M5 then M3 and M2 and M7 while under expression occurred more among M1, 2, 3 and 4 and last one M5, comparing the incidence of MN1 among FAB brought significant difference as p value obtained was 0.000 as in table 5 and figure 4.

Table 5 Distribution of MN1 Expression Status Across FAB Subtypes of Acute Myeloid Leukemia

FAB subtype	MN1 expression, n (%)		Total, n (%)	*P value*
	Under-expression, n (%)	Over-expression, n (%)		
M0	0 (0.0%)	1 (1.3%)	1 (1.0%)	<0.001*
M1	8 (33.3%)	4 (5.3%)	12 (12.0%)	
M2	7 (29.2%)	5 (6.6%)	12 (12.0%)	
M3	3 (12.5%)	13 (17.1%)	16 (16.0%)	
M4	4 (16.7%)	20 (26.3%)	24 (24.0%)	
M5	2 (8.3%)	17 (22.4%)	19 (19.0%)	
M6	0 (0.0%)	7 (9.2%)	7 (7.0%)	
M7	0 (0.0%)	9 (11.8%)	9 (9.0%)	
Total	**24 (100.0%)**	**76 (100.0%)**	**100 (100.0%)**	

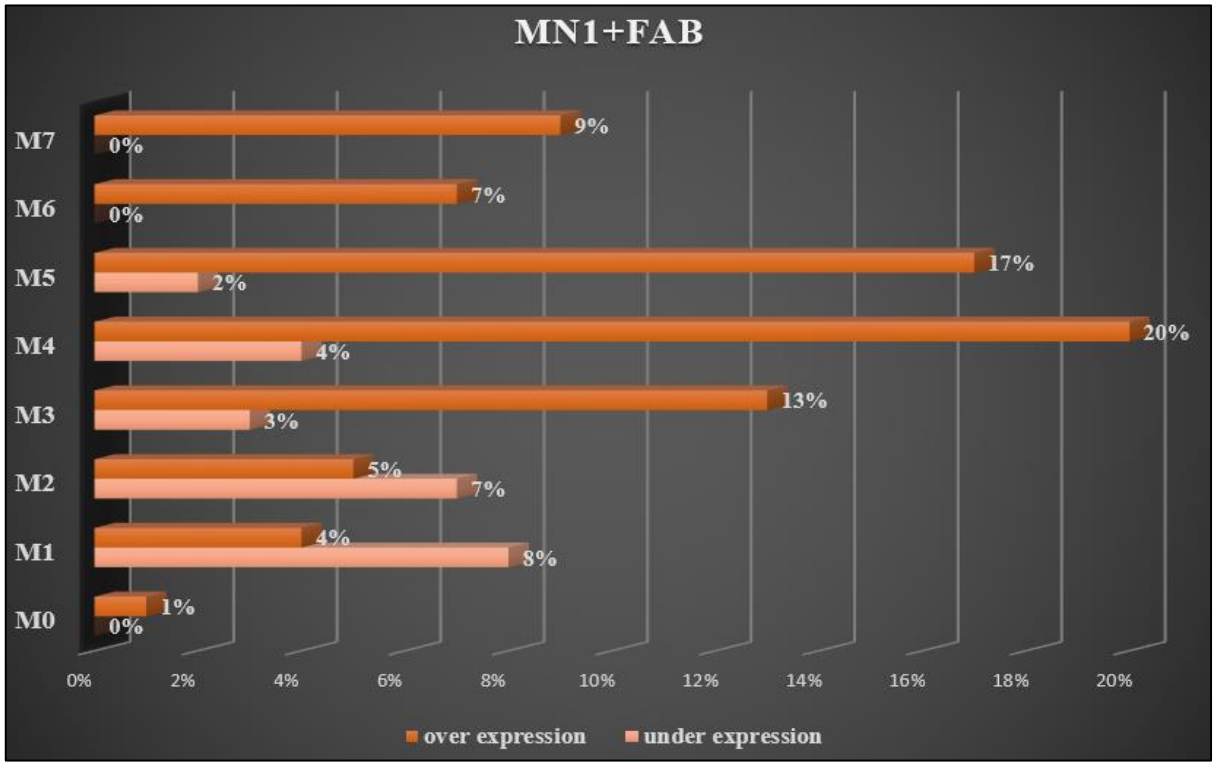

Fig 5 MN1 Occurrence Among Patients with FAB AML Classification

Comparing incidence of MN1 according to hepatomegaly, MN1 over expressed among 21% and absent among 3% and under expressed among 2% and absent among 74, giving significant difference as p value 0.000. MN1 gene among lymphadenopathy complications, comparing between present and absence of the mutation gave significant difference (p value 0.000) as MN1 occurred among 20% of over expression and absent lymphadenopathy 4% occurred, MN1 occurred among 7% of lymphadenopathy and absent among 69% of absent lymphadenopathy. MN1 gene presence among incidence of splenomegaly among AML patients, comparison showed that MN1 over expression occurred among 23 of splenomegaly and 1% without, under expression of MN1 occurred among 5% of patients with splenomegaly and 71% of not with splenomegaly giving significant difference with p value 0.000, as in table 6 and figures 6—8.

Table 6 Association Between MN1 Expression Status and Disease-Related Complications
A. Hepatosplenomegaly

MN1 expression	Present, n (%)	Absent, n (%)	*P value*
Over-expression (n = 24)	21 (77.8%)	3 (22.2%)	<0.001*
Under-expression (n = 76)	2 (2.6%)	74 (97.4%)	
Total	**23**	**77**	

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B. Lymphadenopathy

MN1 expression	Present, n (%)	Absent, n (%)	*P value*
Over-expression (n = 24)	20 (83.3%)	4 (16.7%)	<0.001*
Under-expression (n = 76)	7 (9.2%)	69 (90.8%)	
Total	**27**	**73**	

C. Splenomegaly

MN1 expression	Present, n (%)	Absent, n (%)	*P value*
Over-expression (n = 24)	23 (95.8%)	1 (4.2%)	<0.001*
Under-expression (n = 76)	5 (6.6%)	71 (93.4%)	
Total	**28**	**72**	

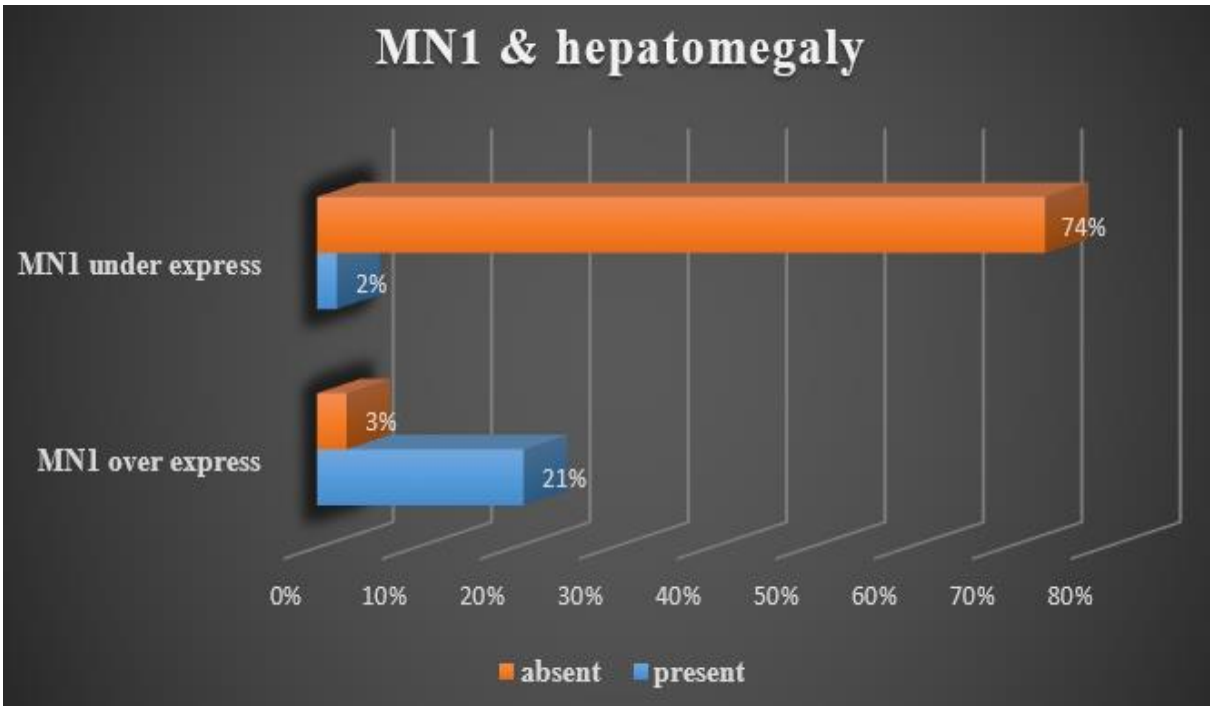

Fig 6 MN1gene Among Hepatomegaly Incidence

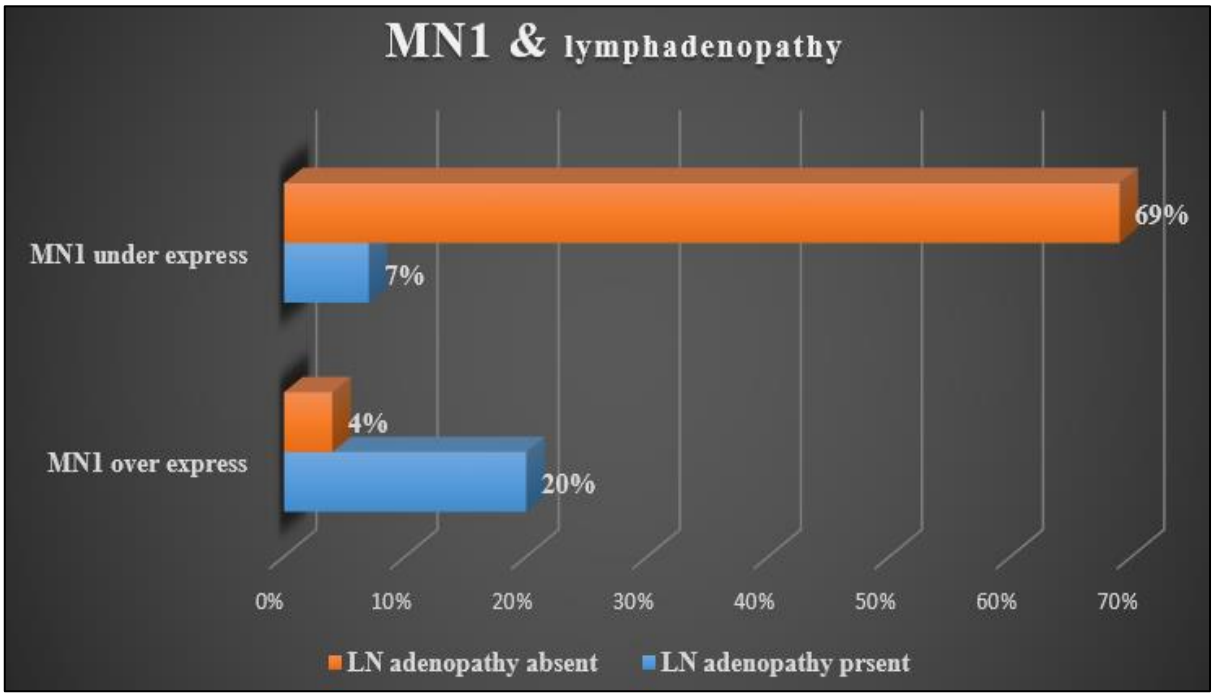

Fig 7 MN1 Gene and Incidence of Lymph Adenopathy

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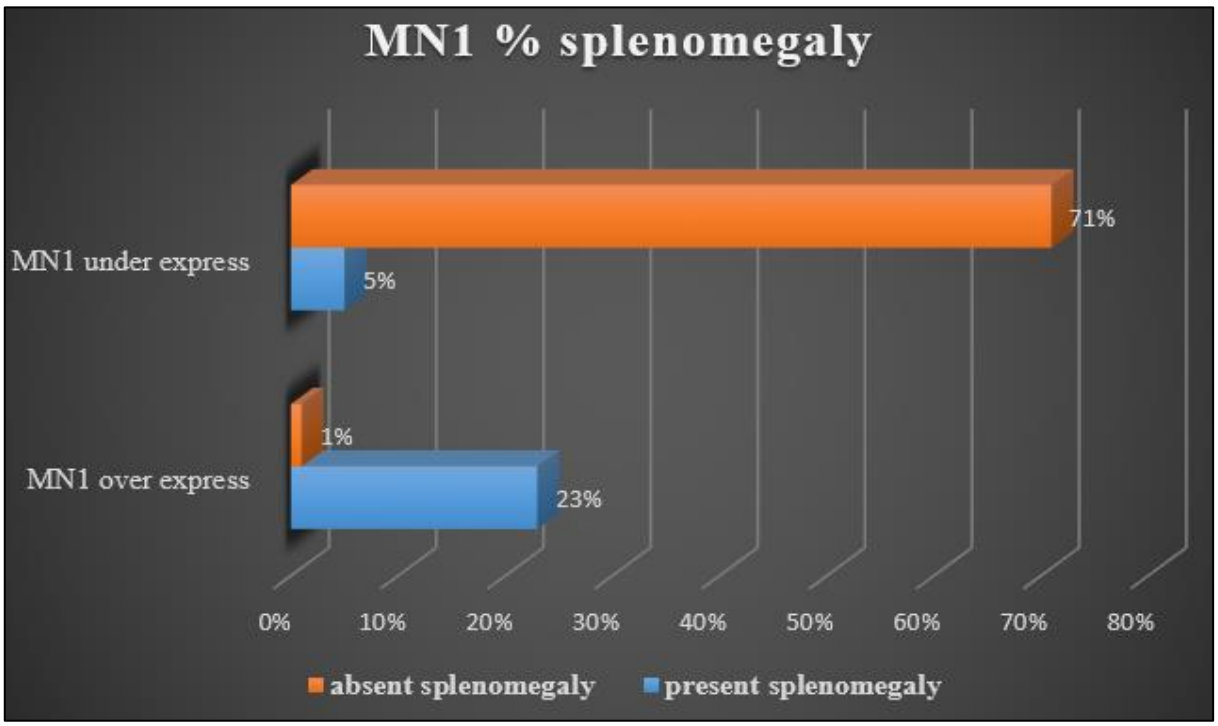

Fig 8 MN1 Gene and Splenomegaly

Association of MN1 mutation incidence and presence of complication among patients, there was high association as each complication has p value <0.05 as in table 7.

Table 7 Association Between MN1 Expression and FAB Subtype and Clinical Features Among Study Participants

Variable	MN1 expression	Present, n (%)	Absent, n (%)	Total, n	Chi-square	*P value*
FAB subtype (any)	Over-expression	24 (100.0%)	0 (0.0%)	24	27.93	0.001*
	Under-expression	76 (100.0%)	0 (0.0%)	76		
	Total	**100 (100.0%)**	**0 (0.0%)**	**100**		
Splenomegaly	Over-expression	23 (95.8%)	1 (4.2%)	24	70.5	<0.001*
	Under-expression	5 (6.6%)	71 (93.4%)	76		
	Total	**28 (28.0%)**	**72 (72.0%)**	**100**		
Hepatomegaly	Over-expression	21 (87.5%)	3 (12.5%)	24	64.3	<0.001
	Under-expression	2 (2.6%)	74 (97.4%)	76		
	Total	**23 (23.0%)**	**77 (77.0%)**	**100**		
Lymphadenopathy	Over-expression	20 (83.3%)	4 (16.7%)	24	49.8	<0.001*
	Under-expression	7 (9.2%)	69 (90.8%)	76		
	Total	**27 (27.0%)**	**73 (73.0%)**	**100**		

IV. DISCUSSION

The mean age of patients in this study was 49.35 years therefore indicates a predominantly middle-aged/adult AML population, consistent with an observation out of a study found that AML incidence remained highest in older adults across the entire study period, with little variation over time (Tahreem Malik et al 2025)[16]. The difference may reflect differences in referral patterns, population structure, inclusion criteria, and access to hematological diagnosis, this study also showed a female predominance (60%), with a male-to-female ratio of approximately 0.67:1. Unlike a Chinese study had proportion of males was higher than that of females (Male 53.75%; Female 46.25% (Xingyu Wan et al 2025)[17]. Similarly, the large multicenter analysis demonstrated approximately equal or modest male predominance across most FAB subtypes; for example, males represented 50% of M2, 50% of M4 and 50% of M5 cases. Thus, the female predominance observed in this Sudanese cohort may represent a population-specific finding or may be influenced by the characteristics of the study population.

➢ *Leukemia Clinical Characteristics:*

- *FAB Subtype Distribution*

The most striking difference between this study's findings and several published studies is the predominance of FAB-M4. In this study, M4 accounted for 24%, followed by M5 (19%), M3 (16%), M1 and M2 (12% each), M7 (9%), M6 (7%), and M0 (1%).

A partial agreement obtained by outcomes of a Sudanese study conducted as retrospective study by (Sitelbanat H. Omer et al 2017) [18] from January 2014 to

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December 2016. The total number of subjects was 140 that included both adults and children. The patients were diagnosed on the basis of bone marrow morphology using FAB classification. Among 140patients, 70 were males and 70 were females with male to female ratio 1:1. The age ranged between 3 years to 101 years with a mean age of 33 years. AML-M3 was the predominant French-American-British (FAB) subtype (29.3%) followed by M2 (19.3%), M4 (15%), M0 (12.9%), M1 (10%), M5 (6.5%), M7 (5%) and M6 (2.1%).

- *Hematological Parameters*

Our patients had a mean WBC count of 35.36 ± 7.1 $\times 10^9$/L, mean hemoglobin of 9.01 ± 0.23 g/dL, platelet count of 55.4 ± 8.89 $\times 10^9$/L, peripheral blood blasts of 52.8 ± 2.89%, and bone marrow blasts of 63.2 ± 2.1%. These findings demonstrate the typical hematological pattern of AML, characterized by substantial blast proliferation together with anemia and thrombocytopenia. These agree with a study (Bushra Noor et al 2025)[19] found anemia presented in 82.35%, while high WBC count in 41%, decrease in platelet count in 94%, AML

This study showed bone marrow blast percentage in your study (63.2%) was somewhat lower than the median of 73% reported in the large multicenter AML cohort, although substantial variation existed between FAB subtypes. The lower value in the study cohort could reflect differences in disease burden at presentation, patient selection, or the methods used to estimate marrow blasts.

A partial agreement obtained by a study (Al-Adhami A A. I et al)[20], anemia found among 8.40.94 g/dL, and Blast count was 75.26 16.04%). Leukocytosis was detected in (62.9%), and Thrombocytopenia in (86.3%).

- *Extramedullary Manifestations*

This study found extramedullary infiltration in 60% of patients. Specifically, lymphadenopathy was present in 27%, splenomegaly in 28%, and hepatomegaly in 23%. These findings indicate a relatively substantial burden of extramedullary disease. These in agreement with a study by (Yufan Lin et al 2025) [21], mentioned that extramedullary disease occurs in 1%-30% of acute myeloid leukemia (AML) patients, which been associated with adverse outcomes.

- *Important Point About your M7 Result*

Our study identified 9% M7, which is substantially higher than the frequencies reported in most published series. M7 was absent in the South Indian study and occurred at only about 1% in the large multicenter study. This is an important finding that should be discussed cautiously. Because M7 is uncommon, a relatively high percentage in a sample of only 100 patients could reflect local case enrichment, referral patterns, or limitations of morphology-only FAB classification. Confirmation using immunophenotyping would strengthen the classification.

Our findings demonstrated a distinct clinicopathological pattern compared with several previously published AML studies. The present study included 100 AML patients aged 16–67 years, with a mean age of 49.35 ± 11.1 years and a female predominance (60%).

The FAB subtype distribution also differed considerably. In the present study, FAB-M4 was the most frequent subtype (24%), followed by M5 (19%) and M3 (16%). The predominance of M4 and M5 in the present study appears to distinguish this Sudanese cohort from several international populations. The present study also demonstrated anemia, thrombocytopenia, leukocytosis and a high blast burden, with mean peripheral and bone marrow blast percentages of 52.8% and 63.2%, respectively. The relatively high frequency of extramedullary manifestations, including lymphadenopathy (27%), splenomegaly (28%) and hepatomegaly (23%), may be related in part to the substantial representation of monocytic/myelomonocytic AML subtypes, these differences may reflect geographical and population variation, referral patterns, sample size, and differences in morphological diagnostic practices. Importantly, FAB classification is primarily morphology-based and has largely been superseded by genetically and molecularly defined AML classifications, so comparisons with modern AML cohorts should be interpreted with caution.

One correction to consider: “M4 was the most frequent, then followed by M1 and M2 (12% each). M7 = 9%, which is unusually high compared with most published cohorts.

The present study demonstrated that MN1 overexpression was predominant among Sudanese AML patients, occurring in 76% of cases compared with 24% showing under expression. This finding is broadly consistent with an Egyptian study by (Roxan E Shafik et al 2017)[22] in which 67.4% of AML patients demonstrated high MN1 expression, although the proportion of overexpression in the present study was higher.

The distribution of MN1 expression across FAB subtypes showed a significant association ($p < 0.001$), with particularly high frequencies of overexpression among M4 and M5 patients. This finding is partly supported by previous molecular study (Cintia Carella et al 2007)[23] demonstrating marked MN1 expression in subsets of FAB-M4 AML, particularly cases carrying inv(16), although MN1 expression appears to vary according to the underlying genetic abnormality.

In contrast, the Egyptian study (Roxan E Shafik et al 2017)[22] demonstrated a different distribution, with M2 being the most frequent subtype among patients with high MN1 expression and M5 represented only among patients with low expression. These differences may reflect variations in population characteristics, FAB subtype distribution, genetic background and methods used to define high MN1 expression.

The present study further demonstrated strong associations between MN1 overexpression and hepatomegaly, splenomegaly and lymphadenopathy.

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The strong associations observed in the present study may partly reflect the high proportion of M4/M5 AML, which accounted for 43% of the cohort, since monocytic AML has distinctive biological characteristics and a propensity for tissue involvement. Nevertheless, this hypothesis requires confirmation through larger studies incorporating cytogenetic and molecular markers such as inv(16), NPM1, FLT3 and CEBPA. Previous study (Michael Heuser et al 2006)[24] has also demonstrated that high MN1 expression is associated with inferior treatment response, increased relapse and shorter overall and disease-free survival, suggesting that MN1 may have potential prognostic value in AML.

Also a consistency found by A study by (A. Ningombam, D. et al 2024)[25] evaluated the prognostic role of MN1 expression in adult CN-AML patients among One hundred and sixty-three de-novo adult AML patients by real-time PCR. MN1 expression was correlated with the clinical characteristics of the patients and their outcomes. Higher MN1 expression was associated with NPM1 wild-type ($p<0.0001$), and a high MN1 expression was associated with poor event-free survival (Hazard Ratio 2.47, 95% Confidence Interval: 1.42-4.3; $p<0.0001$) and overall survival (Hazard Ratio 4.18, 95% Confidence Interval: 2.17-8.08; $p<0.0001$). On multivariate analysis, the MN1 copy number emerged as an independent predictor of EFS ($p<0.0001$) and OS ($p<0.0001$).

V. CONCLUSION

- Demographic characteristics: AML was observed across a wide age range, with females comprising a higher proportion of the study population than males.
- Hematological findings: Patients showed anemia, thrombocytopenia, leukocytosis, and high blast percentages in both peripheral blood and bone marrow.
- FAB classification: M4 was the most common AML subtype, followed by M5 and M3, whereas M0 was the least frequent subtype.
- Bone marrow findings: Hyper cellular bone marrow was observed in 80% of patients.
- Extramedullary manifestations: Extramedullary involvement occurred in 60% of patients. Lymphadenopathy, splenomegaly, and hepatomegaly were present in 27%, 28%, and 23%, respectively.
- MN1 expression: MN1 overexpression was detected in 76% of patients, while 24% showed under expression.
- Association with FAB subtypes: MN1 expression was significantly associated with FAB classification ($p < 0.001$), with overexpression being particularly frequent in M4, M5, and M3 subtypes.
- Association with complications: MN1 overexpression was significantly associated with splenomegaly, hepatomegaly, and lymphadenopathy ($p < 0.001$).
- Clinical significance: The findings suggest that increased MN1 expression may be related to specific AML biological characteristics, particularly monocytic differentiation and extramedullary involvement.
- MN1 may have potential as a molecular marker for AML subtype characterization and identification of extramedullary disease.

RECOMMENDATION

Larger studies incorporating standardized AML classification, cytogenetic and molecular markers, and clinical outcome data are needed to establish the diagnostic and prognostic significance of MN1 expression.

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