

Prognostic Impact of *Wilms' Tumour 1* Mutation in Patients with Acute Myeloid Leukaemia

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ABSTRACT

Objective: To detect *WT1* gene alterations among individuals diagnosed with acute myeloid leukaemia (AML) and investigate their relation to the response of induction therapy.

Study Design: A descriptive study.

Place and Duration of the Study: Department of Haematology, Armed Forces Institute of Pathology, Rawalpindi, Pakistan, from June to December 2023.

Methodology: The study enrolled all freshly diagnosed AML patients who underwent clinical, haematological, and molecular testing. Based on their *WT1* mutation status, participants were categorised into distinct groups and assessed after four weeks of induction therapy. Independent t-test and chi-square tests were used to analyse the variables, while odds ratios (ORs) with 95% confidence intervals (CIs) were computed using cross-tabulation.

Results: Within the cohort of 98 newly diagnosed AML cases, patients had a mean age of 36.5 years, showing a male predominance with a male-to-female ratio of 1.22:1. *WT1* mutations were detected in 12 (12.2%) patients. These patients showed significantly lower haemoglobin, higher leucocyte counts, reduced platelet counts, and higher bone marrow blast percentage ($p < 0.05$). Complete remission occurred in 75% of *WT1*-mutated versus 62.8% of wild-type patients ($p = 0.408$). Although not statistically significant, *WT1* mutations demonstrated a trend towards a more aggressive presentation and poorer therapeutic response.

Conclusion: *WT1* mutation in AML is associated with aggressive disease and less differentiated French-American-British (FAB) subtypes. Although remission rates were lower in *WT1*-mutated cases, the difference was not statistically significant. Larger prospective studies are needed to establish its prognostic significance and guide individualised therapy.

Key Words: Acute myeloid leukaemia, *WT1* mutation, Induction therapy, Prognosis, FAB classification.

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INTRODUCTION

Acute myeloid leukaemia (AML) represents a diverse group of haematological cancers characterised by uncontrolled proliferation of immature myeloid precursors within bone marrow and peripheral circulation.¹ Despite progress in molecular characterisation and treatment options, overall survival remains limited, especially among high-risk groups.² Consequently, there is an ongoing need to identify reliable prognostic biomarkers that can aid in risk stratification and inform treatment decisions.

Located on the short arm of chromosome 11 at position 11p13, the *Wilms' tumour 1* (*WT1*) gene produces a transcription factor essential for proper cellular development and has been linked to multiple cancer types, including AML.³

WT1 is highly expressed in leukaemic blasts and has been shown to promote leukaemogenesis through various mechanisms, such as inhibition of apoptosis, regulation of cell cycle progression, and modulation of cellular differentiation.⁴ Notably, approximately 10% of patients with AML harbour *WT1* mutations, which have been connected to diverse clinical trajectories.⁵

Multiple research groups have examined how *WT1* mutations influence AML prognosis, yielding contradictory findings. Some studies have reported an association between *WT1* mutations and adverse clinical outcomes, such as lower complete remission (CR) rates, shorter disease-free survival (DFS), and shorter overall survival (OS).^{6,7} However, other studies have failed to demonstrate a significant prognostic impact of *WT1* mutations or have reported conflicting findings.^{8,9}

These discrepancies in published literature may stem from numerous factors, including variations in patient demographics, treatment protocols, and molecular detection techniques. Furthermore, the prognostic significance of *WT1* alterations might be modified by coexisting molecular abnormalities, particularly *FLT3*-ITD and *NPM1* mutations, which are recognised as having a substantial impact on AML patient outcomes.¹⁰

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Considering the potential importance of *WT1* mutations as prognostic indicators in AML, a thorough understanding of their clinical ramifications is essential. This study sought to examine the prognostic implications of *WT1* mutations within an AML patient cohort. This study aimed to specifically assess how these mutations influence treatment response following induction chemotherapy while considering other pertinent clinical and molecular variables.

By elucidating the prognostic role of *WT1* mutations in AML, this study may contribute to improving risk stratification and personalised treatment approaches, ultimately improving patient outcomes.

METHODOLOGY

This investigation employed a prospective cohort study design and was executed within the Department of Haematology, Armed Forces Institute of Pathology, Rawalpindi, Pakistan, from June to December 2023. Ethical approval was obtained from the Institutional Review Board of Armed Forces Institute of Pathology, Rawalpindi, Pakistan (Ref. No. FC-HEM22-17/READ-IRB/23/2189). The sample size for this study was determined using the WHO calculator, considering a previously observed probability of *WT1* gene mutation to be 6.8%.¹¹ These calculations determined an optimal sample size of 98 participants, allowing for a 5% error margin with 95% confidence level. Participant recruitment employed a non-probability convenience sampling technique. All patients received comprehensive study information and provided informed consent. The maximum available participants (98) during the study timeframe were enrolled.

The study enrolled patients with recent cytogenetically normal AML diagnoses, irrespective of age or gender. Patients with secondary AML transformed from other blood disorders, and those with previous chemotherapy or radiation exposure, were excluded.

Clinical information, including age, gender, presenting symptoms, and physical examination findings, was recorded at presentation. Haematological evaluation was performed on 3 mL of EDTA-anticoagulated blood for complete blood counts, including total leucocyte count (TLC), haemoglobin concentration, platelet count, and peripheral blood blast percentage. Bone marrow examination findings were documented, and cases were segregated with morphological classification according to French-American-British (FAB) AML subtype criteria.

For molecular analysis, 5 mL of EDTA-anticoagulated bone marrow aspirate was obtained from each patient at the time of diagnosis. Genomic DNA was extracted using the QIAamp DNA Blood Mini Kit according to the manufacturer's instructions. *WT1* mutation screening targeted exons 7 and

9, which harbour the majority of clinically significant mutations. Real-time fluorescent qualitative Polymerase Chain Reaction (PCR) utilised specific primers targeting *WT1* mutation on the ABI-7500 real-time PCR system to amplify these critical regions. Each PCR run included positive controls, negative controls, and internal quality control samples. PCR results stratified patients into *WT1*-positive and *WT1*-negative groups.

All patients received standardised induction therapy according to the institutional protocol. After the completion of induction therapy, bone marrow aspiration was repeated to assess remission status. CR required bone marrow blasts <5%, absolute neutrophil count >1x10⁹/L, and platelet count >100x10⁹/L.

Data entry utilised Microsoft Excel with analysis through the Statistical Package for Social Sciences (SPSS) version 26.0. Normality of quantitative data was assessed using the Shapiro-Wilk test. Continuous variables were expressed as mean \pm standard deviation (SD), while categorical variables were presented as frequencies and percentages. An independent t-test was utilised to compare the two variables. Chi-square testing determined associations between *WT1* mutation status and clinical-haematological parameters, encompassing TLC, haemoglobin, platelet count, blast percentage, and FAB subtype. The odds ratio (OR) with 95% confidence interval (CI) was calculated using a cross-tabulation analysis. Statistical significance was set at $p \leq 0.05$.

RESULTS

The study cohort included 98 patients diagnosed with *de novo* AML who met the inclusion criteria. The mean age at diagnosis was 36.5 years, with ages spanning from 7 to 68 years, reflecting a relatively young patient population. Gender distribution revealed a slight male predominance, with 54 (55.1%) males and 44 (44.9%) females, thereby a male-to-female ratio of approximately 1.22:1.

Molecular analysis revealed *WT1* gene mutations in 12 (12.2%) patients, while the remaining 86 patients (87.8%) harboured wild-type *WT1*. Comparative analysis of baseline haematological parameters between *WT1*-mutated and wild-type patients revealed clinically relevant differences, although statistical significance was not achieved for all the variables (Table I). Patients with *WT1* mutations exhibited tendencies toward more aggressive disease characteristics, manifested by reduced haemoglobin concentrations (mean 7.7 \pm 0.5 g/dL vs. 8.7 \pm 0.9 g/dL, $p = 0.001$), raised TLC (mean 46.4 \pm 13.2 $\times 10^9$ /L vs. 35.3 \pm 11.4 $\times 10^9$ /L, $p = 0.003$), diminished platelet counts (mean 38.8 \pm 12.9 $\times 10^9$ /L vs. 55.3 \pm 17.0 $\times 10^9$ /L, $p = 0.002$), and elevated bone marrow blast proportions (mean 73.0 \pm 5.7% vs. 62.0 \pm 7.3%, $p = 0.001$).

Table I: Baseline clinico-haematological characteristics of AML patients stratified by WT1 mutation status.

Characteristics	WT1-mutated (n = 12)	WT1-wild type (n = 86)	p-values*
Mean age, years	34.6 ± 18.1	36.7 ± 17.4	0.697
Male gender, n (%)	7 (13.0)	47 (87.0)	0.813
Female gender, n (%)	5 (11.4)	39 (88.6)	0.819
Mean haemoglobin, g/dL	7.7 ± 0.5	8.7 ± 0.9	0.001
Mean total leucocyte count, ×10 ⁹ /L	46.4 ± 13.2	35.3 ± 11.4	0.003
Mean platelet count, ×10 ⁹ /L	38.8 ± 12.9	55.3 ± 17.0	0.002
Mean bone marrow blasts, %	73.0 ± 5.7	62.0 ± 7.3	0.001

*Independent samples t-test. WT1: Wilms' tumour 1.

Table II: Distribution of FAB morphological subtypes according to WT1 mutation status.

FAB subtypes	WT1-mutated (n = 12) n (%)	WT1-wild type (n = 86) n (%)
M0 (Minimally differentiated)	0 (0.0)	8 (9.3)
M1 (Without maturation)	5 (41.7)	18 (20.9)
M2 (With maturation)	6 (50.0)	22 (25.6)
M3 (Acute promyelocytic)	0 (0.0)	12 (14.0)
M4 (Acute myelomonocytic)	1 (8.3)	15 (17.4)
M5 (Acute monocytic)	0 (0.0)	8 (9.3)
M6 (Acute erythroid)	0 (0.0)	2 (2.3)
M7 (Acute megakaryoblastic)	0 (0.0)	1 (1.2)

FAB: French-American-British; WT1: Wilms' tumour 1.

Table III: Induction chemotherapy response according to WT1 mutation status.

Response category	WT1-mutated (n = 12) n (%)	WT1-wild type (n = 86) n (%)	OR (95% CI)	p-values*
CR	9 (75.0)	54 (62.8)	0.84 (0.58-1.21)	0.408
Refractory disease	3 (25.0)	32 (37.2)	1.48 (0.54-4.12)	0.408

*Chi-square test. CR: Complete remission; WT1: Wilms' tumour 1; OR: Odds ratio.

According to the FAB classification criteria, the distribution of AML subtypes differed between WT1-mutated and wild-type patients (Table II). WT1 mutations were predominantly associated with morphologically less differentiated subtypes, particularly M1 (acute myeloblastic leukaemia without maturation) and M2 (acute myeloblastic leukaemia with maturation). In contrast, the wild-type cohort showed a more heterogeneous distribution across various FAB subtypes.

Treatment response to standard induction chemotherapy represented a critical endpoint for assessing the prognostic impact of WT1 mutations (Table III). CR was achieved in 9 out of 12 WT1-mutated patients (75.0%), compared to 54 out of 86 wild-type patients (62.8%).

On the contrary, refractory disease was observed in 3 out of 12 WT1-mutated patients (25.0%), compared to 32 of 86 wild-type patients (62.8%).

The OR for achieving CR in WT1-mutated patients was 0.84 (95% CI: 0.58-1.21), indicating approximately half the odds of remission compared to wild-type patients, although CIs crossed unity. Conversely, the OR for refractory disease was 1.48 (95% CI: 0.54-4.12), suggesting nearly twice the odds of treatment failure, although without statistical significance.

DISCUSSION

The prognostic impact of WT1 mutations in AML has generated extensive research interest, with contradictory results emerging across numerous studies. In this study, the

observed trends suggested an association between WT1 mutations and adverse clinical outcomes, although the differences did not reach statistical significance, potentially due to the limited sample size.

This study investigated the prognostic significance of WT1 gene mutation among 98 patients with *de novo* acute myeloid leukaemia, revealing a mutation frequency of 12.2%. This frequency aligns closely with recent large-scale genomic studies. Bhatnagar *et al.* reported WT1 mutations in approximately 5-12% of adults with AML in their Alliance for Clinical Trials in Oncology study, demonstrating remarkable consistency across diverse patient populations.¹² Similarly, a thorough 2024 review by Debnath *et al.* documented WT1 mutation frequencies of approximately 10% in adult AML cohorts across multiple studies.¹³ These concordant findings validate the molecular screening methodology of the study and confirm that WT1 mutations, while relatively uncommon, represent a recurrent genetic aberration in AML pathogenesis.

The data revealed that WT1-mutated patients presented with markedly more aggressive haematological parameters at diagnosis. These cases demonstrated notably reduced haemoglobin levels (7.7 vs. 8.7 g/dL, $p = 0.001$), substantially elevated TLC (46.4 vs. 35.3 × 10⁹/L, $p = 0.003$), diminished platelet counts (38.8 vs. 55.3 × 10⁹/L, $p = 0.002$), and considerably elevated bone marrow blast proportions (73.0% vs. 62.0%, $p = 0.001$). Goel *et al.* demonstrated through using RNA sequencing that elevated WT1 expression at diagnosis

was associated with worse haematological parameters and adverse outcomes in AML patients.¹⁴ Furthermore, Baranwal *et al.* in their 2024 transplantation study reported that *WT1*-mutated patients consistently exhibited more aggressive disease features, including higher blast counts and reduced platelet levels at presentation.¹⁵ The biological basis for these aggressive features likely relates to *WT1*'s critical function in regulating haematopoietic stem cell self-renewal and differentiation. When loss-of-function mutations occur, they appear to promote uncontrolled proliferation while simultaneously impairing normal maturation pathways.¹⁶

When examining treatment outcomes, this study revealed that 9 (75%) of *WT1*-mutated patients achieved CR, compared with 54 (62.8%) of wild-type patients, yielding an OR of 0.84 (95% CI: 0.58-1.21). The limited number of *WT1*-mutated patients ($n = 12$) necessitates cautious interpretation of treatment response data. While trends were observed, the study was underpowered to definitively establish significant differences in clinical outcomes. The CIs for ORs are consequently wide, reflecting this statistical uncertainty. The trend toward lower remission rates in the *WT1*-mutated group is consistent with several recent investigations. Wang *et al.*, in their comprehensive 2021 analysis, reported that *WT1*-mutated patients had significantly lower CR rates following standard induction chemotherapy, particularly when co-occurring mutations were present.¹⁷ Similarly, Yu T *et al.* demonstrated in their 2023 study that high *WT1* expression in non-M3 AML was associated with reduced rates of CR and higher relapse rates.¹⁸ It is worth noting that the modest effect size observed in the study may reflect the relatively small number of *WT1*-mutated patients ($n = 12$), which inherently limits the statistical power to detect significant differences even when clinically meaningful trends exist.

Interestingly, the observed refractory disease rate of 3 (25.0%) in *WT1*-mutated patients *versus* 32 (37.2%) in wild-type patients (OR 1.48, 95% CI: 0.54-4.12) diverges somewhat from several previous reports. Atluri *et al.*, in their 2023 Alliance study, found that *WT1*-mutated patients with concurrent *FLT3*-ITD mutations had significantly higher treatment failure rates and markedly inferior survival outcomes compared to those without *FLT3*-ITD.¹⁹ However, the findings of the study are also supported by local genomic data from Pakistan, where Shahid *et al.* reported substantial genetic heterogeneity in AML, emphasising the need for population-specific molecular characterisation of prognostic markers, including *WT1*.²⁰ This finding highlights an important point: the absence of statistical significance in refractory disease analysis may be explained by the complex genetic architecture of AML. In reality, *WT1* mutations rarely occur in isolation, and their prognostic impact appears highly context-dependent, influenced by the specific constellation of accompanying genetic alterations present in each patient.

It is important to acknowledge that the prognostic significance of *WT1* mutations remains somewhat debatable in contemporary AML research. While this study and several

others suggest adverse associations, the picture is far from clear-cut. Similar findings were reported by Tien *et al.*, who showed that concomitant *WT1* mutations adversely affected prognosis in specific molecular subgroups of AML, particularly in patients with double-mutant CEBPA.²¹ Burd *et al.*, in the landmark Beat AML Master Trial, demonstrated that dominant *WT1* mutations (those with a variant allele fraction ≥ 0.30) had distinctly different clinical implications compared to subclonal mutations. This suggests that simply categorising patients as *WT1*-mutated or wild-type may be an oversimplification that fails to capture the underlying biological complexity.²²

This study was carried out at a single tertiary care centre with a modest sample size, which may restrict the extent to which the findings can be generalised to broader populations. Follow-up duration was short, restricting assessment of long-term survival and relapse outcomes. Extended molecular profiling, including co-mutation analysis, was not performed, which could have provided deeper insights into the prognostic effect of *WT1* mutations. Additionally, treatment response was evaluated after only one induction cycle, without long-term monitoring of minimal residual disease.

CONCLUSION

Mutation of *WT1* in AML is associated with distinct clinical and morphological characteristics, including more aggressive disease at presentation and a predominance in less differentiated FAB subtypes (M1 and M2). However, the limited sample size and absence of long-term outcome data prevent definitive conclusions about their independent prognostic value. Future prospective studies with comprehensive molecular profiling—evaluating co-mutations, allelic burden, and mutation sites—are needed to clarify the independent prognostic role of *WT1* mutations. Such research will enhance understanding of disease biology and support more individualised therapeutic approaches for AML patients.

ETHICAL APPROVAL:

Ethical approval was obtained from the Institutional Review Board of Armed Forces Institute of Pathology, Rawalpindi, Pakistan (Ref. No. FC-HEM22-17/READ-IRB/23/2189).

PATIENTS' CONSENT:

Informed consent of the patients was obtained before the initiation of the study.

COMPETING INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

MA: Conception and design of the study; acquisition, analysis, and interpretation of data.

MB, SF, RM, TA: Conception and design of the study; critical review of the manuscript for important intellectual content.

HK: Acquisition, analysis, and interpretation of data.

All authors approved the final version of the manuscript to be published.

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