A Single Institution Experience on Clinical Outcomes of *TP-53* Mutant Acute Myeloid Leukemia

Binoy Yohannan, Frances Cervoni-Curet, Adan Rios
Division of Hematology and Oncology
McGovern Medical School, The University of Texas Health Science Center at Houston

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Background

- *TP 53* regulates DNA repair and is known as the guardian of the genome.
- Mutations in *TP-53* gene are observed in up to 20% of newly diagnosed AML and more commonly in secondary AML.
- *TP53*-mutated AML has a poor response to conventional dose intense chemotherapy with a median overall survival (OS) of 4 to 6 months.

Materials and Methods

- We retrospectively reviewed all cases of *TP53* mutant AML treated at Memorial Hermann Hospital, Texas Medical Center between January 1, 2013, and June 30, 2022.
- Baseline demographic variables, cytogenetics, induction regimen and survival data were obtained from electronic medical records.

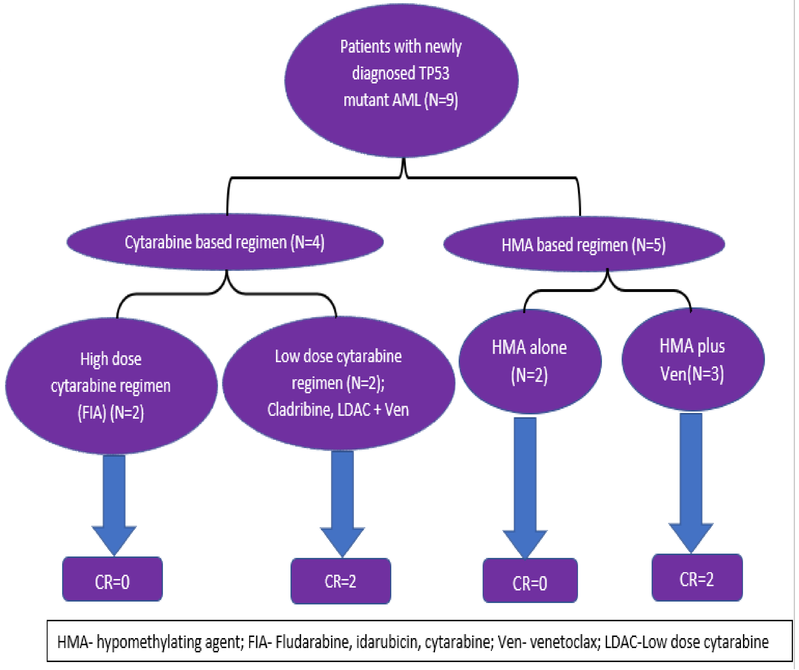
Results

- Among 130 patients with AML, we identified 9 patients (6.9%) with TP 53 mutation.

Table 1: clinical characteristics of patients with P 53 mutant A

Characteristics	Median	Range	N/%
Age, years	66	41–80	
Subject races			
Whites			2 (22)
African Americans			5 (55)
Hispanics			2 (22)
WBC, × 10 ^{9}/L	2.8	3–71	
Platelets, × 10 ^{9}/L	43	34–72	
BM blasts, %	50	1–77	
De novo AML			5 (55)
Secondary AML			4 (44)
Cytogenetics			
Complex			8 (88)
Insufficient metaphases			1
Co-occurring mutations			
DNMT3A			1
FLT3-ITD			2
TET2			1
ASXL1			2
IDH1			2
U2AF1			1
Early deaths			
Death<30 days			1 (11%)
Death<60 days			2 (22%)

Results



HMA- hypomethylating agent; FIA- Fludarabine, idarubicin, cytarabine; Ven- venetoclax; LDAC-Low dose cytarabine

- None received allogenic stem cell transplant (ASCT) in CR 1, but one patient underwent ASCT in CR 2 and died 2 months later from graft failure.
- The median OS in the entire cohort was 180 days (95% CI. 39.0-393.0 days).

Discussion

- *TP53* mutant AML is frequently seen in older age and associated with complex cytogenetics.
- *TP 53* mutations are enriched in secondary AML and the median OS is less than 6 months.
- Patients with *TP53* mutant AML treated with standard anthracycline and cytarabine based regimens have poor outcome.
- *TP53* mutant status provides important prognostic information and may be used to select the induction regimen.

Conclusion

- Our study confirms the poor outcome of patients with *TP 53* mutant AML.
- *TP 53* mutant AML has a poor response to dose intense chemotherapy regimens.
- LDAC or HMA based regimens are well tolerated and can be used as an effective bridging regimen to ASCT.

References

1.Tim Grob,et al ; Molecular characterization of mutant TP53 acute myeloid leukemia and high-risk myelodysplastic syndrome. Blood 2022; 139 (15): 2347–2354