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A CASE REPORT: ACUTE MYELOID LEUKEMIA

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ABSTRACT

Acute myeloid leukemia (AML) is a complex blood cancer marked by a range of genetic and epigenetic alterations that impact the risk of patients and their responses to treatment. As we acquire more insights from detailed genetic research, it becomes evident that a comprehensive profile of emerging mutations is vital for personalizing therapy and enhancing patient outcomes. The articles under review emphasize how certain mutations, particularly TET2, ETV6, SATB1, EZH2, PTPN11, and U2AF1, influence prognosis, resistance to therapies, and the development of targeted and epigenetic treatments. Focusing on regulators affect clinical behaviour, including DNA hypermethylation and chemotherapy resistance. Additionally, disruptions in ETV6 provide valuable insights into the alterations in blood cell production processes.

INTRODUCTION

Acute myeloid leukemia (AML) is a genetically and epigenetically diverse haematological malignancy that arises from the transformation of hematopoietic stem cells. Across all age groups, AML exhibits a spectrum of diagnostic and prognostic markers, including recurring chromosomal abnormalities, such as t (8;21) AML1-ETO, t (15;17) PML-RARA, and t (16;16) CBFB-MYH11, along with mutations in FLT3-ITD, CEBPA, KIT, NPM1, and ASXL1. The field is increasingly recognizing the value of advanced molecular approaches, such as chromosomal microarray analysis (CMA) and next-generation sequencing (NGS), in refining risk stratification and guiding therapy. The disease is often framed through the "two-hit" hypothesis, wherein Class I lesions drive proliferation and survival, and Class II lesions impede differentiation, shaping both disease biology and therapeutic targeting. Paediatric AML (p-AML) displays more frequent and distinct cytogenetic abnormalities and epigenetic differences than adult AML, underscoring the need to consider age-specific disease mechanisms and treatment responses.[1] A parallel emphasis in the literature is on the role of epigenetic regulation in AML pathogenesis and therapy. Epigenetic modifications, including DNA methylation, histone modifications, and noncoding RNAs, affect hematopoietic differentiation and are frequently dysregulated in AML. This epigenetic landscape complements canonical genetic alterations and informs both prognosis and therapeutic avenues, particularly in paediatric cohorts where epigenetic profiles show notable differences from adults.[2] Building on this foundation, current and emerging therapeutic strategies are increasingly targeting leukaemia stem cells (LSCs), which are implicated in disease initiation, maintenance, and chemoresistance. Evidence suggests that AML can originate from leukaemia-initiating populations of LSCs, and therapeutic approaches aiming to eradicate these cells are promising in preclinical and early clinical contexts. Notably, strategies targeting telomerase activity, such as the telomerase inhibitor imetelstat, are being explored in trials, reflecting a broader shift toward targeting the fundamental vulnerabilities of LSCs. These innovations emphasize immunotherapeutic modalities and novel agents that exploit LSC biology and are applicable to paediatrics and adult AML.[3]

CASE STUDY

A 25-year-old male patient was admitted in the hospital with the c/o fatigue, generalized weakness for 2 weeks easy bruising since 1-week, low grade fever and night sweat for 5 days, mild shortness of breath on excretion.

The patient was suffering from a known case of type-2 diabetes mellitus and hypertension for 4 years and the medications using are tablet xylomet 500mg (BD), Telma 40mg (OD)

The patient's social history was he was a smoker for years i.e.20 packs/day.

HAEMATOLOGICAL PATTERNS

Parameters	Tested values	Normal values
Hb (g/dl)	7.8	13-17
RBC (millions/cumm)	3.2	4.0-5.5
WBC (cells/cumm)	4.8	4000-10000
Eosinophils	0	1-6
Hbalc (%)	7.8	3-5
Uric acid (mg%)	9.2	2.6-7.2
LDH micron/lt	620	140-280

Other investigations
Peripheral smear-blast cells – 32%
USG abdomen – splenomegaly, reticulocyte – low

Bone marrow biopsy – hypercellular >20% myeloblast
Cytogenetics – FLT3 – ITD mutation (+)

TREATMENT PLAN

TRADE NAME	GENERIC NAME	DOSE	ROA	FRQ	INDICATION
1.INJ.CYTOSAR	Cytarabine	100mg	IV	-	Drugs of choice for aml
2.INJ.CERUBIDI-NE	Daunorubicin	60mg	IV	OD	Inhibits topoisomerase 2 &DNA, RNA synthesis
3.TAB.ZYLORIC	allopurinol	300mg	PO	OD	It decreases the level of uric acid
4.INJ.EMESET	Ondansetron	8mg	IV	SOS	Reduces the episode of vomiting caused by chemotherapy
5.TAB.TAXIM-U	Cefixime	200mg	PO	BD	Decreases the infection as the levels of WBC'S are high
6.INJ.ZOSYN	Piperacillin+ Tazobactam	4.5mg	IV	BD	Decreases the severity of infection
7.CAP.STAURINZ	Midostaurin	25mg	PO	BD	Decreases the rate of mutations
8.INJ.PANTOP	pantoprazole	40mg	IV	OD	Decrease the acid reflex caused by other drugs

DISCUSSION

In my case study a 25-year-old patient was diagnosed with acute myeloid leukemia, and we give following medication to decrease the symptoms and the medications prescribed were:
INJ CYTOSAR is a drug of choice for aml.
INJ.CERUBIDINE: Inhibits topoisomerase 2 & NA, RNA synthesis.
TAB.ZYLORIC: It decreases the level of uric acid.
CAP.STAURINZ: Decreases the rate of mutations.

CONCLUSION

The patient was a 25-year-old male with a medical history of type 2 Diabetes Mellitus and Hypertension, who was diagnosed with Acute Myeloid Leukemia (AML) – FLT3-ITD positive, based on clinical symptoms, blood tests, and a bone marrow biopsy. The clinical manifestations of fatigue, weakness, easy bruising, fever, and night sweats are typical of bone marrow failure and leukemic cell proliferation. Laboratory tests indicated anemia, decreased RBC count, increased LDH level, hyperuricemia, and the presence of blast cells, confirming active malignant cell turnover. FLT3-ITD mutations are associated with poor prognosis, as they facilitate the rapid growth of leukemic cells and increase the risk of relapse. Nevertheless, incorporating midostaurin (an FLT3 inhibitor) into the treatment plan significantly enhances therapeutic efficacy by directly targeting the molecular mutation. The chemotherapy regimen consisting of cytarabine and daunorubicin embodies the standard "7+3" induction therapy, which seeks to achieve complete remission by eliminating leukemic blasts. Supportive measures, such as allopurinol, mitigate the risk of tumor lysis syndrome, whereas antibiotics and antiemetics assist in managing the infection risks and side effects of chemotherapy. Given the patient's existing conditions (DM and HTN), it is essential to closely monitor blood glucose levels, renal function, and infection status throughout chemotherapy. Strong encouragement for smoking cessation is advised to improve recovery and minimize complications of lung surgery.

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