LABORATORY REPORT

Reg. No	:	**60220300069**	Reg. Date	:	18-Feb-2026 12:38
Name	:	**ADARSH JAIN**	Collection on	:	17-Feb-2026 12:38
Sex/Age	:	**Male / 43 Years**	Report Date	:	18-Feb-2026 19:24
Ref. By	:	Dr. Sachin Bansal DNB Hem (Hemato Onco)	Tele. No	:	
Location	:	SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL	Dispatch At	:	

BONE MARROW ASPIRATION SLIDES FOR REVIEW

Received unstained bone marrow aspirate smears for reporting.

Significant clinical history: C/O dyspnoea on exertion for 20 days found to have anaemia and leukopenia, no hepatosplenomegaly. ? Acute leukaemia.

CBC & Peripheral smear: Hb: 8.5gm/dl, WBC: 980/cmm, Platelet: 2,51,000/cmm Differential P: 11%, L: 58%, M: 05%, Blasts: 26%, Occasional nRBC seen.

Cellularity of fragments: Fragments are absent.

Cell trails: Cellular

Trephine imprints: Cellular

Morphology: Cellular trephine imprint shows normal haematopoiesis is suppressed by 90% blasts. These blasts are small in size, large round nucleus, fine nuclear chromatin, an inconspicuous nucleolus, scanty pale blue cytoplasm.

Very occasional erythroid and myeloid precursors are seen.

Megakaryocytes are not seen.

Impression: **Acute leukaemia.**

Trephine biopsy, Immunophenotyping and cytogenetic study is pending at this point in time.

*** End of report ***

Note: Stained and unstained slides are returned with report. Please preserve them carefully.

Dr. Avinash B Panchal
MBBS,DCP
G-44623

TEST REPORT

Reg. No.	: 60220300069	**Reg. Date** : 18-Feb-2026 12:38	**Ref.No** :	**Approved On**	: 18-Feb-2026 14:11
Name	: ADARSH JAIN			**Collected On**	: 17-Feb-2026 12:38
Age	: 43 Years	**Gender:** Male	**Pass. No. :**	**Dispatch At**	:
Ref. By	: Dr. Sachin Bansal DNB Hem (Hemato Onco)			**Tele No.**	:
Location	: SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL				

Test Name	Results	Units	Bio. Ref. Interval
Lactate Dehydrogenase (LDH)	239.00	U/L	4 - 20 dys :225 - 600 20 dys-11 month :180-435 1-3 years : 160-370 4 - 15 yrs : 120 - 300 > 15 yrs :135 - 214

Lactate To Pyruvate Uv-IFCC

Sample Type: Serum
Lactate dehydrogenase (LDH) activity is present in all cells of the body with the highest concentrations in the heart, liver, muscle, kidney, lung, and erythrocytes. Appearance of LDH in the serum occurs only after prolonged hypoxia and is elevated in a number of clinical conditions including cardiorespiratory diseases, malignancy, hemolysis, and disorders of the liver, kidneys, lung, and muscle. For fluid, LDH may be used to differentiate transudative from exudative effusions. Lactate dehydrogenase (LDH) activity is one of the most sensitive indicators of in vitro hemolysis. Causes can include transportation via pneumatic tube and vigorous mixing. Contamination with erythrocytes will falsely elevate results, because the analyte level in erythrocytes is higher than in normal sera.

This is an electronically authenticated report."Please verify the authenticity of this report by scanning the QR code to ensure data integrity."

Test done from collected sample.

Approved by:

Dr. Rina Prajapati
D.C.P. DNB (Path)
G-21793

Generated On : 06-Mar-2026 15:50

TEST REPORT

Reg. No.	: 60220300069	**Reg. Date** : 18-Feb-2026 12:38	**Ref.No** :	**Approved On**	: 18-Feb-2026 14:54
Name	: ADARSH JAIN			**Collected On**	: 17-Feb-2026 12:38
Age	: 43 Years	**Gender:** Male	**Pass. No. :**	**Dispatch At**	:
Ref. By	: Dr. Sachin Bansal DNB Hem (Hemato Onco)			**Tele No.**	:
Location	: SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL				

Test Name	Results	Units	Bio. Ref. Interval
Hepatitis B surface Antigen (HBsAg)	0.28	S/Co	<1.0 : Negative, >=1.0 : Positive

CMIA

Sample Type: Serum

Hepatitis B surface antigen (HBsAg) is the first serologic marker appearing in the serum at 6 to 16 weeks following HBV infection. In acute infection, HBsAg usually disappears in 1 to 2 months after the onset of symptoms. Persistence of HBsAg for greater than 6 months indicates development of either a chronic carrier or chronic HBV infection. A positive result is indicative of acute or chronic hepatitis B virus (HBV) infection, or chronic HBV carrier state. The presence of HBsAg is frequently associated with HBV infectivity, especially when accompanied by the presence of hepatitis Be antigen or HBV DNA.

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Test done from collected sample.

Approved by:

Dr. Razvin Somani

M.D. Pathology
Reg. No.:-G-51211

Generated On : 06-Mar-2026 15:50

TEST REPORT

Reg. No.	: 60220300069	**Reg. Date** : 18-Feb-2026 12:38	**Ref.No** :	**Approved On**	: 18-Feb-2026 15:06
Name	: ADARSH JAIN			**Collected On**	: 17-Feb-2026 12:38
Age	: 43 Years	**Gender:** Male	**Pass. No. :**	**Dispatch At**	:
Ref. By	: Dr. Sachin Bansal DNB Hem (Hemato Onco)			**Tele No.**	:
Location	: SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL				

Test Name	Results	Units	Bio. Ref. Interval
ANTI HCV	0.11	S/Co	Non Reactive <1.0 Reactive >=1.0

CLIA

Sample Type: Serum

1. The HCV Antibodies assay is a screening test used for qualitative detection of antibodies to Hepatitis C Virus infection in human serum or plasma.
2. The test result should be correlated clinically and in relation with other diagnostic markers for HCV infection.
3. A negative test does not exclude the possibility of exposure to or infection with HCV virus.
4. Patients with auto-immune liver disease may show falsely reactive results.
5. The test has a Sensitivity of 100% and Specificity of 99.63%.

Limitations of the test:

1) Heterophile antibodies in human serum can react with reagent immunoglobulins, interfering with in-vitro immunoassays.

This is an electronically authenticated report."Please verify the authenticity of this report by scanning the QR code to ensure data integrity."

Test done from collected sample.

Approved by:

Dr. Razvin Somani

M.D. Pathology
Reg. No.:-G-51211

Generated On : 06-Mar-2026 15:50

TEST REPORT

Reg. No.	: 60220300069	**Reg. Date** : 18-Feb-2026 12:38	**Ref.No** :	**Approved On**	: 18-Feb-2026 15:06
Name	: ADARSH JAIN			**Collected On**	: 17-Feb-2026 12:38
Age	: 43 Years	**Gender:** Male	**Pass. No. :**	**Dispatch At**	:
Ref. By	: Dr. Sachin Bansal DNB Hem (Hemato Onco)			**Tele No.**	:
Location	: SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL				

Test Name	Results	Units	Bio. Ref. Interval
Human Immunodeficiency Virus (HIV I & II Ag/Ab Combo)	0.07	Index	Non reactive : <1.0, Reactive : >=1.0
CMIA			

Sample Type: Serum

Notes:

1) A NON REACTIVE result implies that no Anti HIV-1 or HIV-2 antibodies have been detected in the sample by this method. This means that either the patient has not been exposed to HIV-1 or HIV-2 infection or the sample has been tested during the " Window phase" (before the development of detectable levels of antibodies).

2) A REACTIVE result suggests the possibility of HIV-1 or /and HIV-2 infection. However these results must be verified by a confirmatory WESTERN BLOT method before declaring the patient positive for HIV-1 or HIV-2 infection.

3) Very high levels of IgM antibodies or anti-HLA A,B,C,DR antibodies can give false positive reaction.

4) Neonates born of HIV infected mothers may have HIV infection or can be uninfected despite the presence of maternal antibodies to HIV in their blood. Such neonates should undergo additional testing such as Polymerase chain reaction (PCR) to ascertain their status of infection.

5) Pre & post test counseling for HIV testing is responsibility of referring physician.

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Test done from collected sample.

Approved by:

Dr. Razvin Somani

M.D. Pathology
Reg. No.:-G-51211

Generated On : 06-Mar-2026 15:50

LABORATORY REPORT

Reg. No	:	**60220300069**	Reg. Date	:	18-Feb-2026 12:38
Name	:	**ADARSH JAIN**	Collection on	:	17-Feb-2026 12:38
Sex/Age	:	**Male / 43 Years**	Report Date	:	26-Feb-2026 13:07
Ref. By	:	Dr. Sachin Bansal DNB Hem (Hemato Onco)	Tele. No	:	
Location	:	SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL	Dispatch At	:	

CYTOGENETIC REPORT

Sample Type : Heparinized bone marrow
Clinical Diagnosis : **IPT- S/O Near Early T cell precursor acute lymphoblastic leukemia**
Test performed : Chromosomal study

Chromosome Results *(As per cytogenetic nomenclature ISCN 2024)*

Modal Chromosome No.(s) : **45/47/46**

Modal Chromosome Karyotype : **45,XY,add(2)(q37),add(4)(q35),der(7)?inv(7)(p15q36),-15,del(16)(q12-13)[7]/ 47,XY,+4[1]/46,XY[12]**

Diagnostic Interpretation

Cytogenetic analysis revealed an abnormal male chromosome complement where eight (40%) out of twenty metaphases are abnormal. Seven (35%) metaphases showed 45 chromosome number with loss of chromosome 15, addition of unknown material on long (q) arms of chromosome 2, 4, derivative chromosome 7 possibly pericentric inversion of chromosome 7 and deletion of the long (q) arm of chromosome 16.
One (5%) metaphase showed gain of chromosome 4. However, clonality for trisomy 4 cannot be established as it is seen in a single metaphase only.
Advised correlation with clinical features, hematological findings & UNI NGS ALL panel for final risk stratification.

Laboratory Analysis :

Type of culture : Unstimulated overnight culture	**No. of culture(s) :** 02
No. of metaphase counted : 20	**Banding Resolution :** 350 – 400 BHPS
No. of metaphase analyzed : 20	**Banding method :** GTL method
No. of metaphase karyotyped : 20	

Dr Meenu Angi
MD Pathology,
PDF Cytogenetics,
Consultant Cytogenetics

Dr. Amisha Shah
M.D. Pathology
G-11265

LABORATORY REPORT

Reg. No	:	**60220300069**	Reg. Date	:	18-Feb-2026 12:38
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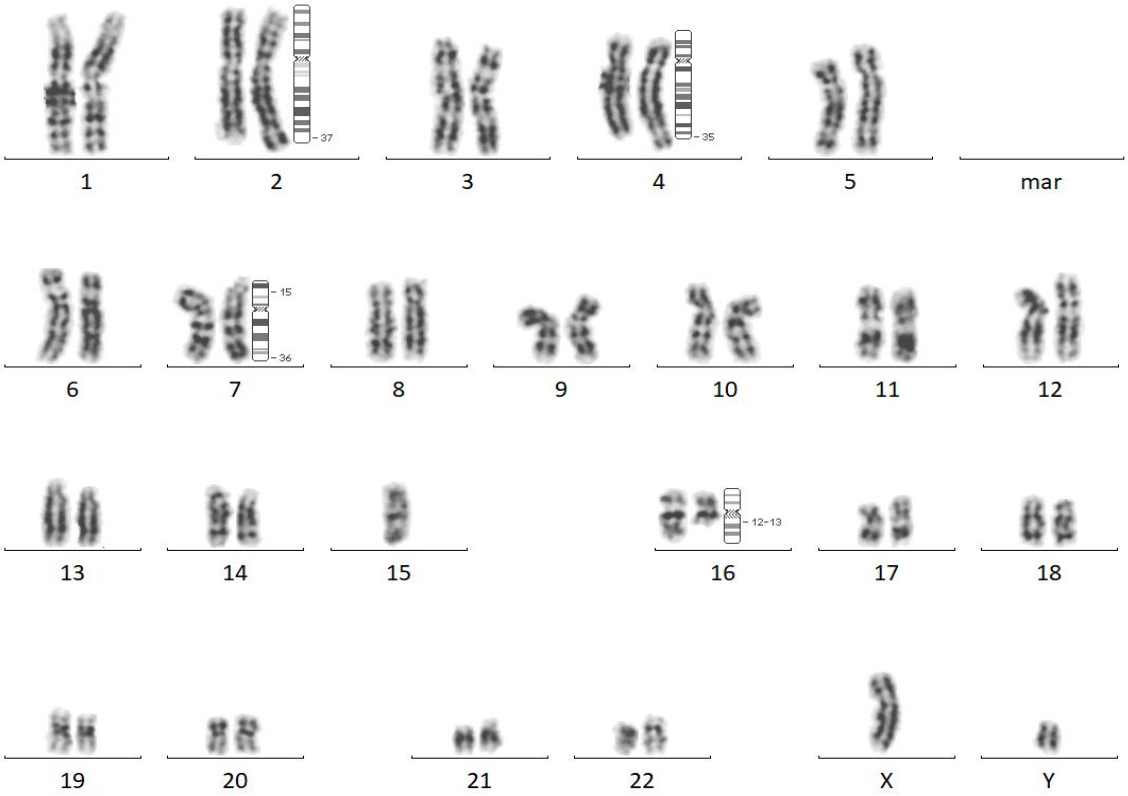
Location	:	SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL	Dispatch At	:	



Dr Meenu Angi
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PDF Cytogenetics,
Consultant Cytogenetics

Dr. Amisha Shah
M.D. Pathology
G-11265

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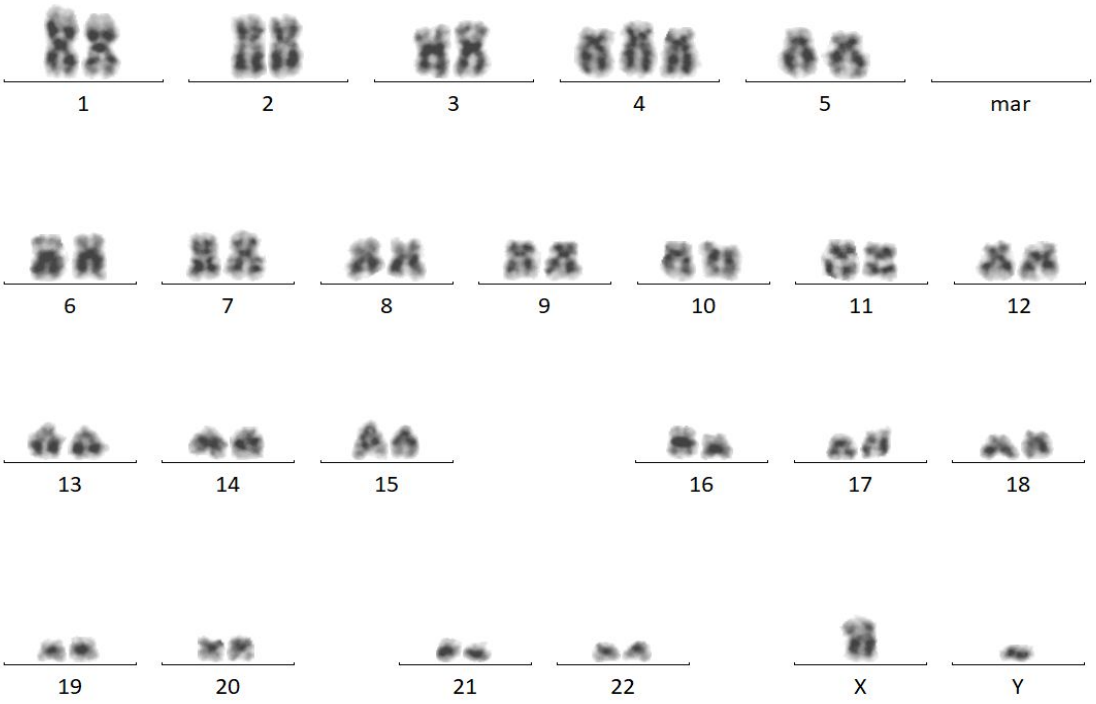

Dr Meenu Angi
MD Pathology,
PDF Cytogenetics,
Consultant Cytogenetics

Dr. Amisha Shah
M.D. Pathology
G-11265

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Location	:	SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL	Dispatch At	:	

IMMUNOPHENOTYPING REPORT
(Leukemia / lymphoma panel)

Clinical history: Acute leukemia
Specimen: Bone marrow
Method/ Instrument / Software: Flowcytometry / Beckman Coulter DxFLEX 10 color / Kaluza
Cell preparation method: Stain - Lyse - Wash
Gating strategy: CD45 Vs SSC

Result:

Marker	Intensity	Interpretation	Marker	Intensity	Interpretation
Myeloid / Mono			**T cell / NK cell**		
CD11b	**Moderate**	**Positive**	CD1a	-	Negative
CD117	**Variable**	**Positive**	**CD5**	**Moderate**	**Positive**
CD33	**Moderate**	**Positive**	sCD3	-	Negative
CD64	-	Negative	CD4	-	Negative
CD36	-	Negative	CD2	-	Negative
CD13	-	Negative	**CD7**	**Bright**	**Positive**
CD14	-	Negative	CD8	-	Negative
CD15	**Dim subset**	**Positive**	**cCD3**	**Moderate**	**Positive**
CD16	-	Negative	**CD56**	**Dim subset**	**Positive**
cMPO	-	Negative			
Other			**B cell**		
HLA-DR	-	Negative	CD10	-	Negative
CD34	**Moderate**	**Positive**	CD19	-	Negative
CD123	-	Negative	CD20	-	Negative
CD38	**Dim**	**Positive**	cCD79a	-	Negative
CD45	**Dim**	**Positive**			

Dr. Avinash B Panchal
MBBS,DCP
G-44623

LABORATORY REPORT

Reg. No	:	**60220300069**	Reg. Date	:	18-Feb-2026 12:38
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Location	:	SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL	Dispatch At	:	

Viability: 95%
Abnormal population: **84% T lineage lymphoblasts**
Size by forward scatter: Small
Side scatter: Low
Description: An abnormal cell population with dim CD45, low side scatter and small size by forward scatter seen. Among T cell markers these cells show positive expression of CD5, CD7, cCD3, dim subset CD56 and negative of CD1a, CD4, sCD3, CD8, CD2.
There is aberrant myeloid expression of CD11b, CD33, CD15 and CD117. CD34, CD38 are also positive. These findings are consistent with T lineage lymphoblasts.

Impression: Immunophenotyping findings of the Bone marrow sample are suggestive of **Near Early T cell precursor acute lymphoblastic leukemia.**

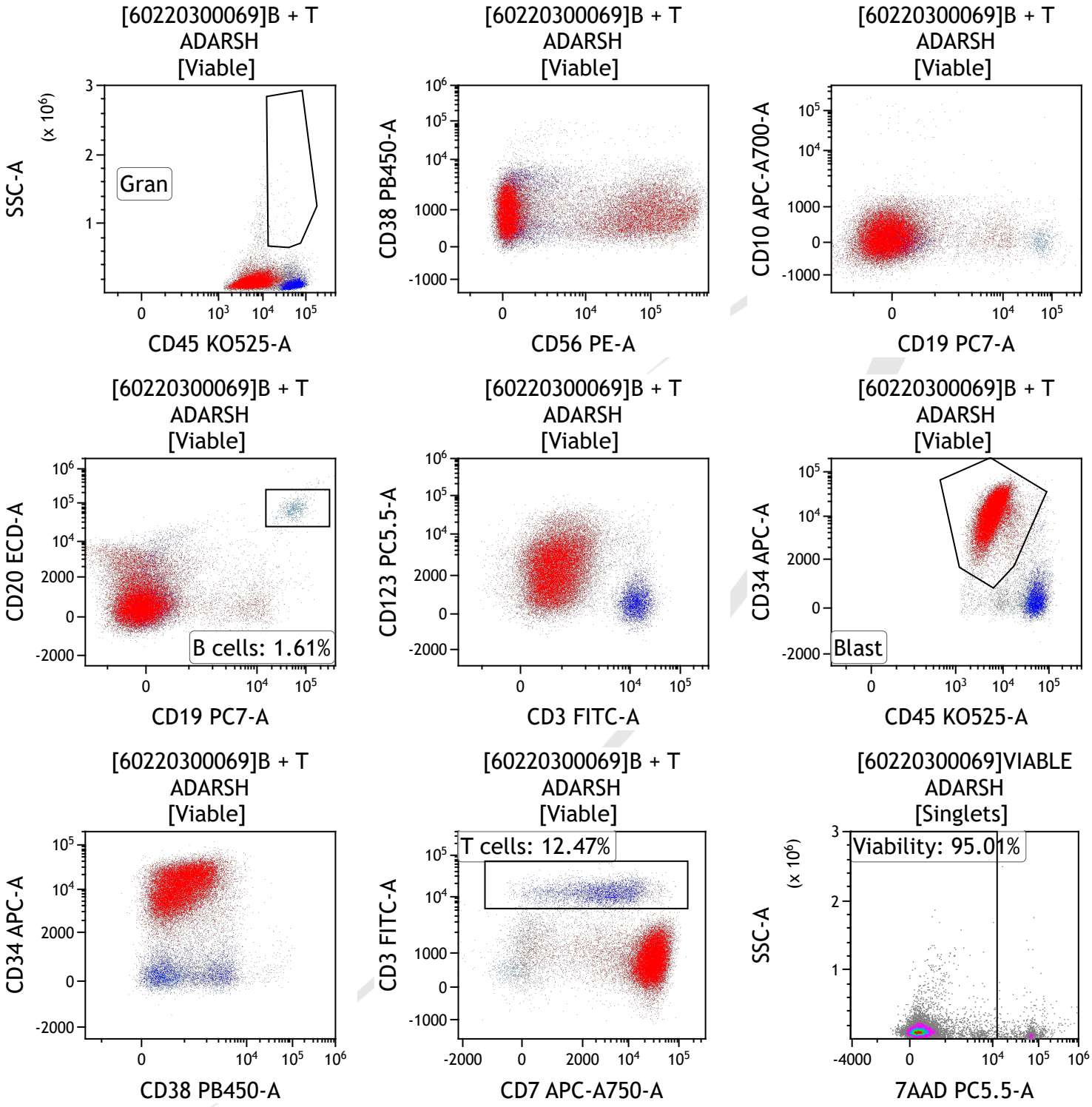
Aberrancy detected: CD11b, CD33, CD15, CD117

Advise: Cytogenetic studies.

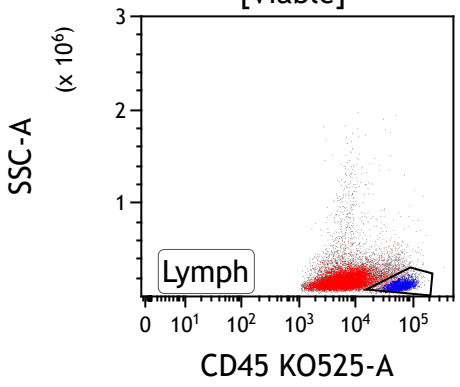
Note: Isolated flowcytometry analysis never confirm the final diagnosis of the disease. They only help in arriving at a diagnosis in conjunction with clinical presentation, cytogenetic study, molecular tests and other hematological parameters.

Prepared by:
Dr Aneeta Shahni
PhD (Hematology)

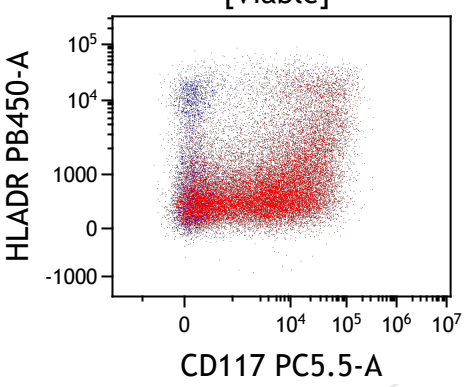
Dr. Avinash B Panchal
MBBS,DCP
G-44623


[60220300069]B + T
ADARSH
[Viable]
SSC-A
(x 10⁶)
Gran
CD45 KO525-A
[60220300069]B + T
ADARSH
[Viable]
CD38 PB450-A
CD56 PE-A
[60220300069]B + T
ADARSH
[Viable]
CD10 APC-A700-A
CD19 PC7-A
[60220300069]B + T
ADARSH
[Viable]
CD20 ECD-A
B cells: 1.61%
CD19 PC7-A
[60220300069]B + T
ADARSH
[Viable]
CD123 PC5.5-A
CD3 FITC-A
[60220300069]B + T
ADARSH
[Viable]
CD34 APC-A
Blast
CD45 KO525-A
[60220300069]B + T
ADARSH
[Viable]
CD34 APC-A
CD38 PB450-A
[60220300069]B + T
ADARSH
[Viable]
CD3 FITC-A
T cells: 12.47%
CD7 APC-A750-A
[60220300069]VIABLE
ADARSH
[Singlets]
SSC-A
(x 10⁶)
Viability: 95.01%
7AAD PC5.5-A

[60220300069]
MYELOID ADARSH
[Viable]



[60220300069]MYELOID
ADARSH
[Viable]



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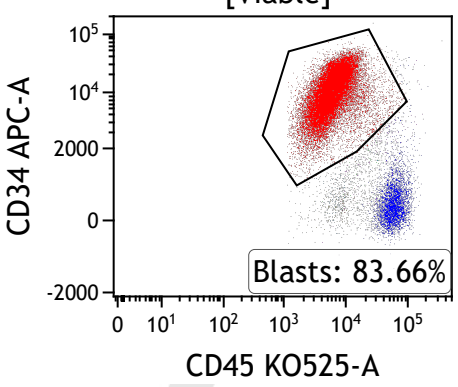
ADARSH
[Viable]



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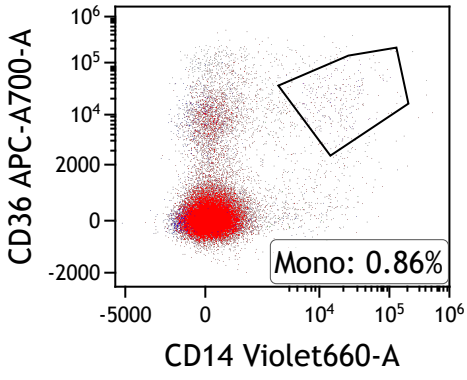
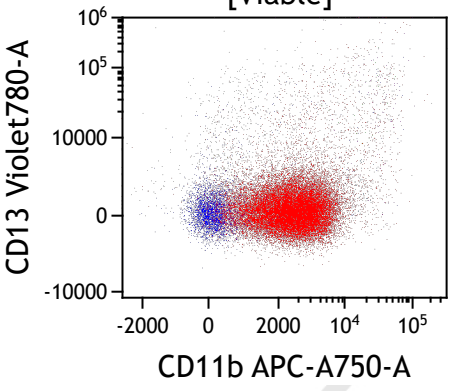
ADARSH
[Viable]



[60220300069]MYELOID
ADARSH
[Viable]



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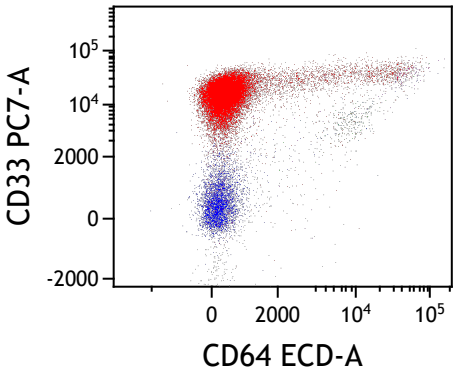
ADARSH
[Viable]



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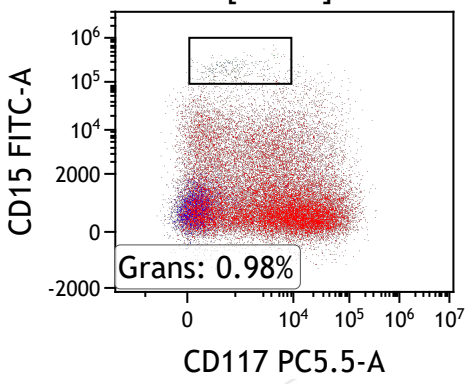

[60220300069]MYELOID
ADARSH
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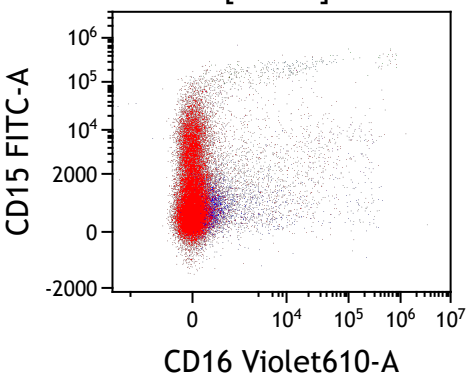
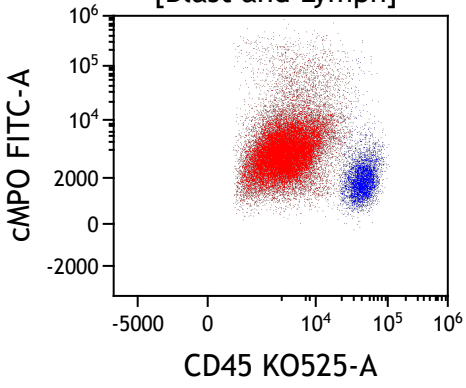
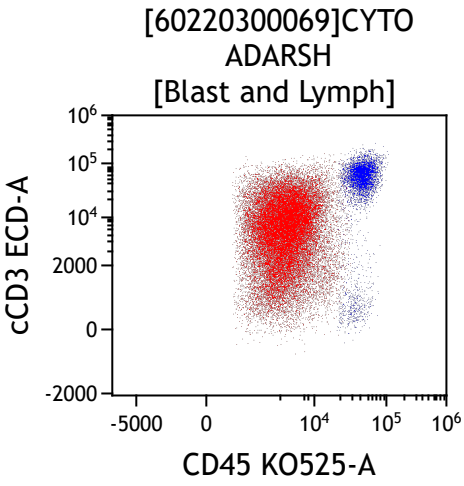
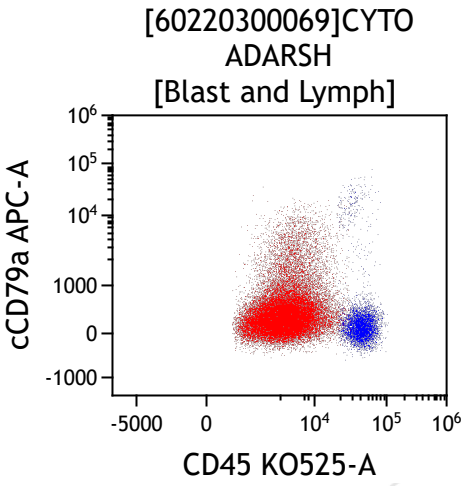


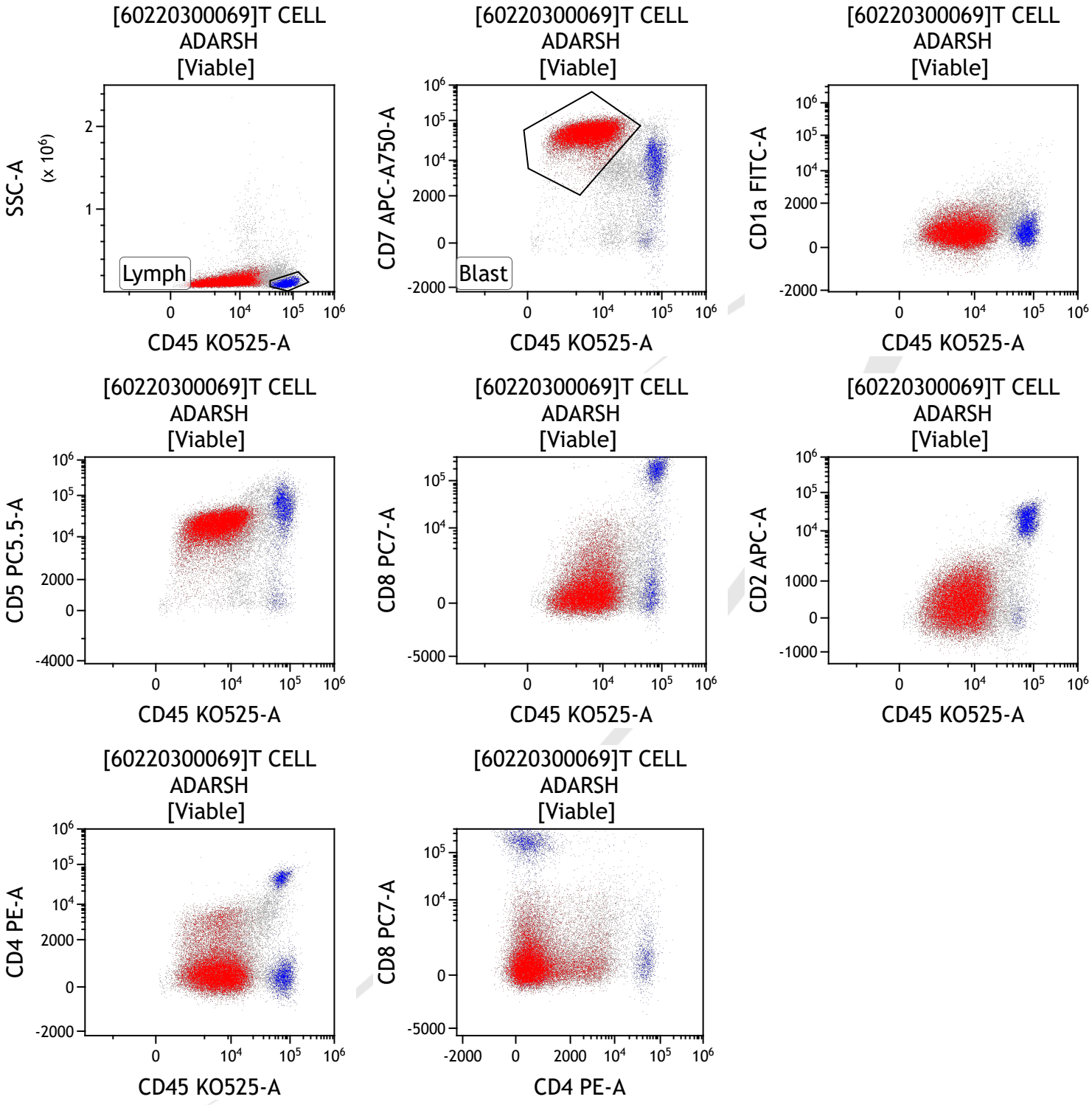
[60220300069]CYTO
ADARSH
[Blast and Lymph]



Lab Facility :Unipath House, Beside Sahjanand college, Opposite Kamdenu Complex, Panjarapole, Ambawadi, Ahmedabad-380015
Unigenome: 2A,3A,3B PASL -House, Beside Sahjanand college, Opposite Kamdenu Complex, Panjarapole, Ambawadi, Ahmedabad-380015,Gujarat
Phone: +91 -79-49006800,07699991171 | **Whatsapp:** 6356005900 | **Email:** info@unipath.in | **Website:** www.unipath.in






[60220300069]T CELL
ADARSH
[Viable]
SSC-A
(x 10⁶)
Lymph
CD45 KO525-A
[60220300069]T CELL
ADARSH
[Viable]
CD7 APC-A750-A
Blast
CD45 KO525-A
[60220300069]T CELL
ADARSH
[Viable]
CD1a FITC-A
CD45 KO525-A
[60220300069]T CELL
ADARSH
[Viable]
CD5 PC5.5-A
CD45 KO525-A
[60220300069]T CELL
ADARSH
[Viable]
CD8 PC7-A
CD45 KO525-A
[60220300069]T CELL
ADARSH
[Viable]
CD2 APC-A
CD45 KO525-A
[60220300069]T CELL
ADARSH
[Viable]
CD4 PE-A
CD45 KO525-A
[60220300069]T CELL
ADARSH
[Viable]
CD8 PC7-A
CD4 PE-A

Unigenome – A Division of USLL
2A, 3A, 3B PASL - House, Beside Sahjanand College, Opposite Kamdenu Complex, Panjarapole, Ambawadi, Ahmedabad - 380015, Gujarat
Ph. No. – 079 – 49006800 I Website – www.unigenome.in | Email ID – info@unigenome.in

Patient Details

Patient Name	**ADARSH JAIN**	**Sample Id/LabID**	**60220300069**
Gender	Male	**DOB/AGE**	43 Yrs
Ref.By	Dr. Sachin Bansal	**Date of Sample Collection**	18-Feb-2026
Sample Type	Bone Marrow	**Date of Receipt**	18-Feb-2026
Tumor Cellularity	NA	**Date of Report**	06-Mar-2026
Location	SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL		

UNI NGS ALL PANEL (DNA mutations, CNVs, RNA Fusions)

Clinical Details:

Clinical Diagnosis : IPT- S/O Near Early T cell precursor acute lymphoblastic leukemia
Modal Chromosome Karyotype : 45,XY,add(2)(q37),add(4)(q35),der(7)?inv(7)(p15q36),-15,del(16)(q12-13)[7]/ 47,XY,+4[1]/46,XY[12]

RESULT

POSITIVE:

- Clinically Relevant Pathogenic Variant Identified in NRAS Gene.

- Variant of Uncertain Significance Identified in NOTCH1 Gene.

- No Fusion Identified.

Variants Identified:

Table-1: SNV Identified

Gene/Transcript	Locus	Variant/Amino Acid Change	Total Coverage/VAF	Impact on Protein Function	Variant classification	TIER
NRAS NM_002524.5	chr1:115256535	c.176C>A p.Ala59Asp	716X 16.20%	Gain-of-Function	Pathogenic	IIC
NOTCH1 NM_017617.5	chr9:139397768	c.5033T>C p.Leu1678Pro	1996X 18.64%	Loss-of-Function	VUS	None

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2A, 3A, 3B PASL - House, Beside Sahjanand College, Opposite Kamdenu Complex, Panjarapole, Ambawadi, Ahmedabad - 380015, Gujarat
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Relevant Biomarkers

Tier	Genomic Alteration	Relevant Therapies (In this cancer type)	Relevant Therapies (In other cancer type)	Clinical Trials
IIC	*NRAS p.(A59D) c.176C>A* Allele Frequency: 16.20%	None*	None*	2

*** Public data sources included in relevant therapies: FDA**[1], NCCN, **EMA**[2], ESMO
Tier Reference: Li et al. *Standards and Guidelines for the Interpretation and Reporting of Sequence Variants in Cancer: A Joint Consensus Recommendation of the Association for Molecular Pathology, American Society of Clinical Oncology, and College of American Pathologists.* J Mol Diagn. 2017 Jan;19(1):4-23.

Comments:

Benign and likely benign variants identified are not reported. These findings should be correlated with other clinical and laboratory tests for a definite conclusive interpretation.

Variants Description:

NRAS:c.176C>A:p.Ala59Asp: Pathogenic: The p.Ala59Asp variant (also known as c.176C>A), was detected in NRAS gene on chromosome 1 at position 115256535 with variant allele frequency of 16.20% (represented by 116 reads). This mutation is having a total depth of 716X. It is located at exon 3 of NM_002524.5 transcript and was found to change amino acid, Alanine to Aspartic acid at codon 59. It leads to Gain-of-Function. It is a Hotspot variant. It is represented by rs1570874751 in dbSNP and COSM253327 in Cosmic database. It is predicted as deleterious by SIFT and MutationTaster which is an in-silico DNA variant effect prediction tool. This variant was not found in the population frequency database like gnomAD, ExAC and 1000G database.

NOTCH1:c.5033T>C:p.Leu1678Pro: Variant of Uncertain Significance: The p.Leu1678Pro variant (also known as c.5033T>C), was detected in NOTCH1 gene on chromosome 9 at position 139397768 with variant allele frequency of 18.64% (represented by 372 reads). This mutation is having a total depth of 1996X. It is located at exon 27 of NM_017617.5 transcript and was found to change amino acid, Leucine to Proline at codon 1678. It leads to Loss-of-Function. It is a Hotspot variant. It is predicted as deleterious by SIFT and MutationTaster which is an in-silico DNA variant effect prediction tool. This variant was not found in the population frequency database like gnomAD, ExAC and 1000G database.

Biomarker Descriptions

NOTCH1 p.(L1678P) c.5033T>C

notch 1

Background: The NOTCH1 gene encodes the notch receptor 1 protein, a type 1 transmembrane protein and member of the NOTCH family of genes, which also includes NOTCH2, NOTCH3, and NOTCH4. NOTCH proteins contain multiple epidermal growth factor (EGF)-like repeats in their extracellular domain, which are responsible for ligand binding and homodimerization, thereby promoting NOTCH signaling[1]. Following ligand binding, the NOTCH intracellular domain is released, which activates the transcription of several genes involved in regulation of cell proliferation, differentiation, growth, and metabolism[2,3]. In cancer, depending on the tumor type, aberrations in the NOTCH family can be gain of function or loss of function suggesting both oncogenic and tumor suppressor roles for NOTCH family members[4,5,6,7].

Alterations and prevalence: Somatic mutations in NOTCH1 are observed in 15-20% of head and neck cancer, 5-10% of glioma, melanoma, gastric, esophageal, lung, and uterine cancers[8,9,10]. Activating mutations in either the heterodimerization or PEST domains of NOTCH1 have been reported in greater than 50% of T-cell acute lymphoblastic leukemia[11,12].

Potential relevance: Currently, no therapies are approved for NOTCH1 aberrations.

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Biomarker Descriptions (continued)

NRAS p.(A59D) c.176C>A

NRAS proto-oncogene, GTPase

Background: The NRAS proto-oncogene encodes a GTPase that functions in signal transduction and is a member of the RAS superfamily which also includes KRAS and HRAS. RAS proteins mediate the transmission of growth signals from the cell surface to the nucleus via the PI3K/AKT/MTOR and RAS/RAF/MEK/ERK pathways, which regulate cell division, differentiation, and survival[13,14,15].

Alterations and prevalence: Recurrent mutations in RAS oncogenes cause constitutive activation and are found in 20-30% of cancers. NRAS mutations are particularly common in melanomas (up to 25%) and are observed at frequencies of 5-10% in acute myeloid leukemia, colorectal, and thyroid cancers[9,16]. The majority of NRAS mutations consist of point mutations at G12, G13, and Q61[9,17]. Mutations at A59, K117, and A146 have also been observed but are less frequent[10,18].

Potential relevance: Currently, no therapies are approved for NRAS aberrations. The EGFR antagonists, cetuximab[19] and panitumumab[20], are contraindicated for treatment of colorectal cancer patients with NRAS mutations in exon 2 (codons 12 and 13), exon 3 (codons 59 and 61), and exon 4 (codons 117 and 146)[18]. In 2022, the FDA has granted fast track designation to the pan-RAF inhibitor, KIN-2787[21], for the treatment of NRAS-mutant metastatic or unresectable melanoma. In 2023, the FDA has granted fast track designation to the pan-RAF inhibitor, naporafenib, in combination with trametinib[22] for NRAS-mutated unresectable or metastatic melanoma. In 2024, the FDA has granted fast track designation to the MAPK pathway inhibitor, IMM-1-104[23], for the treatment of NRAS-mutant metastatic or unresectable melanoma. NRAS mutations are associated with poor prognosis in patients with low-risk myelodysplastic syndrome[24] as well as melanoma[25]. In a phase III clinical trial in patients with advanced NRAS-mutant melanoma, binimetinib improved progression free survival (PFS) relative to dacarbazine with median PFS of 2.8 and 1.5 months, respectively[26].

Relevant Therapy Summary

● In this cancer type ○ In other cancer type ◑ In this cancer type and other cancer types ✖ No evidence

NRAS p.(A59D) c.176C>A

Relevant Therapy	FDA	NCCN	EMA	ESMO	Clinical Trials*
trametinib, steroid, chemotherapy	✖	✖	✖	✖	● (I/II)
JZP-815	✖	✖	✖	✖	● (I)

* Most advanced phase (IV, III, II/III, II, I/II, I) is shown and multiple clinical trials may be available.

Methodology

Nucleic acid (DNA/RNA) was extracted from EDTA PB/BM sample, using standard Qiagen nucleic acid isolation kits. Brifely, 10ng of DNA/RNA was amplified using customised panel and sequencing was perfomed using Ion S5 platform as per user manual. The sequencing reads QC, mapping on hg19 human reference genome, variant calling (SNVs, small InDels, CNVs) and annotation was carried out with IonReporter™ (IR) Software 5.18.2.0. Latter uses RefSeq database was used for identification and characterization of genes associated variants. The annotation for variants was derived using various diseases databases like dbSNP, OMIM, ClinVar. The population frequency information from 1000 genomes, ExAC, GnomAD and ESP was used for elimination of common variants/ polymorphism.For prediction of possible impact of coding non-synonymous SNVs on the structure and function of protein, PolyPhen-2 andSIFT score was used. Further Oncomine Reporter software was used for annotating variants with curated list of relevant labels, guidelines, and global clinical trials.UNI NGS ALL panel will analyze across 203 genes categorized by somatic alteration type 82 Hotspots genes, 24 Focal CNV gains, 44 Full CDS for DEL mutations, 88 Fusion drivers genes.

Run QC statistics
Sample is sequenced at Average base coverage depth of 2,030. The Target base coverage at 500X is 92.18%.

Variant Classification

Tier I	Variants with strong clinical significance	Level A evidence	FDA-approved therapy included in professional guidelines
		Level B evidence	Well-powered studies with consensus from leaders in the field
Tier II	Variants with potential clinical significance	Level C evidence	FDA-approved therapies for different tumor types or investigational therapies. Multiple small published studies with some consensus
		Level D evidence	Preclinical trials or few case reports without consensus.
Tier III	Variants of unknown clinical significance		Not observed at significant allele frequency in the general or specific subpopulation databases, or pan-cancer or tumor-specific variant databases. No convincing published evidence of cancer association

Evidence-based variant Categorization

Tier I	Variants with strong clinical significance	Level A evidence	FDA-approved therapy included in professional guidelines
		Level B evidence	Well-powered studies with consensus from leaders in the field
Tier II	Variants with potential clinical significance	Level C evidence	FDA-approved therapies for different tumor types or investigational therapies. Multiple small published studies with some consensus
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Genes Assayed

Genes Assayed for the Detection of DNA Sequence Variants (UNI NGS ALL PANEL)

ABL1, ABL2, ALK, ACVR1, AKT1, ASXL1, ASXL2, BRAF, CALR, CBL, CCND1, CCND3, CCR5, CDK4, CIC, CREBBP, CRLF2, CSF1R, CSF3R, CTNNB1, DAXX, DNMT3A, EGFR, EP300, ERBB2, ERBB3, ERBB4, ESR1, EZH2, FASLG, FBXW7, FGFR1, FGFR2, FGFR3, FLT3, GATA2, GNA11, GNAQ, H3F3AP4, HDAC9, H3C2, HRAS, IDH1, IDH2, IL7R, JAK1, JAK2, JAK3, KDM4C, KDR, KIT, KRAS, MAP2K1, MAP2K2, MET, MPL, MSH6, MTOR, MYC, MYCN, NCOR2, NOTCH1, NPM1, NRAS, NT5C2, PAX5, PDGFRA, PDGFRB, PIK3CA, PIK3R1, PPM1D, PTPN11, RAF1, RET, RHOA, SETBP1, SETD2, SH2B3, SH2D1A, SMO, STAT3, STAT5B, TERT, TPMT, USP7, ZMYM3

Genes Assayed for the Detection of DNA Sequence Variants(Full Exon Coverage) (UNI NGS ALL PANEL)

APC, ARID1A, ARID1B, ATRX, CDKN2A, CDKN2B, CEBPA, CHD7, CRLF1, DDX3X, DICER1, EBF1, EED, FAS, GATA1, GATA2, GATA3, ID3, IKZF1, KDM6A, KMT2D, MYOD1, NF1, NF2, PHF6, PRPS1, PSMB5, PTCH1, PTEN, RB1, RUNX1, SMARCA4, SMARCB1, SOCS2, SUFU, SUZ12, TCF3, TET2, TP53, TSC1, TSC2, WHSC2, WT1, XIAP

Genes Assayed for the Detection of Copy Number Variation(CNV) (UNI NGS ALL PANEL)

ABL2, ALK, BRAF, CCND1, CDK6, CDK4, EGFR, ERBB2, ERBB3, FGFR1, FGFR2, FGFR3, FGFR4, GLI1, GLI2, IGF1R, JAK1, JAK2, KIT, KRAS, MDM2, MET, MYC, MYCN, PDGFRA, PIK3CA, JAK3, MDM4

Genes Assayed for the Fusions (UNI NGS ALL PANEL)

ABL1, ABL2, AFF3, ALK, BCL11B, BCOR, BCR, BRAF, CAMTA1, CCND1, CIC, CREBBP, CRLF2, CSF1R, DUSP22, EGFR, ETV6, EWSR1, FGFR1, FGFR2, FGFR3, FLT3, FOSB, FUS, GLI1, GLIS2, HMGA2, JAK2, KAT6A, KMT2A, KMT2B, KMT2C, KMT2D, LMO2, MAML2, MAN2B1, MECOM, MEF2D, MET, MKL2, MLLT10, MN1, MYB, MYBL1, MYH11, MYH9, NCOA2, NCOR1, NOTCH1, NOTCH2, NOTCH4, NPM1, NR4A3, NTRK1, NTRK2, NTRK3, NUP214, NUP98, NUTM1, NUTM2B, PAX3, PAX5, PAX7, PDGFB, PDGFRA, PDGFRB, PLAG1, RAF1, RANBP17, RARA, RECK, RELA, RET, ROS1, RUNX1, SS18, SSBP2, STAG2, STAT6, TAL1, TCF3, TFE3, TP63, TSLP, TSPAN4, UBTF, USP6, WHSC2, YAP1, ZMYND11, ZNF384

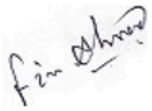
Limitations and Disclaimer

1. This test was developed and its performance characteristics determined by UNIGENOME, Ahmedabad. It has not been cleared or approved by the US Food and Drug Administration and NABL.
2. This NGS test used does not allow definitive differentiation between germline and somatic variants if FFPE is used. However, variants with variant allele frequency at nearly 50% or 100% should be considered Germline mutation. To rule out germ line mutations, repeat analysis using peripheral blood/saliva sample is recommended.
   3.False negative results may be due to sampling issues, errors in sample handling, mislabeling, transportation issues, technical limitations of the assay and mutations frequency below the limit of detection of the assay, i.e., 5% for SNVs and 10% for short indels. It is also possible some complex insertion/deletion variants may not be identified.
3. Sanger confirmation of reported mutations is available on request with additional charges.
4. The classification and interpretation of all the variants is carried out based on the current state of scientific knowledge and medical understanding and may change over time with more information available in future.
5. This report should not be considered as medical advice. Results of this test need be interpreted within the context of clinical findings and other relevant clinical and laboratory data and should not be used alone.
6. Likely benign and benign variants are not reported and can be provided upon request.

Report Signed By

DR.SPANDAN CHAUDHARY, Ph.D.

(Chief Scientist, NGS Division)



DR.FIROZ AHMAD, Ph.D.

(HEAD, Clinical Genomics)

DR.NEERAJ ARORA, MD

PDF(Mol.Hemat and Hematopath)

LAB DIRECTOR

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LABORATORY REPORT

Reg. No	:	**60220300069**	Histo / Cyto No :	**H26108619**	Reg. Date	:	18-Feb-2026 12:38
Name	:	**ADARSH JAIN**			Collectedon	:	17-Feb-2026 12:38
Sex/Age	:	**Male / 43 Years**			Report Date	:	20-Feb-2026 11:45
Ref. By	:	Dr. Sachin Bansal DNB Hem (Hemato Onco)			Tele. No	:	
Location	:	SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL			Dispatch At	:	

HISTOPATHOLOGY REPORT

Specimen :
Bone marrow trephine biopsy.

Clinical Diagnosis :
Significant clinical history: C/O dyspnoea on exertion for 20 days found to have anaemia and leukopenia, no hepatosplenomegaly. ? Acute leukaemia.
CBC & Peripheral smear: Hb: 8.5gm/dl, WBC: 980/cmm, Platelet: 2,51,000/cmm Differential P: 11%, L: 58%, M: 05%, Blasts: 26%, Occasional nRBC seen.
Aspirate Impression: Acute leukaemia
IPT Impression: Immunophenotyping findings of the Bone marrow sample are suggestive of Near Early T cell
precursor acute lymphoblastic leukemia.

Gross Description :
One bony fragment - 1.7 cm.
All in 1 block.

Microscopic Description :
Cellularity - Markedly hypercellular marrow. There is diffuse infiltrate of medium sized immature cells with round nuclei, fine chromatin, indistinct nucleoli and scant cytoplasm.
Erythroids - markedly decreased.
Myeloids - markedly decreased..
Megakaryocytes - Decreased.
Reticulin - No increase.
Bone trabeculae - Normal.

Diagnosis :
Bone marrow trephine biopsy - **Acute Leukemia.**

DR. SAAISHTA BAKSHI
M.D. Pathology
G-20622

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LABORATORY REPORT

Reg. No	:	**60220300069**	Histo / Cyto No :	**H26108619**	Reg. Date	:	18-Feb-2026 12:38
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Location	:	SUNRISE HEMATOLOGY AND BLOOD CANCER CENTER @ BHOPAL			Dispatch At	:	

Suggest correlation with aspirate and flow cytometry findings for further subtyping.

---------- End Of Report ----------

All stained slides and/or paraffin blocks labeled Histo/Cyto No: H26108619 returned along with report. Please preserve them Carefully.

Containers of all biopsies with entire tissue processing will be discarded within a week after report is approved.

DR. SAAISHTA BAKSHI
M.D. Pathology
G-20622

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