

Dr. Vinay Chopra
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 Chairman & Consultant Pathologist

Dr. Yugam Chopra
 MD (Pathology)
 CEO & Consultant Pathologist

NAME	: Mr. AVTAR SINGH	PATIENT ID	: 1747325
AGE/ GENDER	: 64 YRS/MALE	REG. NO./LAB NO.	: 012502060002
COLLECTED BY	:	REGISTRATION DATE	: 06/Feb/2025 07:35 AM
REFERRED BY	:	COLLECTION DATE	: 06/Feb/2025 07:35AM
BARCODE NO.	: 01525026	REPORTING DATE	: 06/Feb/2025 03:47PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HbA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	5.9	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	122.63	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):	
REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HbA1C) in %
Non diabetic Adults >= 18 years	<5.7
At Risk (Prediabetes)	5.7 - 6.4
Diagnosing Diabetes	>= 6.5
Therapeutic goals for glycemic control	Age > 19 Years
	Goals of Therapy: < 7.0
	Actions Suggested: >8.0
	Age < 19 Years
	Goal of therapy: <7.5

COMMENTS:

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targeting a goal of < 7.0% may not be appropriate.
- High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shortens RBC life span like acute blood loss, hemolytic anemia falsely lowers HbA1c results.
- HbA1c results from patients with HbSS, HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.




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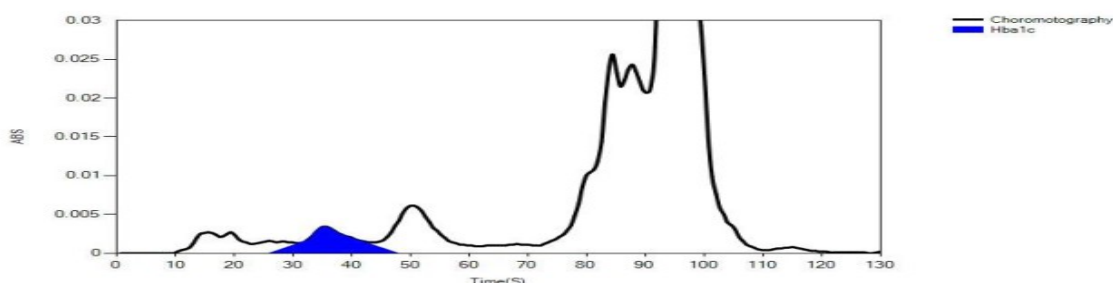
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LIFOTRONIC Graph Report

Name :	Case :	Patient Type :	Test Date : 06/02/2025 17:56:32
Age :	Department :	Sample Type : Whole Blood EDTA	Sample Id : 01525026
Gender :			Total Area : 10106

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	70	2691	8994	84.3
HbA1c	37	62	629	5.9
La1c	26	34	260	2.4
HbF	19	16	17	0.2
Hba1b	14	27	89	0.8
Hba1a	11	27	117	1.1




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BARCODE NO.	: 01525026	REPORTING DATE	: 06/Feb/2025 10:59AM
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CLINICAL CHEMISTRY/BIOCHEMISTRY

CHOLESTEROL: SERUM

CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	125.13	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
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INTERPRETATION:

NATIONAL LIPID ASSOCIATION RECOMMENDATIONS (NLA-2014)	CHOLESTEROL IN ADULTS (mg/dL)	CHOLESTEROL IN ADULTS (mg/dL)
DESIRABLE	< 200.0	< 170.0
BORDERLINE HIGH	200.0 – 239.0	171.0 – 199.0
HIGH	>= 240.0	>= 200.0

NOTE:

- Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
- As per National Lipid association - 2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.




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URIC ACID

URIC ACID: SERUM	6.74	mg/dL	3.60 - 7.70
by URICASE - OXIDASE PEROXIDASE			

INTERPRETATION:-

1. GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.
 2. Uric Acid is the end product of purine metabolism . Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

1. Idiopathic primary gout.
2. Excessive dietary purines (organ meats, legumes, anchovies, etc).
3. Cytolytic treatment of malignancies especially leukemias & lymphomas.
4. Polycythemia vera & myeloid metaplasia.
5. Psoriasis.
6. Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCRETION (BY KIDNEYS)

1. Alcohol ingestion.
2. Thiazide diuretics.
3. Lactic acidosis.
4. Aspirin ingestion (less than 2 grams per day).
5. Diabetic ketoacidosis or starvation.
6. Renal failure due to any cause etc.

DECREASED:-

(A).DUE TO DIETARY DEFICIENCY

1. Dietary deficiency of Zinc, Iron and molybdenum.
2. Fanconi syndrome & Wilsons disease.
3. Multiple sclerosis .
4. Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCRETION

1. Drugs:- Probenecid , sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosteroids and ACTH, anti-coagulants and estrogens etc.

*** End Of Report ***





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