

Dr. Vinay Chopra  
MD (Pathology & Microbiology)  
Chairman & Consultant Pathologist

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

<b>NAME</b>	: Mr. AMIT	<b>PATIENT ID</b>	: 1569219
<b>AGE/ GENDER</b>	: 45 YRS/MALE	<b>REG. NO./LAB NO.</b>	: <b>012408030035</b>
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 03/Aug/2024 11:04 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 03/Aug/2024 11:12AM
<b>BARCODE NO.</b>	: 01514361	<b>REPORTING DATE</b>	: 03/Aug/2024 02:53PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
<b>CLIENT ADDRESS</b>	: 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 <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	5.4	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	108.28	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	
	<b>Age &gt; 19 Years</b>	
Therapeutic goals for glycemic control	Goals of Therapy:	< 7.0
	Actions Suggested:	>8.0
		<b>Age &lt; 19 Years</b>
	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. 4.High
- HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower 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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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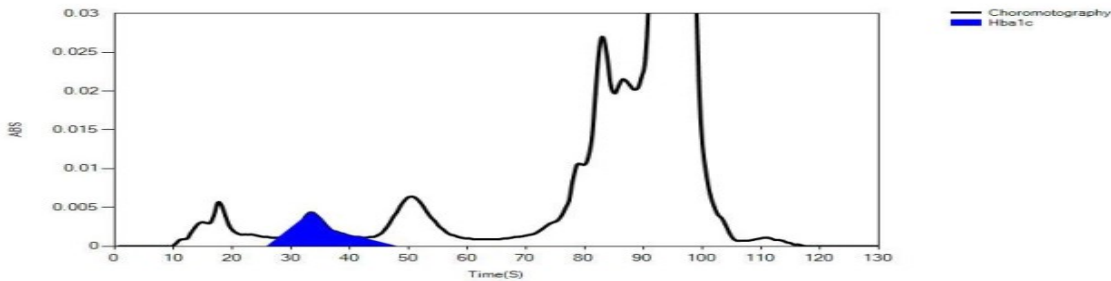
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Test Name	Value	Unit	Biological Reference interval
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LIFOTRONIC Graph Report

Name :	Case :	Patient Type :	Test Date : 03/08/2024 14:40:20
Age :	Department :	Sample Type : Whole Blood EDTA	Sample Id : 01514361
Gender :			Total Area : 12540

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	70	3418	11295	86.1
HbA1c	37	64	708	5.4
La1c	24	43	212	1.6
HbF	21	11	13	0.1
Hba1b	13	58	205	1.6
Hba1a	11	31	107	0.8



  
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<b>BARCODE NO.</b>	: 01514361	<b>REPORTING DATE</b>	: 03/Aug/2024 12:47PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
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### CLINICAL CHEMISTRY/BIOCHEMISTRY

#### URIC ACID

URIC ACID: SERUM	4.36	mg/dL	3.60 - 7.70
<i>by URICASE - OXIDASE PEROXIDASE</i>			

**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. Sick 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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