

# **KOS Diagnostic Lab**

(A Unit of KOS Healthcare)



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

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

: 08/Aug/2024 01:51PM

60.00 - 140.00

**NAME** : Mr. VINEET

**AGE/ GENDER** : 25 YRS/MALE **PATIENT ID** : 1574443

**COLLECTED BY** : 012408080047 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 08/Aug/2024 11:45 AM BARCODE NO. :01514721 **COLLECTION DATE** : 08/Aug/2024 11:46AM

: KOS DIAGNOSTIC LAB **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit **Biological Reference interval** 

REPORTING DATE

mg/dL

# **HAEMATOLOGY GLYCOSYLATED HAEMOGLOBIN (HBA1C)**

GLYCOSYLATED HAEMOGLOBIN (HbA1c): 5.7 4.0 - 6.4

WHOLE BLOOD

**INTERPRETATION:** 

CLIENT CODE.

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

116.89

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

### COMMENTS:

- 1. Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients.
- 2. 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 HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.
- 3. 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, targetting a goal of < 7.0% may not be 4.High

HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications

5. Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.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 gycemic control.

7. Specimens from patients with polycythemia or post-spienctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)





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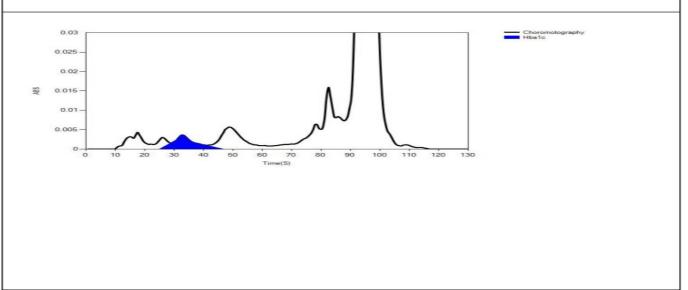
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Test Name Value Unit Biological Reference interval

#### LIFOTRONIC Graph Report

Name :	Case:	Patient Type :	Test Date: 08/08/2024 13:07:10
Age:	Department:	Sample Type: Whole Blood EDTA	Sample ld: 01514721
Gender:			Total Area: 11220

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	70	2872	10118	88.5
HbA1c	36	57	649	5.7
La1c	28	12	156	1.4
HbF	19	30	39	0.3
Hba1b	13	43	154	1.3
Hba1a	10	32	104	0.9





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# CLINICAL CHEMISTRY/BIOCHEMISTRY

**URIC ACID** 

URIC ACID: SERUM 5.62 mg/dL 3.60 - 7.70

by URICASE - OXIDASE PEROXIDASE

#### <u> NTERPRETATION:-</u>

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 leukemais & lymphomas.

4. Polycythemai vera & myeloid metaplasia.

5.Psoriasis.

6. Sickle cell anaemia etc.

### (B).DUE TO DECREASED EXCREATION (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 EXCREATION

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

\*\*\* End Of Report \*\*\*



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