

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

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

<b>NAME</b>	: Mr. BANT RAM	<b>PATIENT ID</b>	: 1610524
<b>AGE/ GENDER</b>	: 70 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 012409120029
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 12/Sep/2024 09:56 AM
<b>REFERRED BY</b>	: CIVIL HOSPITAL (AMBALA CANTT)	<b>COLLECTION DATE</b>	: 12/Sep/2024 09:58AM
<b>BARCODE NO.</b>	: 01516819	<b>REPORTING DATE</b>	: 12/Sep/2024 11:24AM
<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 by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	5.6	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	114.02	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	<b>Age &gt; 19 Years</b>
	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.
- 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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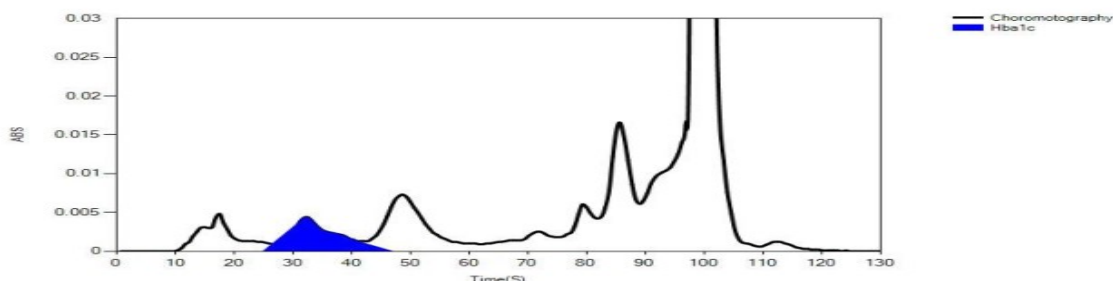
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LIFOTRONIC Graph Report

Name :	Case :	Patient Type :	Test Date : 12/09/2024 11:03:28
Age :	Department :	Sample Type : Whole Blood EDTA	Sample Id : 01516819
Gender :			Total Area : 12446

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	73	7528	11280	88.0
HbA1c	36	73	699	5.4
La1c	28	18	169	1.3
HbF	23	44	39	0.3
Hba1b	12	49	169	1.3
Hba1a	11	31	90	0.7



  
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**ENDOCRINOLOGY**  
**TESTOSTERONE: TOTAL**

TESTOSTERONE - TOTAL: SERUM	7.37	ng/mL	1.26 - 10.20
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by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

**INTERPRETATION:**

1. Testosterone is secreted in females by the ovary and formed indirectly from androstenedione in adrenal glands.
2. In males it is secreted by the testes. It circulates in blood bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form.
3. The bioavailable fraction includes the free form and that "weakly bound" to albumin (40% of the total in men and 20% of the total in women) and bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone.
4. The total testosterone bound to SHBG fluctuates since SHBG levels are affected by medication, disease, sex steroids and insulin.

**CLINIC USE:**

1. Assessment of testicular functions in males
2. Management of hirsutism and virilization in females

**INCREASED LEVELS:**

1. Precocious puberty (Males)
2. Androgen resistance
3. Testotoxicosis
4. Congenital Adrenal Hyperplasia
5. Polycystic ovarian disease
7. Ovarian tumors

**DECREASED LEVELS:**

1. Delayed puberty (Males)
2. Gonadotropin deficiency
3. Testicular defects
4. Systemic diseases

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



  
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