

Dr. Vinay Chopra
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 Chairman & Consultant Pathologist

Dr. Yugam Chopra
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NAME	: Mrs. KAVITA	PATIENT ID	: 1781846
AGE/ GENDER	: 45 YRS/FEMALE	REG. NO./LAB NO.	: 012503070051
COLLECTED BY	:	REGISTRATION DATE	: 07/Mar/2025 11:28 AM
REFERRED BY	: LOOMBA HOSPITAL (AMBALA CANTT)	COLLECTION DATE	: 07/Mar/2025 12:06PM
BARCODE NO.	: 01526640	REPORTING DATE	: 07/Mar/2025 12:24PM
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

HAEMOGLOBIN (HB)

HAEMOGLOBIN (HB) by CALORIMETRIC	11.4 ^L	gm/dL	12.0 - 16.0
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INTERPRETATION:-

Hemoglobin is the protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs.

A low hemoglobin level is referred to as ANEMIA or low red blood count.

ANEMIA (DECREASED HAEMOGLOBIN):

- 1) Loss of blood (traumatic injury, surgery, bleeding, colon cancer or stomach ulcer)
- 2) Nutritional deficiency (iron, vitamin B12, folate)
- 3) Bone marrow problems (replacement of bone marrow by cancer)
- 4) Suppression by red blood cell synthesis by chemotherapy drugs
- 5) Kidney failure
- 6) Abnormal hemoglobin structure (sickle cell anemia or thalassemia).

POLYCYTHEMIA (INCREASED HAEMOGLOBIN):

- 1) People in higher altitudes (Physiological)
- 2) Smoking (Secondary Polycythemia)
- 3) Dehydration produces a falsely rise in hemoglobin due to increased haemoconcentration
- 4) Advanced lung disease (for example, emphysema)
- 5) Certain tumors
- 6) A disorder of the bone marrow known as polycythemia rubra vera,
- 7) Abuse of the drug erythropoietin (Epogen) by athletes for blood doping purposes (increasing the amount of oxygen available to the body by chemically raising the production of red blood cells).

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD




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GLYCOSYLATED HAEMOGLOBIN (HbA1c)

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

ADVICE **KINDLY CORRELATE CLINICALLY**

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:

1. Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance 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 HbA1c. 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, 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
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 glycemic control.
7. 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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CLINICAL CHEMISTRY/BIOCHEMISTRY
GLUCOSE RANDOM (R)

GLUCOSE RANDOM (R): PLASMA <i>by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)</i>	246.15^H	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > OR = 200.0
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INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A random plasma glucose level below 140 mg/dl is considered normal.
2. A random glucose level between 140 - 200 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A random glucose level of above 200 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.




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ENDOCRINOLOGY

BETA HCG - TOTAL (QUANTITATIVE): MATERNAL

BETA HCG TOTAL, PREGNANCY MATERNAL: **7649.78^H** mIU/mL < 5.0

SERUM

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:

MEN:	mIU/ml	< 2.0
NON PREGNANT PRE-MENOPAUSAL WOMEN:	mIU/ml	< 5.0
MENOPAUSAL WOMEN:	mIU/ml	< 7.0
BETA HCG EXPECTED VALUES IN ACCORDANCE TO WEEKS OF GESTATIONAL AGE		
WEEKS OF GESTATION	Unit	Value
4-5	mIU/ml	1500 - 23000
5-6	mIU/ml	3400 - 135300
6-7	mIU/ml	10500 - 161000
7-8	mIU/ml	18000 - 209000
8-9	mIU/ml	37500 - 219000
9-10	mIU/ml	42800 - 218000
10-11	mIU/ml	33700 - 218700
11-12	mIU/ml	21800 - 193200
12-13	mIU/ml	20300 - 166100
13-14	mIU/ml	15400 - 190000
2rd TRIMESTER	mIU/ml	2800 - 176100
3rd TRIMESTER	mIU/ml	2800 - 144400




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1.hCG is a Glycoprotein with alpha and beta chains. Beta subunit is specific to hCG.
 2.It is largely secreted by trophoblastic tissue. Small amounts may be secreted by fetal tissues and by the adult ant pituitary.
INCREASED :
 1.Pregnancy
 2.Gestationalsite & Non gestational trophoblastic neoplasia.
 3.In mixed germ cell tumors.
SIGNIFICANTLY HIGHER THAN EXPECTED LEVEL:
 1.Multiple pregnancies & High risk molar pregnancies are usually associated with levels in excess of one lac mIU/ml.
 2.Erythroblastosis fetalis & Downs syndrome.
DECREASED:
 1.Ectopic pregnancy.
 2.Intra-uterine fetal death.
NOTE:
 1.The test becomes positive 7-9 days after the midcycle surge that precedes ovulation (time of blastocyst implantation). Blood levels rise rapidly after this and double every 1.4 - 2 days.
 2.Peak values are usually seen at 60-80 days of LMP. The levels then begin to taper and ebb out around the 20th week. These low levels are then maintained throughout pregnancy.
 3.Doubling time: In intra-uterine pregnancy, serum hCG levels increase by approximately 66% every 48 hrs.Inappropriately rising serum hCG levels are suggestive of dying or ectopic pregnancy.
CAUTION:
 Spuriously high levels (Phantom hCG) may be seen in presence of heterophilic antibodies (found in some normal people). If persistently raised levels are seen in a non-pregnant patient with no evidence of other obvious causes for such an increase a urine hCG assay may help confirm presence of the heterophile antibodies.




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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM	54.4	ng/mL	DEFICIENCY: < 20.0
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)			INSUFFICIENCY: 20.0 - 30.0
			SUFFICIENCY: 30.0 - 100.0
			TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFERRED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

1. Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.

2. 25-OH--Vitamin D represents the main body reservoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.

3. Vitamin D plays a primary role in the maintenance of calcium homeostasis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (PTH).

4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

1. Lack of sunshine exposure.
2. Inadequate intake, malabsorption (celiac disease)
3. Depressed Hepatic Vitamin D 25- hydroxylase activity
4. Secondary to advanced Liver disease
5. Osteoporosis and Secondary Hyperparathyroidism (Mild to Moderate deficiency)
6. Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:

1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphosphatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:- Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interfere with Vitamin D absorption.

*** End Of Report ***




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