

Dr. Vinay Chopra
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NAME	: Mrs. SANGETA CHOPRA	PATIENT ID	: 1746238
AGE/ GENDER	: 65 YRS/FEMALE	REG. NO./LAB NO.	: 012502050033
COLLECTED BY	:	REGISTRATION DATE	: 05/Feb/2025 11:33 AM
REFERRED BY	:	COLLECTION DATE	: 05/Feb/2025 11:35AM
BARCODE NO.	: 01525000	REPORTING DATE	: 05/Feb/2025 03:18PM
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 <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

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
Age > 19 Years	
Therapeutic goals for glycemic control	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. 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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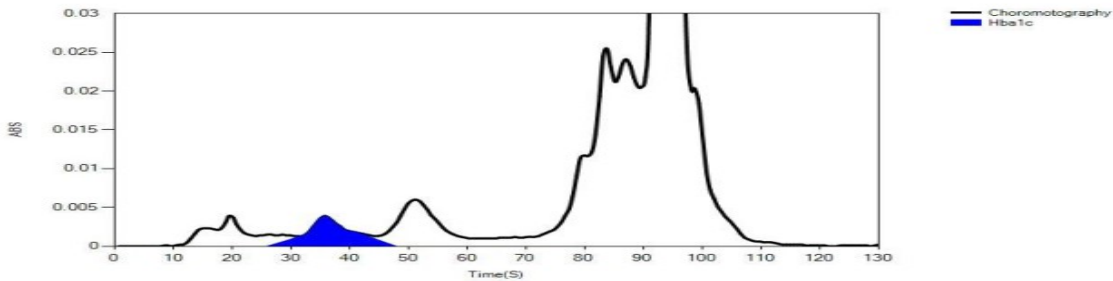
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LIFOTRONIC Graph Report

Name :	Case :	Patient Type :	Test Date : 05/02/2025 17:38:52
Age :	Department :	Sample Type : Whole Blood EDTA	Sample Id : 01525000
Gender :			Total Area : 9736

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	69	2927	8591	82.3
HbA1c	37	60	613	5.9
La1c	26	39	277	2.6
HbF	19	15	14	0.1
Hba1b	14	40	140	1.3
Hba1a	11	23	101	1.0




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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE RANDOM (R)

GLUCOSE RANDOM (R): PLASMA <i>by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)</i>	122.12	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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Test Name	Value	Unit	Biological Reference interval
UREA			
UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	28.76	mg/dL	10.00 - 50.00

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CREATININE

CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	0.63	mg/dL	0.40 - 1.20
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URIC ACID

URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	4.5	mg/dL	2.50 - 6.80
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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.




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Test Name	Value	Unit	Biological Reference interval
POTASSIUM			
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.49	mmol/L	3.50 - 5.00

INTERPRETATION:-

POTASSIUM:


Potassium is the major cation in the intracellular fluid. 90% of potassium is concentrated within the cells. When cells are damaged, potassium is released in the blood.

HYPOKALEMIA (LOW POTASSIUM LEVELS):-


1. Diarrhoea, vomiting & malabsorption.
2. Severe Burns.
3. Increased Secretions of Aldosterone

HYPERKALEMIA (INCREASED POTASSIUM LEVELS):-

1. Oliguria
2. Renal failure or Shock
3. Respiratory acidosis
4. Hemolysis of blood

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Test Name	Value	Unit	Biological Reference interval
SODIUM			
SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	142.7	mmol/L	135.0 - 150.0

INTERPRETATION:-

SODIUM:-

Sodium is the major cation of extra-cellular fluid. Its primary function in the body is to chemically maintain osmotic pressure & acid base balance & to transmit nerve impulse.

HYPONATREMIA (LOW SODIUM LEVEL) CAUSES:-

1. Low sodium intake.
2. Sodium loss due to diarrhea & vomiting with adequate water and inadequate salt replacement.
3. Diuretics abuses.
4. Salt loosing nephropathy.
5. Metabolic acidosis.
6. Adrenocortical insufficiency .
7. Hepatic failure.

HYPERNATREMIA (INCREASED SODIUM LEVEL) CAUSES:-

1. Hyperapnea (Prolonged)
2. Diabetes insipidus
3. Diabetic acidosis
4. Cushings syndrome
5. Dehydration



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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM <i>by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)</i>	82.2	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
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INTERPRETATION:

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

- 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.
- 25-OH--Vitamin D represents the main body resevoir 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.
- Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

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

INCREASED:

- 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.



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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	873	pg/mL	190.0 - 890.0
<i>by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)</i>			

INTERPRETATION:-

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1. Ingestion of Vitamin C	1. Pregnancy
2. Ingestion of Estrogen	2. DRUGS: Aspirin, Anti-convulsants, Colchicine
3. Ingestion of Vitamin A	3. Ethanol ingestion
4. Hepatocellular injury	4. Contraceptive Hormones
5. Myeloproliferative disorder	5. Haemodialysis
6. Uremia	6. Multiple Myeloma

- Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
- In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
- The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
- Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
- Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
- Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
- Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.

NOTE: A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

*** End Of Report ***



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