

Dr. Vinay Chopra
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Chairman & Consultant Pathologist

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

NAME : Mrs. MAMTA SAINI
AGE/ GENDER : 44 YRS/FEMALE
COLLECTED BY : SURJESH
REFERRED BY :
BARCODE NO. : 01516164
CLIENT CODE. : KOS DIAGNOSTIC LAB
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1598945
REG. NO./LAB NO. : 012409020031
REGISTRATION DATE : 02/Sep/2024 10:16 AM
COLLECTION DATE : 02/Sep/2024 10:16 AM
REPORTING DATE : 02/Sep/2024 11:13 AM

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	113.9 ^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
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INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 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 fasting plasma glucose level of above 125 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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URIC ACID

URIC ACID: SERUM	3.76	mg/dL	2.50 - 6.80
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by URICASE - OXIDASE PEROXIDASE

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 leukemais & lymphomas.
- 4.Polycythema 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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM
 by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

16.2^L

ng/mL

DEFICIENCY: < 20.0
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

- 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 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.
- 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).
- 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 Hyperparathyroidism (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
94^L
pg/mL
190.0 - 890.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

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 lgestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
 2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
 3.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.
 4.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).
 5.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.
 6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
 7.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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