

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

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

| | | | |
|-----------------------|--|--------------------------|------------------------|
| NAME | : Mr. ANURAG SHARMA | PATIENT ID | : 1570888 |
| AGE/ GENDER | : 51 YRS/MALE | REG. NO./LAB NO. | : 012408050061 |
| COLLECTED BY | : | REGISTRATION DATE | : 05/Aug/2024 12:39 PM |
| REFERRED BY | : | COLLECTION DATE | : 05/Aug/2024 12:45PM |
| BARCODE NO. | : 01514528 | REPORTING DATE | : 05/Aug/2024 02:27PM |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|-----------|-------|------|-------------------------------|
|-----------|-------|------|-------------------------------|

CLINICAL CHEMISTRY/BIOCHEMISTRY

URIC ACID

| | | | |
|------------------|------|-------|-------------|
| URIC ACID: SERUM | 6.21 | mg/dL | 3.60 - 7.70 |
|------------------|------|-------|-------------|

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.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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

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

15.6^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.

*** End Of Report ***




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