

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

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

<b>NAME</b>	: Mr. SUKHBIR	<b>PATIENT ID</b>	: 1821485
<b>AGE/ GENDER</b>	: 42 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 012504070069
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 07/Apr/2025 05:26 PM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 07/Apr/2025 05:29PM
<b>BARCODE NO.</b>	: 01528546	<b>REPORTING DATE</b>	: 07/Apr/2025 08:04PM
<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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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### URIC ACID

URIC ACID: SERUM	5.5	mg/dL	3.60 - 7.70
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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 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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<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 07/Apr/2025 05:42PM
<b>BARCODE NO.</b>	: 01528546	<b>REPORTING DATE</b>	: 07/Apr/2025 08:33PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM	<b>18.6<sup>L</sup></b>	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

- 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.

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



  
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