

**Dr. Vinay Chopra**  
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 Chairman & Consultant Pathologist

**Dr. Yugam Chopra**  
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<b>NAME</b>	: Mr. ABHISHEK BEDI	<b>PATIENT ID</b>	: 1634227
<b>AGE/ GENDER</b>	: 37 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 012410040057
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 04/Oct/2024 12:39 PM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 04/Oct/2024 01:01PM
<b>BARCODE NO.</b>	: 01518313	<b>REPORTING DATE</b>	: 04/Oct/2024 04:08PM
<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

### MAGNESIUM

MAGNESIUM: SERUM	2.04	mg/dL	1.6 - 2.6
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by XYLIDYL BLUE, SPECTROPHOTOMETRY

#### INTERPRETATION:-

1. Magnesium along with potassium is a major intracellular cation.  
 2. Magnesium is a cofactor of many enzyme systems. All adenosine triphosphate (ATP)-dependent enzymatic reactions require magnesium as a cofactor. 3. Approximately 70% of magnesium ions are stored in bone. The remainder is involved in intermediary metabolic processes; about 70% is present in free form while the other 30% is bound to proteins (especially albumin), citrates, phosphate, and other complex formers. The serum magnesium level is kept constant within very narrow limits. Regulation takes place mainly via the kidneys, primarily via the ascending loop of Henle.

**INCREASED (HYPERMAGNESIA):-** Conditions that interfere with glomerular filtration result in retention of magnesium and hence elevation of serum concentrations.

1. Acute and chronic renal failure.
2. magnesium overload.
3. Magnesium release from the intracellular space.
4. Mild-to-moderate hypermagnesemia may prolong atrioventricular conduction time. Magnesium toxicity may result in central nervous system (CNS) depression, cardiac arrest, and respiratory arrest.

#### DECREASED (HYPOMAGNESIA):-

1. Chronic alcoholism.
2. Childhood malnutrition.
3. Malabsorption.
4. Acute pancreatitis.
5. Hypothyroidism.
6. Chronic glomerulonephritis.
7. Aldosteronism.
8. Prolonged intravenous feeding.

#### NOTE:-

Numerous studies have shown a correlation between magnesium deficiency and changes in calcium-, potassium-, and phosphate-homeostasis which are associated with cardiac disorders such as ventricular arrhythmias that cannot be treated by conventional therapy, increased sensitivity to digoxin, coronary artery spasms, and sudden death. Additional concurrent symptoms include neuromuscular and neuropsychiatric disorders.



*[Signature]*

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Test Name	Value	Unit	Biological Reference interval
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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

<b>VITAMIN D (25-HYDROXY VITAMIN D3): SERUM</b> by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	27.034 <sup>L</sup>	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 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	198.52	pg/mL	190.0 - 830
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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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