

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

**Dr. Yugam Chopra**  
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<b>NAME</b>	: Mr. DEBANG	<b>PATIENT ID</b>	: 1822167
<b>AGE/ GENDER</b>	: 17 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 012504080013
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 08/Apr/2025 08:07 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 08/Apr/2025 08:32AM
<b>BARCODE NO.</b>	: 01528566	<b>REPORTING DATE</b>	: 08/Apr/2025 02:16PM
<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

### LIPID PROFILE : BASIC

<b>CHOLESTEROL TOTAL: SERUM</b> <i>by CHOLESTEROL OXIDASE PAP</i>	141.3	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
<b>TRIGLYCERIDES: SERUM</b> <i>by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)</i>	160.55 <sup>H</sup>	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
<b>HDL CHOLESTEROL (DIRECT): SERUM</b> <i>by SELECTIVE INHIBITION</i>	43.02	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
<b>LDL CHOLESTEROL: SERUM</b> <i>by CALCULATED, SPECTROPHOTOMETRY</i>	66.17	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
<b>NON HDL CHOLESTEROL: SERUM</b> <i>by CALCULATED, SPECTROPHOTOMETRY</i>	98.28	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
<b>VLDL CHOLESTEROL: SERUM</b> <i>by CALCULATED, SPECTROPHOTOMETRY</i>	32.11	mg/dL	0.00 - 45.00
<b>TOTAL LIPIDS: SERUM</b> <i>by CALCULATED, SPECTROPHOTOMETRY</i>	443.15	mg/dL	350.00 - 700.00



  
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CHOLESTEROL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	3.28	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.54	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	3.73	RATIO	3.00 - 5.00

**INTERPRETATION:**

- Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
- As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
- Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
- NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL.
- Additional testing for Apolipoprotein B, hsCRP, Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement



  
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<b>BARCODE NO.</b>	: 01528566	<b>REPORTING DATE</b>	: 08/Apr/2025 03:49PM
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### MAGNESIUM

MAGNESIUM: SERUM	2.19	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.





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## VITAMINS

### VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	407	pg/mL	190.0 - 890.0
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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 ingestion
4. Hepatocellular injury	4. Contraceptive Hormones
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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