





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

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

NAME : Mr. SHREE KANT

AGE/ GENDER : 40 YRS/MALE **PATIENT ID** : 1743423

COLLECTED BY : 012502020056 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 02/Feb/2025 05:59 PM BARCODE NO. :01524837 **COLLECTION DATE** : 02/Feb/2025 06:00PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 02/Feb/2025 06:01PM

CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Value Unit **Biological Reference interval Test Name**

HAEMATOLOGY HAEMOGLOBIN (HB)

15.2 HAEMOGLOBIN (HB) 12.0 - 17.0gm/dL

by CALORIMETRIC

INTERPRETATION:-

Hemoglobin is the protein molecule in red blood cells that carries oxygen from the lungs to the bodys tissues and returns carbon dioxide from the tissues back to the lungs.

A low hemoglobin level is referred to as ANEMIA or low red blood count.

ANEMIA (DECRESED HAEMOGLOBIN):

- 1) Loss of blood (traumatic injury, surgery, bleeding, colon cancer or stomach ulcer)
- 2) Nutritional deficiency (iron, vitamin B12, folate)
- 3) Bone marrow problems (replacement of bone marrow by cancer)
- 4) Suppression by red blood cell synthesis by chemotherapy drugs
- 5) Kidney failure
- 6) Abnormal hemoglobin structure (sickle cell anemia or thalassemia).

POLYCYTHEMIA (INCREASED HAEMOGLOBIN):

- 1) People in higher altitudes (Physiological)
- 2) Smoking (Secondary Polycythemia)
- 3) Dehydration produces a falsely rise in hemoglobin due to increased haemoconcentration
- 4) Advanced lung disease (for example, emphysema)
- 5) Certain tumors
- 6) A disorder of the bone marrow known as polycythemia rubra vera,
- 7) Abuse of the drug erythropoetin (Epogen) by athletes for blood doping purposes (increasing the amount of oxygen available to the body by chemically raising the production of red blood cells).

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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KOS Diagnostic Lab (A Unit of KOS Healthcare)





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CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit **Biological Reference interval**

CLINICAL CHEMISTRY/BIOCHEMISTRY

LIPID PROFILE · BASIC

	LIPID PROFILE	: BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	195.52	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	318.78 ^H	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM by SELECTIVE INHIBITION	44.12	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	87.64	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
NON HDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	151.4 ^H	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
VLDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	63.76 ^H	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	709.82 ^H	mg/dL	350.00 - 700.00



CHOLESTEROL/HDL RATIO: SERUM

by CALCULATED, SPECTROPHOTOMETRY

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RATIO

4.43H



LOW RISK: 3.30 - 4.40

AVERAGE RISK: 4.50 - 7.0



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Test Name	Value	Unit	Biological Reference interval
LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.99	RATIO	MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	7.23 ^H	RATIO	3.00 - 5.00

INTERPRETATION:

1. 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.

2. 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.

3. 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.

4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, 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

5. 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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Test Name Value Unit Biological Reference interval

LIVER FUNCTION TEST (COMPLETE)

	(
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.56	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.11	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.45	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	37.5	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	50.4 ^H	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.74	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para nitrophenyl phosphatase by amino methyl propanol	57.81	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	44.45	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.63	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by Bromocresol green	4.92	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.71	gm/dL	2.30 - 3.50
A : G RATIO: SERUM	1.82	RATIO	1.00 - 2.00

INTERPRETATION

by CALCULATED, SPECTROPHOTOMETRY

NOTE: To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)



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Test Name Value Unit **Biological Reference interval**

REPORTING DATE

DECREASED:

CLIENT CODE.

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)

2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

PROGNOSTIC SIGNIFICANCE:

1.10011001100110111101111011		
	NORMAL	< 0.65
	GOOD PROGNOSTIC SIGN	0.3 - 0.6
	POOR PROGNOSTIC SIGN	1.2 - 1.6



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Test Name Value Unit Biological Reference interval

UREA

UREA: SERUM 29.71 mg/dL 10.00 - 50.00

by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)



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Test Name Value Unit Biological Reference interval

CREATININE

CREATININE: SERUM 1.11 mg/dL 0.40 - 1.40 by ENZYMATIC, SPECTROPHOTOMETRY

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Value Unit **Biological Reference interval Test Name**

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM 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
PREFFERED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

- 1. 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.

 2.25-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.
- 3. 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 harmone (PTH).

 4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.
- **DECREASED:**
- 1.Lack of sunshine exposure.
- 2.Inadequate intake, malabsorption (celiac disease)
- 3. Depressed Hepatic Vitamin D 25- hydroxylase activity
- 4. Secondary to advanced Liver disease
- 5. Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- 6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism. INCREASED:
- 1. 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 hyperphophatemia.

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 interefere with Vitamin D absorption.

*** End Of Report



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