

(A Unit of KOS Healthcare)



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

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

NAME : Mrs. ANITA BUCHAR

AGE/ GENDER : 59 YRS/FEMALE **PATIENT ID** : 1687467

COLLECTED BY : SURJESH REG. NO./LAB NO. :012412010025

REFERRED BY **REGISTRATION DATE** : 01/Dec/2024 10:09 AM BARCODE NO. :01521793 **COLLECTION DATE** : 01/Dec/2024 10:27AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE :01/Dec/2024 10:50AM

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

Value Unit **Biological Reference interval Test Name**

HAEMATOLOGY HAEMOGLOBIN (HB)

HAEMOGLOBIN (HB) 10.9^L 12.0 - 16.0gm/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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CLIENT ADDRESS: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

LIPID PROFILE: BASIC

CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	234.35 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	77.86	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	74.93	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	143.85 ^H	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	159.42 ^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	15.57	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	546.56	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.13	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0



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Test Name	Value	Unit	Biological Reference interval
			MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.92	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.04 ^L	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

CALCIUM

CALCIUM: SERUM 8.71 mg/dL 8.50 - 10.60

by ARSENAZO III, SPECTROPHOTOMETRY

<u>INTERPRETATION:-</u>

- 1. Serum calcium (total) estimation is used for the diagnosis and monitoring of a wide range of disorders including diseases of bone, kidney, parathyroid gland, or gastrointestinal tract.
- 2. Calcium levels may also reflect abnormal vitamin D or protein levels.
- 3.The calcium content of an adult is somewhat over 1 kg (about 2% of the body weight). Of this, 99% is present as calcium hydroxyapatite in bones and <1% is present in the extra-osseous intracellular space or extracellular space (ECS).
- 4. In serum, calcium is bound to a considerable extent to proteins (approximately 40%), 10% is in the form of inorganic complexes, and 50% is present as free or ionized calcium.

NOTE:-Calcium ions affect the contractility of the heart and the skeletal musculature, and are essential for the function of the nervous system. In addition, calcium ions play an important role in blood clotting and bone mineralization.

HYPOCALCEMIA (LOW CALCIUM LEVELS) CAUSES:-

- 1. Due to the absence or impaired function of the parathyroid glands or impaired vitamin-D synthesis.
- 2. Chronic renal failure is also frequently associated with hypocalcemia due to decreased vitamin-D synthesis as well as hyperphosphatemia and skeletal resistance to the action of parathyroid hormone (PTH).
- 3. NOTE:- A characteristic symptom of hypocalcemia is latent or manifest tetany and osteomalacia.

HYPERCALCEMIA (INCREASE CALCIUM LEVELS) CAUSES:-

- 1.Increased mobilization of calcium from the skeletal system or increased intestinal absorption.
- 2. Primary hyperparathyroidism (pHPT)
- 3. Bone metastasis of carcinoma of the breast, prostate, thyroid gland, or lung

NOTE:-Severe hypercalcemia may result in cardiac arrhythmia.



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Test Name	Value	Unit	Biological Reference interval
	IRON PROFILE		
	IKON PROFILE		
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	45.72	μg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY	255.78	μg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETERY	301.5	μg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	15.16	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)	214.07	mg/dL	200.0 - 350.0

INTERPRETATION:

INTERIALION.			
VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:

1. Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia, anemia of chronic disease and thalassemia syndromes.

2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BINDING CAPACITY (TIBC):

1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:

1.Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.

*** End Of Report ***



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