

(A Unit of KOS Healthcare)



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

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

NAME : Mrs. MONIKA SONI

AGE/ GENDER : 56 YRS/FEMALE **PATIENT ID** : 1826580

COLLECTED BY : SURJESH :012504110021 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 11/Apr/2025 09:35 AM BARCODE NO. :01528791 **COLLECTION DATE** : 11/Apr/2025 09:48AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 11/Apr/2025 10:28AM

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

Value Unit Test Name **Biological Reference interval**

HAEMATOLOGY HAEMOGLOBIN (HB)

HAEMOGLOBIN (HB) gm/dL 12.0 - 16.011.6^L

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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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA 90.84 mg/dL NORMAL: < 100.0

by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

INTERPRETATION
IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.

2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.

3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.



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Test Name	Value	Unit	Biological Reference interval
	LIPID PROFILE	: BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	258.53 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	255.09 ^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	47.41	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	160.1 ^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	211.12 ^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	51.02 ^H	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	772.15 ^H	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	5.45 ^H	RATIO	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	3.38 ^H	RATIO	MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0	
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	5.38 ^H	RATIO	HIGH RISK: > 6.0 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

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

URIC ACID

URIC ACID: SERUM 6.8 mg/dL 2.50 - 6.80

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 leukemais & lymphomas.

4. Polycythemai vera & myeloid metaplasia.

5.Psoriasis.

6. Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCREATION (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 EXCREATION

1.Drugs:-Probenecid, sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosterroids and ACTH, anti-coagulants and estrogens etc.



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IMMUNOPATHOLOGY/SEROLOGY

REPORTING DATE

RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM

RHEUMATOID (RA) FACTOR QUANTITATIVE: IU/mL NEGATIVE: < 18.0

BORDERLINE: 18.0 - 25.0

by NEPHLOMETRY POSITIVE: > 25.0

CLIENT CODE.

INTERPRETATION:-RHEUMATOID FACTOR (RA):

- 1. Rheumatoid factors (RF) are antibodies that are directed against the Fc fragment of IgG altered in its tertiary structure.
 2. Over 75% of patients with rheumatoid arthritis (RA) have an IgM antibody to IgG immunoglobulin. This autoantibody (RF) is diagnostically useful although it may not be etiologically related to RA.

 3. Inflammatory Markers such as ESR & C-Reactive protein (CRP) are normal in about 60 % of patients with positive RA.
- 4. The titer of RF correlates poorly with disease activity, but those patients with high titers tend to have more severe disease course. 5. The test is useful for diagnosis and prognosis of rheumatoid arthritis.

RHEUMATOID ARTHIRITIS:

- 1. Rheumatoid Arthiritis is a systemic autoimmune disease that is multi-functional in origin and is characterized by chronic inflammation of the membrane lining (synovium) joints which ledas to progressive joint destruction and in most cases to disability and reduction of quality life.
- 2. The disease spredas from small to large joints, with greatest damage in early phase.
- 3. The diagnosis of RA is primarily based on clinical, radiological & immunological features. The most frequent serological test is the measurement of RA factor

CAUTION (FALSE POSTIVE):-

- 1. RA factor is not specific for Rheumatoid arthiritis, as it is often present in healthy individuals with other autoimmune diseases and chronic infections. 2. Non rheumatoid and rheumatoid arthritis (RA) populations are not clearly separate with regard to the presence of rheumatoid factor (RF) (15% of RA patients have a nonreactive titer and 8% of nonrheumatoid patients have a positive titer).
- 3. Patients with various nonrheumatoid diseases, characterized by chronic inflammation may have positive tests for RF. These diseases include systemic lupus erythematosus, polymyositis, tuberculosis, syphilis, viral hepatitis, infectious mononucleosis, and influenza.

 4. Anti-CCP have been discovered in joints of patients with RA, but not in other form of joint disease. Anti-CCP2 is HIGHLY SENSITIVE (71%) & more
- specific (98%) than RA factor.
 5. Upto 30 % of patients with Seronegative Rheumatoid arthiritis also show Anti-CCP antibodies.
- 6. The positive predictive value of Anti-CCP antibodies for Rheumatoid Arthiritis is far greater than Rheumatoid factor.

*** End Of Report ***



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