

KOS Diagnostic Lab

(A Unit of KOS Healthcare)



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

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

NAME : Mrs. KAMLA

AGE/ GENDER : 55 YRS/FEMALE **PATIENT ID** : 1570164

COLLECTED BY :012502030019 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 03/Feb/2025 08:53 AM BARCODE NO. :01524861 **COLLECTION DATE** : 03/Feb/2025 08:58AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE :03/Feb/2025 10:56AM

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

Value Unit **Biological Reference interval Test Name**

CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA NORMAL: < 100.0 117.05^H mg/dL

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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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST





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Test Name Value Unit Biological Reference interval

URIC ACID

URIC ACID: SERUM 4.83 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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Value Unit **Biological Reference interval Test Name**

IMMUNOPATHOLOGY/SEROLOGY RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM

RHEUMATOID (RA) FACTOR QUANTITATIVE: IU/mL 0.96 NEGATIVE: < 18.0

BORDERLINE: 18.0 - 25.0 **SERUM**

by NEPHLOMETRY POSITIVE: > 25.0

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 antibody in the second has a strategies and the part of the PA. 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 tast 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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