

# **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

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**NAME** : Mrs. GEETANJALI

AGE/ GENDER : 28 YRS/FEMALE **PATIENT ID** : 1675954

**COLLECTED BY** REG. NO./LAB NO. :012411190035

REFERRED BY **REGISTRATION DATE** : 19/Nov/2024 12:23 PM BARCODE NO. :01521087 **COLLECTION DATE** : 19/Nov/2024 12:25PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 19/Nov/2024 02:09PM

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

**Value** Unit **Biological Reference interval Test Name** 

# **HAEMATOLOGY**

### **ERYTHROCYTE SEDIMENTATION RATE (ESR)**

ERYTHROCYTE SEDIMENTATION RATE (ESR)

mm/1st hr

by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

### INTERPRETATION:

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and autoimmune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.

2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein

3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

### CONDITION WITH LOW ESR

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis), and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR. NOTE:

- NOTE:
   ESR and C reactive protein (C-RP) are both markers of inflammation.
   Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
   CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.
   If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
   Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
   Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspiring cortisons, and quining may decrease it. aspirin, cortisone, and quinine may decrease it



CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST



KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana



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COLLECTED BY : REG. NO./LAB NO. : 012411190035

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 : 19/Nov/2024 12:23 PM

 BARCODE NO.
 : 01521087
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 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 19/Nov/2024 01:19 PM

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

Test Name Value Unit Biological Reference interval

# CLINICAL CHEMISTRY/BIOCHEMISTRY URIC ACID

URIC ACID: SERUM 3.95 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.



DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUĞAM CHOPRA CONSULTANT PATHOLOGIST MBBS , MD (PATHOLOGY)



KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: Ilnd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana



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

RHEUMATOID (RA) FACTOR QUANTITATIVE: IU/mL NEGATIVE: < 18.0 271.58<sup>H</sup>

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 by a strategically related to 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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