



PATIENT NAME : ISHAMMA

REF. DOCTOR : SELF

ISHAMMA	ACCESSION NO : <b>4182YK007572</b>	AGE/SEX : 79 Years Female
	PATIENT ID : ISHAF2009474182	DRAWN : 19/11/2025 11:56:01
	CLIENT PATIENT ID:	RECEIVED : 19/11/2025 11:59:17
	ABHA NO :	REPORTED : 20/11/2025 17:23:37

Test Report Status	Results	Biological Reference Interval	Units
<b>Preliminary</b>			

**BIOCHEMISTRY**

**SERUM PROTEIN CAPILLARY ELECTROPHORESIS MIPM** RESULT PENDING



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ULR No.666000016172685-4126



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**SERUM FREE LIGHT CHAINS (KAPPA & LAMBDA)** RESULT PENDING

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**HAEMATOLOGY - CBC**

**CBC + ESR**

**BLOOD COUNTS, EDTA WHOLE BLOOD**

HEMOGLOBIN	8.3 (Rechecked)	12.0 - 15.0	g/dL
<small>METHOD : PHOTOMETRIC METHOD BY SLS HAEMOGLOBIN</small>			
RED BLOOD CELL COUNT	<b>2.68 Low</b>	3.80 - 4.80	mil/ $\mu$ L
<small>METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION</small>			
WHITE BLOOD CELL COUNT	<b>2.32 Low</b>	4.0 - 10.0	thou/ $\mu$ L
<small>METHOD : AUTOMATED CELL COUNTER</small>			
PLATELET COUNT	70 (Rechecked on smear)	150 - 410	thou/ $\mu$ L
<small>METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION</small>			

**RBC AND PLATELET INDICES**

HEMATOCRIT	<b>26.1 Low</b>	36.0 - 46.0	%
<small>METHOD : RBC PULSE HEIGHT DETECTION</small>			
MEAN CORPUSCULAR VOLUME (MCV)	97.4	83.0 - 101	fL
<small>METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM</small>			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	31.0	27.0 - 32.0	pg
<small>METHOD : CALCULATED PARAMETER</small>			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	31.8	31.50 - 34.50	g/dL
<small>METHOD : CALCULATED PARAMETER</small>			
RED CELL DISTRIBUTION WIDTH (RDW)	<b>17.3 High</b>	11.60 - 14.0	%
<small>METHOD : CALCULATED PARAMETER</small>			
MEAN PLATELET VOLUME (MPV)	8.5	6.80 - 10.90	fL
<small>METHOD : FLOW CYTOMETRY</small>			

**WBC DIFFERENTIAL COUNT**

SEGMENTED NEUTROPHILS	47	40.0 - 80.0	%
<small>METHOD : FLUORESCENCE FLOW CYTOMETRY</small>			
LYMPHOCYTES	37	20.0 - 40.0	%
<small>METHOD : FLUORESCENCE FLOW CYTOMETRY</small>			
MONOCYTES	3	2.0 - 10.0	%
<small>METHOD : FLUORESCENCE FLOW CYTOMETRY</small>			

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EOSINOPHILS		<b>13 High</b>	1.0 - 6.0	%
METHOD : FLUORESCENCE FLOW CYTOMETRY				
BASOPHILS		0	0.0 - 1.0	%
METHOD : FLUORESCENCE FLOW CYTOMETRY				
ABSOLUTE NEUTROPHIL COUNT		<b>1.09 Low</b>	2.0 - 7.0	thou/μL
METHOD : FLUORESCENCE FLOW CYTOMETRY				
ABSOLUTE LYMPHOCYTE COUNT		<b>0.86 Low</b>	1.0 - 3.0	thou/μL
METHOD : FLUORESCENCE FLOW CYTOMETRY				
ABSOLUTE MONOCYTE COUNT		<b>0.08 Low</b>	0.20 - 1.0	thou/μL
METHOD : FLUORESCENCE FLOW CYTOMETRY				
ABSOLUTE EOSINOPHIL COUNT		0.30	0.02 - 0.50	thou/μL
ABSOLUTE BASOPHIL COUNT		0		thou/μL

**ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD**

SEDIMENTATION RATE (ESR)	<b>130 High</b>	0 - 35	mm at 1 hr
METHOD : MODIFIED WESTERGRN			

**Interpretation(s)**

BLOOD COUNTS,EDTA WHOLE BLOOD-The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.

RBC AND PLATELET INDICES-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait (<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT-The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to show mild disease.

(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients A.-P. Yang, et al. International Immunopharmacology 84 (2020) 106504)

This ratio element is a calculated parameter and out of NABL scope.

ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD- **TEST DESCRIPTION** :- Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of clear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

- ESR is not diagnostic it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition.CRP is superior to ESR because it is more sensitive and reflects a more rapid change.

**TEST INTERPRETATION** : **Increase** in: Infections, Vasculitides, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy, Estrogen medication, Aging.

Finding a very accelerated ESR(>100 mm/hour) in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).

In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm /hr(95 if anemic). ESR returns to normal 4th week post partum.

**Decreased** in: Polycythemia vera, Sickle cell anemia

**LIMITATIONS** : **False elevated** ESR : Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia

**False Decreased** : Poikilocytosis,(SickleCells,spherocytes),Microcytosis, Low fibrinogen, Very high WBC counts, Drugs(Quinine, salicylates)

REFERENCE : Nathan and Oski's Haematology of Infancy and Childhood, 5th edition 2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin 3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis,10th edition.

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**HAEMATOLOGY**

**RETICULOCYTE COUNT, EDTA WHOLE BLOOD**

RETICULOCYTE COUNT	1.6	0.5 - 2.5	%
METHOD : SUPRAVITAL STAIN - BRILLIANT CRESYL BLUE AND MICROSCOPY			

**Comments**

This is the CORRECTED RETICULOCYTE count

**Interpretation(s)**

RETICULOCYTE COUNT, EDTA WHOLE BLOOD-Reticulocytes are juvenile red cells and contain a reticular (mesh-like) network of RNA. The number of reticulocytes is a good indicator of erythropoetic activity, and can be used to monitor the response to treatment of anemia. Decrease in reticulocytes can be attributed to suppression of erythropoiesis due to chemotherapy, aplastic anemia and other hypoproliferative anemias.

An increased number of reticulocytes (reticulocytosis) indicates accelerated erythropoiesis either as compensation for excessive red cell loss (e.g. hemolysis or bleeding) or, when a marrow starved of iron, vitamin B12 or folate receives the appropriate nutrient.

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**NEPHELOMETRY**

**CERULOPLASMIN, SERUM**

CERULOPLASMIN	29.10	Female : 16-45	mg/dL
METHOD : IMMUNOTURBIDIMETRIC ASSAY			

**COMPLEMENT C3, SERUM**

COMPLEMENT C3	140.0	Adult : 90-180	mg/dL
METHOD : IMMUNOTUBIDIMETRY			

**COMPLEMENT C4, SERUM**

COMPLEMENT C4	39.3	Adult : 10.0-40.0	mg/dL
METHOD : IMMUNOTUBIDIMETRY			

**Interpretation(s)**

CERULOPLASMIN, SERUM-Ceruloplasmin, a late acute phase reactant is the principal Copper containing protein of plasma. The most important clinical application of Ceruloplasmin is in the diagnosis of

Wilson disease, where concentrations of Ceruloplasmin are reduced. On a pathochemical level, the disease, which is accompanied by reduced ceruloplasmin synthesis, occurs as a consequence of missing Cu(2+) incorporation into the molecule due to defective metallothionein. This results in pathological deposits of copper in the liver (with accompanying development of cirrhosis), brain (with neurological symptoms), cornea (Kayser-Fleischer ring), and kidneys (hematuria, proteinuria, aminoaciduria). In homozygous carriers, ceruloplasmin levels are severely depressed. Heterozygous carriers exhibit either no decrease at all or just a mild decrease.

The rare Menkes syndrome is a genetically caused copper absorption disorder with concomitant lowering of the ceruloplasmin level. Protein loss syndromes and liver cell failures are the most important causes of acquired ceruloplasmin depressions.

Low levels of Ceruloplasmin may also be found in malnutrition, malabsorption, nephrosis and severe liver disease. Ceruloplasmin is itself affected by infections and liver function. Birth control pills increase Ceruloplasmin, so does pregnancy.

**Cautions**

Ceruloplasmin levels are affected by infections (ceruloplasmin is a late acute phase reactant) and liver function.

Birth control pills and pregnancy increase ceruloplasmin levels.

COMPLEMENT C3, SERUM-Complement is a complex biological system which works in conjunction with antibody and other factors to protect the body from invasion by pathogens. When activated by either the classical or alternative pathway Complement acts on biological membranes and may cause cell death. Complement C3 and Complement C4 levels are important in determining inherited or acquired deficiencies.

Low values of C3 in serum or plasma confirm complement fixation or degradation of C3, in vitro. Profound hypocomplementemia associated with grossly low levels of C3, are usual in post - Streptococcal glomerulonephritis. Decreased levels of C3 in Systemic Lupus Erythematosus suggest renal involvement. C3 deficiency is the most severe of all inherited complement defects and associated with severe bacterial infections. Low C3 levels may also be detected in severe liver disease.

C3 levels may rise in a variety of inflammatory and necrotic disorders as part of the acute-phase plasma protein response.

COMPLEMENT C4,SERUM-Complement is a complex biological system which works in conjunction with antibody and other factors to protect the body from invasion by pathogens. When activated by either the classical or alternative pathway Complement acts on biological membranes and may cause cell death. Complement C4 levels are

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important in determining inherited or acquired deficiencies.

C4 may be decreased in systemic lupus erythematosus, early glomerulonephritis, immune complex disease, cryoglobulinemia, hereditary angioedema, and congenital C4 deficiency. Low C4 with high anti - ds DNA antibodies confirms the diagnosis of Systemic Lupus Erythematosus (SLE) and may help monitor SLE activity.

C4 levels may rise in a variety of inflammatory and necrotic disorders as part of the acute-phase plasma protein response.

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**SEROLOGY**

**CRP, SEMI-QUANTITATIVE, SERUM**

C-REACTIVE PROTEIN	<b>13.80 High</b>	Cut-off : <5	mg/L
METHOD : IMMUNOTUBIDIMETRY			

**Comments**

Kindly correlate clinically.  
Kindly contact lab within 24 hrs, if clinically not correlated.

**Interpretation(s)**

CRP, SEMI-QUANTITATIVE, SERUM-C - reactive protein (CRP) is an acute phase reactant protein that has the property of showing elevations in concentrations in response to stressful or inflammatory states that occur with infection, injury, surgery, trauma or other tissue necrosis.

Synthesis of CRP increases within 4-6 hours of onset of inflammation, reaching peak values within 1-2 days. CRP levels also fall quickly after resolution of inflammation since its half life is 6 hours. The main limitation of CRP is in its non-specific response and should not be interpreted without a complete clinical history and evaluation.: Latex particle agglutination

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**EIA - AUTO IMMUNE**

**ANTI - CCP ANTIBODIES,SERUM**

ANTI - CCP ANTIBODIES	<7.00	< or = 17.00	U/mL
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METHOD : ECLIA

**Interpretation(s)**

ANTI - CCP ANTIBODIES,SERUM-Clinical Utility:-

- Rheumatoid arthritis (RA) is a systematic autoimmune disease that is multi-functional in origin and is characterized by chronic inflammation of the membrane lining(synovium) joints which commonly leads to progressive joint destruction and in most cases to disability and reduction of quality of life. The disease spreads from small to large joints, with the greatest damage in early phase.
  - The diagnosis of RA is primarily based on clinical, radiological and immunological features. The most frequent serological test is the measurement of rheumatoid factor (RF). The IgM class is the most common and is found in 60-80% of RA patients. RF is not specific for RA, as it is often present in healthy individuals and patients with other autoimmune diseases and chronic infections. Citrullinated proteins have been discovered in the joints of patients with rheumatoid arthritis but not in other forms of joint disease. The Citrullinated proteins in the joints correspond to the presence of the citrulline antibodies in the blood and suggest a possible role for these antibodies in the development of rheumatoid arthritis.
  - Anti-CCP test is used for the detection of the IgG class of autoantibodies specific to cyclic Citrullinated peptide (CCP) in human serum or plasma (EDTA). Autoantibody levels represent one parameter in a multi-criterion diagnosis process, encompassing both clinical and laboratory-based assessments.
  - The citrulline antibody appears early in the course of rheumatoid arthritis and is present in the blood of most patients with the disease. When the citrulline antibody is detected in a patient's blood, there is 90-95% likelihood that the patient has rheumatoid arthritis. The test for the citrulline antibody is therefore useful in the diagnosis of patients with unexplained joint inflammation, especially when the traditional blood test for rheumatoid factor is negative. The citrulline antibody also has prognostic (predictive) value since it is associated with a greater tendency towards more destructive forms of rheumatoid arthritis.
  - Detection of anti -CCP antibodies is used as an aid in the diagnosis of Rheumatoid arthritis(RA) and should be used in conjunction with other clinical information.
  - A negative anti CCP test result after treatment for Rheumatoid arthritis (RA) or other conditions may indicate a positive response to treatment, but it doesn't always mean the disease is gone. While anti-CCP levels often decrease with successful treatment, they don't always correlate perfectly with disease activity, and some individuals may remain positive despite symptom improvement
- Importance of Monitoring:  
Even with a negative anti-CCP test, ongoing monitoring is crucial to assess overall disease activity and adjust the treatment as needed.



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**MICRO BIOLOGY**

<b>ANA</b>				
ANA	<b>2.2 High</b>	<1.0 : Negative > OR = 1.0 : Positive		RATIO

<b>DS DNA / (ANTI)</b>				
DS DNA / (ANTI)	29.1	<100 : Negative > or =100 : Positive		IU/ml

**Interpretation(s)**  
ANA-METHOD - ELISA  
SAMPLE TYPE - SERUM

The ANA Screen is done to detect IgG antibodies to nuclear antigens. ANA positivity is seen in patients with a number of systemic autoimmune disease. Males and females older than 80 years of age have a 50% incidence of low titer ANA. Various medications can induce a "lupoid" condition. ELISA sometimes may give false positive/negative results. ANA detection by IFA is considered as a gold standard method to confirm the results. Indirect immunofluorescence assay (IFA) is more useful in detecting unknown antigens also which cannot be detected by ELISA.

DS DNA / (ANTI)-Methods:ELISA

Sample Type : Serum

Anti ds-DNA is a specific assay for confirming the diagnosis of systemic lupus erythematosus (SLE). Antibodies to DNA, either single or double-stranded, are found primarily in systemic lupus erythematosus, and are important, but not sufficient for diagnosing that condition. Such antibodies are present in 80% to 90% of SLE cases. They are also present in smaller fractions of patients with other rheumatic disorders, and in chronic active hepatitis, infectious mononucleosis, and biliary cirrhosis. Monitoring levels of anti-DNA antibody may be of use in evaluating response to therapy, but should be regarded as a guide rather than a rigid dictator of treatment. Antibody levels correlate particularly well with activity of lupus nephritis.

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METALS	
<u>COPPER, SERUM</u>	RESULT PENDING

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