

(A Unit of KOS Healthcare)



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

NAME : Mrs. RANJU DEVI

AGE/ GENDER : 42 YRS/FEMALE PATIENT ID : 1653815

COLLECTED BY : REG. NO./LAB NO. : 012410260034

REFERRED BY: DR HARDEEP SINGHREGISTRATION DATE: 26/Oct/2024 11:00 AMBARCODE NO.: 01519579COLLECTION DATE: 26/Oct/2024 11:27 AM

CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 26/Oct/2024 12:56PM

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

Test Name Value Unit Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY CERULOPLASMIN

CERULOPLASMIN: SERUM 43.92 mg/dL 22.0 - 61.0

by NEPHELOMETRY

INTERPRETION:

- 1. Ceruloplasmin is an acute phase protein and a transport protein. This glycoprotein belongs to the alpha 2-globulin electrophoretic fraction and contains 8 copper atoms per molecule.
- 2.Incorporation of copper into the structure occurs during the synthesis of ceruloplasmin in the hepatocytes. After secretion from the liver, ceruloplasmin migrates to copper-requiring tissue where the copper is liberated during catabolism of the ceruloplasmin molecule.
- 3. Main function of ceruloplasmin is to regulate ionic state of iron and transportation of copper to tissues
- 4.In addition to transporting copper, ceruloplasmin has a catalytic function in the oxidation of iron (Fe[2+] to Fe[3+]), polyamines, catecholamines, and polyphenols.
- 5.Decreased concentrations occur during recessive autosomal hepatolenticular degeneration (Wilson disease 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.
- 6. 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.

NOTE

- 1. Ceruloplasmin is a sensitive acute phase reactant, increases occur during acute and chronic inflammatory processes. Birth control pills and pregnancy increase ceruloplasmin levels. Testing should be avoided if any of the above history is elicited prior to testing.
- 2. Factors which increase ceruloplasmin synthesis are cytokines, pregnancy & estrogens.
- 3. Ceruloplasmin levels are not always extremely low in patients with Wilson disease.



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

Value Unit **Biological Reference interval Test Name**

IMMUNOPATHOLOGY/SEROLOGY ANTI SMOOTH MUSCLE ANTIBODY (ASMA): ELISA

ANTI SMOOTH MUSCLE ANTIBODY (ASMA) - ELISA 36.4 by ELISA (ENZYME LINKED IMMUNOASSAY)

IU/mL

NEGATIVE: 0.0 - 40.0 BORDERLINE: 40.0 - 45.0 HIGHLY POSITIVE: >45.0

INTERPRETATION:

1. Smooth muscle autoantibodies (SMA) are found in approximately 3% of normal adult caucasians.
2. High titres (>=1:160) of SMA are found in approximately 97% of patients with autoimmune chronic active hepatitis. SMA are found less frequently in uveitis, drug induced hepatitis, alcoholic liver disease, primary pulmonary hypertension and transiently in acute hepatitis and other viral infections including infectious mononucleosis.

3.Low titer antibodies may be found in the sera of patients with viral infections, malignancies and in the normal population.
4.The presence of SMA is not predictive of the development of liver disease.

5. The absence of ASMA indicates non autoimmune forms of chronic hepatitis.



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Value Unit **Biological Reference interval Test Name**

IMMUNOGLOBIN IgG

IMMUNOGLOBIN-G (IgG): SERUM 16.91^H 7.0 - 16.0gm/L

by NEPHLOMETRY

INTERPRETATION: 1.Immunoglobulin is a humoral antibody consisting of two light and two heavy chains in the molecule.

2. Approximately 80% of serum immunoglobulins is IgG. Its major function is neutralization of toxin in tissues spaces.

3. Antibodies of the IgG class are produced in response to most bacteria and viruses. IgG is the only immunogloblin that can cross the placental barrier and provide passive immune protection for fetus and new born till about 6 month.

4.Increased levels may be seen in SLE, chronic liver diseases, infectious diseases and cystic fibrosis. Monoclonal IgG increases in IgG myeloma. 5.Decreased synthesis of IgG is found in congenital/acquired immunodeficiencies and in selective subclass deficiency such as bruton type

agammaglobinulinemia.

6.Decreased IgG concentrations are seen in protein-losing enteropathies, nephrotic syndrome and in skin burns.



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Value Unit **Biological Reference interval Test Name**

SPECIAL INVESTIGATIONS

LIVER KIDNEY MICROSOMAL (LKM) - 1 ANTIBODY - WITH REFLEX TO TITRES: IFA

LIVER KIDNEY MICROSOMAL (LKM) ANTIBODY - IFA NEGATIVE (-ve)

by IFA (IMMUNO FLUORESCENT ASSAY)

INTERPRETATION:

1. Autoimmune reactivities are not by themselves diagnostic, but must be correlated with other laboratory & clinical findings.
2. Autoantibodies which react with liver - kidney microsomal (LKM) antigens occurs in various forms of chronic hepatitis.
3. Test conducted on Serum Comments Three types of Liver Kidney Microsomal (LKM) antibodies are detected of which Anti LKM1 is most important. Serum antibodies which are directed against the target antigen cytochrome P450 (LKM- I) are considered as markers of Autoimmune hepatitis (AIH) Type II. Anti LKM1 antibodies are detected in Autoimmune hepatitis Type 2 (80-95%) and also in Hepatitis associated with Autoimmune polyglandular disease Type 1.
4. 50 - 75% of all individuals affected by this disease are children. 3. Extrahepatic syndromes such as arthralgia's, glomerulonephritis, vitiligo and chronic inflammatory bowel diseases are frequently associated with this form of AIH.
5. Anti LKM2 antibodies are less frequently detected and are usually associated with Drug induced Hepatitis.

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6. Anti LKM3 antibodies are rarely detected and are usually associated with Autoimmune Hepatitis Type 2 (8-19%), Hepatitis C (6%) and Hepatitis D (6-13%).



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ANTI NUCLEAR ANTIBODY/FACTOR (ANA/ANF) - WITH REFLEX TO TITRES: IFA (HEP-2)

ANTI NUCLEAR ANTIBODY (ANA) - IFA, HEp2

NEGATIVE (-ve)

NEGATIVE (-ve)

by IFA (IMMUNO FLUORESCENT ASSAY)

INTERPRETATION:

1.Anti Nuclear antibody (ANA) in dilutions is recommended for all positive results and follow up

2.Immunofluorescence microscopy using human cellular extracts like HEp-2 cells is a sensitive test for detection of serum antibodies that react specifically with various cellular proteins and nucleic acids

3.Test conducted on Serum

INTERPRETATION GUIDELINES: (Sample screening Dilution - 1:100):

Negative: No Immunofluorescence

+: Weak Positive (1:100)

++: Moderate Positive (1:320)

+++ : Strong Positive (1:1000)

++++: Very strong Positive (1:3200)

COMMENTS:

Anti Nuclear antibody (ANA / ANF) is a group of autoantibodies directed against constituents of cell nuclei including DNA, RNA & various nuclear proteins. These autoantibodies are found with high frequency in patients with connective tissue disorders specially SLE. Since positive ANA results have been reported in healthy individuals, these reactivities are not by themselves diagnostic but must be correlated with other laboratory and clinical findings.

PATTERN	DISEASE ASSOCIATION
NUCLEAR	
Homogenous	SLE & other connective tissue disorders, Drug induced SLE
Peripheral	SLE & other connective tissue disorders
Speckled Coarse	Mixed connective Tissue Disorders (MCTD), Scleroderma-Polymyositis Overlap Syndrome, Raynauds Phenomenon, Psoariasis, Sjogrens Syndrome, Systemic Sclerosis.
Speckled Fine	SLE,Sjogrens syndrome,Scleroderma,Myositis,MCTD
NUCLEAR DOTS	



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Few		Auto-immune & Viral disease- Primary Biliay Cirrhosis & Chronic Active Hepatitis, Rarely Collagen Vascular disease		
Multiple	Primary Biliary Cirrhosis (>30%)	Primary Biliary Cirrhosis (>30%)		
Centromere	CREST syndrome, Progresive Systemic Sclerosis			
NUCLEOLAR				
Homogeneous	Scleroderma, Myositis, Raynauds Phenomena, SLE & Rheumatoid arthiritis			
Clumpy	Systemic sclerosis & Scleroderma			
CYTOPLASMIC				
Mitochondrial	Primary Biliary Cirrhosis, Scleroderma & Overlap syndrome			
Ribosomal	SLE (10-20%)			

*** End Of Report ***



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