

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

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

NAME	: Mr. HEMANT MANCHANDA	PATIENT ID	: 1782525
AGE/ GENDER	: 34 YRS/MALE	REG. NO./LAB NO.	: 012503070076
COLLECTED BY	:	REGISTRATION DATE	: 07/Mar/2025 04:59 PM
REFERRED BY	: ROTARY HOSPITAL (AMBALA CANTT)	COLLECTION DATE	: 07/Mar/2025 05:57PM
BARCODE NO.	: 01526665	REPORTING DATE	: 07/Mar/2025 06:39PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

CERULOPLASMIN

CERULOPLASMIN: SERUM by NEPHELOMETRY	20.13^L	mg/dL	22.0 - 61.0
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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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IMMUNOPATHOLOGY/SEROLOGY

LIVER KIDNEY MICROSOMAL (LKM) - 1 ANTIBODY: ELISA

LIVER KIDNEY MICROSOMAL (LKM) ANTIBODY - ELISA 3.3	RU/mL	NEGATIVE: < 25.0
: SERUM		BORDERLINE: 20.0 - 25.0
by ELISA (ENZYME LINKED IMMUNOSORBENT ASSAY)		POSITIVE: > 25.0

INTERPRETATION:

1. Autoimmune hepatitis (AIH) is a distinct chronic inflammatory liver disease, characterized by the attack of the immune system directed against "self" antigens, especially those expressed in the liver 1, 2.
2. It occurs in both sexes and all age groups, however, women are more likely victims of AIH than men. In women, 70 % of diagnosed cases of AIH occur between the ages of 15 and 40.
3. Hepatomegaly and splenomegaly are the most common pathological findings associated with AIH.
4. Abnormalities of the immune system that mark AIH include autoantibodies to liver antigens, hyper-gammaglobulinemia, and an increased CD4/CD8 ratio in peripheral blood and liver.
5. Liver-Kidney Microsomal (LKM1) antibodies can be induced not only by autoimmune mechanisms, but also by drugs such as tienic acid, dihydralazine, halothane, phenytoin, phenobarbital, carbamazepine and by Hepatitis C and D infections




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Test Name	Value	Unit	Biological Reference interval
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IMMUNOGLOBIN IgG

IMMUNOGLOBIN-G (IgG): SERUM by NEPHLOMETRY	10.14	gm/L	7.0 - 16.0
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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 immunoglobulin 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 agammaglobulinemia.
- 6.Decreased IgG concentrations are seen in protein-losing enteropathies, nephrotic syndrome and in skin burns.




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TUMOUR MARKER

ALPHA FETO PROTEIN (AFP): TUMOR MARKER

ALPHA FETO PROTEIN (AFP)	4.814	ng/mL	0.0 - 10.0
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TUMOUR MARKER: SERUM

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:

1. Alpha-fetoprotein (AFP) is a glycoprotein that is produced in early fetal life by the liver, GIT & yolk sac and by a variety of tumors including hepatocellular carcinoma, hepatoblastoma, and nonseminomatous germ cell tumors of the ovary and testis (eg, yolk sac and embryonal carcinoma). Most studies report elevated AFP concentrations in approximately 70% of patients with hepatocellular carcinoma. Elevated AFP concentrations are found in 50% to 70% of patients with non seminomatous testicular tumors.
2. It is a major component of fetal plasma, reaching a peak concentration of 3mg/mL at 12 weeks of gestation. Following birth, it clears from circulation, falling to 100 ng/ mL by 150 days and reaching adult values by end of 1 year.
3. AFP is elevated during pregnancy. Persistence of AFP in the mother following birth is a rare hereditary condition.
3. Neonates have markedly elevated AFP levels (>100,000 ng/mL) that rapidly fall to below 100 ng/mL by 150 days and gradually return to normal over their first year.
4. Concentrations of AFP above the reference range also have been found in serum of patients with benign liver disease (eg, viral hepatitis, cirrhosis), gastrointestinal tract tumors and, along with carcinoembryonic antigen in ataxia telangiectasia.

CAUTION:

1. It is not recommended to use this assay for the initial diagnosis of the above mentioned malignancies.
2. It is best used for monitoring of therapy and to look for relapse of malignancies that have been surgically excised or cleared with chemo/radiotherapy.
3. Failure of the AFP value to return to normal by approximately 1 month after surgery suggests the presence of residual tumor.
4. Elevation of AFP after remission suggests tumor recurrence; however, tumors originally producing AFP may recur without an increase in AFP.

NOTE:

A difference of > 20% between two measurements is considered to be medically significant. The assay is used only as an adjunct to diagnosis and monitoring/ diagnosis should be confirmed by other tests/procedures.




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Test Name	Value	Unit	Biological Reference interval
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SPECIAL INVESTIGATIONS

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:

- Immunofluorescence microscopy using human cellular extracts like Hep-2 cells is sensitive for detection of serum antibodies that react specifically with various cellular proteins and nucleic acid.
- Test conducted on serum
- Patients are reported as per international consensus ANA Patterns (ICAP)

INTERNATIONAL GUIDELINES FOR GRADING

GRADE	REMARKS
Negative (-ve)	No fluorescence
1+	Minimum fluorescence
2+	Mildly positive
3+	Significantly positive
4+	Strongly positive

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 (ICAP)	ICAP CODE	ANTIGEN ASSOCIATION	DISEASE ASSOCIATION
NUCLEAR PATTERNS			
Homogenous	AC-1	dsDNA, nucleosomes, histones	SLE, Drug-induced lupus, Juvenile idiopathic




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Test Name	Value	Unit	Biological Reference interval
			arthritis
Speckled	AC-2,4,5	hnRNP, U1RNP, Sm, SS-A/Ro (Ro 80), SS-B/La, RNA polymerase III, Mi-2, Ku	MCTD, SLE, DM, SSc/PM overlap
Dense fine speckled	AC-2	DFS70/LEDGF	Rare in SLE, Sjogren's syndrome, SSc
Fine speckled	AC-4	SS-A/Ro (Ro 80), T1F1β, SS-B/La, Mi-2, T1F1γ, Ku, RNA helicase A, replication protein A	Sjogren's syndrome, SLE, DM, SSc/PM overlap
Large/Coarse speckled	AC-5	hnRNP, U1RNP, Sm, RNA polymerase III	MCTD, SLE, SSc
Centromere	AC-3	CENP-A/B	Limited cutaneous SSc, PBC
Discrete nuclear dots	AC-6,7		
Multiple nuclear dots	Ac-6	Sp-100, PML proteins, MJ/NXP-2	PBC, SARD, PM/DM
Few nuclear dots	Ac-7	P80-coilin, SMN	Sjogren's syndrome, SLE, SSc, PM, asymptomatic individuals
Nucleolar	AC-8,9,10		
Nucleolar homogenous	AC-8	PM/Scl-75, PM/Scl-100, Thi/To, B23/nucleophosmin, nucleolin, No55/SC65	SSc, SSc/PM overlap
Nucleolar clumpy	AC-9	U3-smRNP/fibrillarin	SSc
Nucleolar punctate	Ac-10	RNA polymerase 1, hUBF/NOR-90	SSc, Sjogren's syndrome
Nuclear envelope	AC-11,12		
Smooth nuclear envelope	AC-11	Lamin A,B,C or lamin associated proteins	SLE, Sjogren's syndrome, Seronegative arthritis




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Test Name	Value	Unit	Biological Reference interval
Punctate nuclear envelope	AC-12	Nuclear pore complex proteins (gp210)	PBC
Pleomorphic	AC-13,14		
PCNA-like	AC-13	PCNA	SLE, other conditions
CENP-F like	AC-14	CENP-F	Cancer, other conditions
CYTOPLASMIC PATTERNS			
Fibrillar	AC-15,16,17		
Linear/actin	AC-15	Actin, non-muscle myosin, MCTD	MCTD, Chronic active hepatitis, Liver cirrhosis, Myasthenia gravis, Crohn's disease, PBC, Long term hemodialysis, rare in SARD other than MCTD
Filamentous/microtubules	AC-16	Vimentin, cytokeratins	Infections or inflammatory conditions, Long term hemodialysis, Alcoholic liver disease, SARD, Psoriasis, healthy controls
Segmental	AC-17	Alpha-actin, vinuculin, tropomyosin	Myasthenia gravis, Crohn's disease, Ulcerative colitis
Speckled	AC-18,19,20		
Discrete dots/GW body like	AC-18	SGW182, Su/Ago2,	PBC, SARD, neurological and autoimmune conditions
Dense fine speckled	AC-19	PL-7, PL-12, ribosomal P proteins	Anti-synthetase syndrome, PM/DM, SLE, Juvenile SLE, Neuropsychiatric SLE




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Test Name	Value	Unit	Biological Reference interval
Fine speckled	AC-20	Jo-1/histidyl-Trna synthetase	Anti-synthetase syndrome, PM/DM, limited SSc, Idiopathic pleural effusion
Reticular/AMA (Mitochondrial)	AC-21	PDC-E2/M2, BCOADC-E2 OGDC-E2, E1 α subunit of PDC, E3BP/proteinX	Common in PBC, SSc, rare in other SARD
Polar/ Golgi like	AC-22	Giantin/macrogolgin, golgin-97, golgin-245	Rare in Sjogren's syndrome, SLE, RA, MCTD,GPA, Idiopathic cerebellar ataxia, Paraneoplastic cerebellar degeneration,viral infections
Rods and rings	AC-23	IMPDH2, others	HCV patients post IFN/Ribavirin therapy,rare in SLE, Hashimoto's and healthy controls
MITOTIC PATTERNS			
Centrosome	AC-24	Pericentrin, ninein, Cep250, Cep110	Rare in SSc, Raynaud's phenomenon, infections (viral and mycoplasma)
Spindle fibres	AC-25	HsEg5	Rare in Sjogren's syndrome, SLE, other SARD
NuMA like	AC-26	Centrophilin	Sjogren's syndrome, SLE, other
Intracellular bridges	AC-27	Aurora kinase B, CENP-E,MSA-2, KIF-14, MKLP-1	Rare in SSc, Raynaud's phenomenon, malignancy




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Test Name	Value	Unit	Biological Reference interval
Mitotic chromosome coat	AC-28	Modified histone H3, MCA-1	Rare in Discoid lupus erythematous, Chronic lymphocytic leukemia, Sjogren's syndrome, and Polymyalgia rheumatica




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Test Name	Value	Unit	Biological Reference interval
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ANTI SMOOTH MUSCLE ANTIBODY (ASMA) - WITH REFLEX TO TITRES: IFA

ANTI SMOOTH MUSCLE ANTIBODY (ASMA) - IFA	NEGATIVE (-ve)	NEGATIVE (-ve)
<small>by IFA (IMMUNO FLUORESCENT ASSAY)</small>		

INTERPRETATION:

1. Smooth muscle autoantibodies (SMA) are found in approximately 3% of normal adult caucasians.
2. High titres ($\geq 1:160$) 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.

*** End Of Report ***




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