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NAME	**: Mr. NAVNEET**		
AGE/ GENDER	: 33 YRS/MALE	**PATIENT ID**	: 1188947
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012407210002**
REFERRED BY	:	**REGISTRATION DATE**	: 21/Jul/2024 07:09 AM
BARCODE NO.	: 01513523	**COLLECTION DATE**	: 21/Jul/2024 07:55AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 21/Jul/2024 10:17AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

FERRITIN

FERRITIN: SERUM	10	ng/mL	10.0 - 290.0

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy.

DECREASED:

1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.
2. Hypothyroidism.
3. Vitamin-C deficiency.

INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

1. Hemochromatosis or hemosiderosis.
2. Wilson Disease.

INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

1. Transfusion overload
2. Excess dietary Iron
3. Porphyria Cutanea tada
4. Ineffective erythropoiesis.

INCREASED FERRITIN WITHOUT IRON OVERLOAD:

1. Liver disorders (NASH) or viral hepatitis (B/C).
2. Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
3. Leukaemia, hodgkin's disease.
4. Alcohol excess.
5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma, synthesis of ferritin by tumour cells.
6. Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can thererfore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive proteins to rule out any inflammatory conditions.

2. Patients with iron deficiency anaemia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.

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

Test Name	Value	Unit	Biological Reference interval

CERULOPLASMIN

CERULOPLASMIN: SERUM *by NEPHELOMETRY*	34.9	mg/dL	22.0 - 61.0

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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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

HEPATITIS C VIRUS (HCV) ANTIBODIES SCREENING

HEPATITIS C ANTIBODY (HCV) TOTAL	NON - REACTIVE		
RESULT			

by IMMUNOCHROMATOGRAPHY

INTERPRETATION:

1.Anti HCV total antibody assay identifies presence IgG antibodies in the serum . It is a useful screening test with a specificity of nearly 99%.
2.It becomes positive approximately 24 weeks after exposure. The test can not isolate an active ongoing HCV infection from an old infection that has been cleared. All positive results must be confirmed for active disease by an HCV PCR test .

FALSE NEGATIVE RESULTS SEEN IN:

1.Window period
2.Immunocompromised states.

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Test Name	Value	Unit	Biological Reference interval

HEPATITIS B SURFACE ANTIGEN (HBsAg) ULTRA

HEPATITIS B SURFACE ANTIGEN (HBsAg): SERUM	0.21	S/CO	NEGATIVE: < 1.0 POSITIVE: > 1.0
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
HEPATITIS B SURFACE ANTIGEN (HBsAg) RESULT	NON REACTIVE		
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			

INTERPRETATION:

RESULT IN INDEX VALUE	REMARKS
< 1.30	NEGATIVE (-ve)
>=1.30	POSITIVE (+ve)

Hepatitis B Virus (HBV) is a member of the Hepadna virus family causing infection of the liver with extremely variable clinical features. Hepatitis B is transmitted primarily by body fluids especially serum and also spread effectively sexually and from mother to baby. In most individuals HBV hepatitis is self limiting, but 1-2 % normal adolescent and adults develop Chronic Hepatitis. Frequency of chronic HBV infection is 5-10% in immunocompromised patients and 80 % neonates. The initial serological marker of acute infection is HBsAg which typically appears 2-3 months after infection and disappears 12-20 weeks after onset of symtoms. Persistence of HBsAg for more than 6 months indicates carrier state or Chronic Liver disease.

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Test Name	Value	Unit	Biological Reference interval

SPECIAL INVESTIGATIONS

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

ANTI NUCLEAR ANTIBODY (ANA) - IFA, HEp2 *by IFA (IMMUNO FLUORESCENT ASSAY)*	Low Positive	NEGATIVE (-ve)
PRIMARY DILUTION *by IFA (IMMUNO FLUORESCENT ASSAY)*	1:100	
PRIMARY INTENSITY (GRADE) ON IF *by IFA (IMMUNO FLUORESCENT ASSAY)*	++	
ANA PATTERN *by IFA (IMMUNO FLUORESCENT ASSAY)*	Cytoplasmic with Nuclear Homogenous	
END POINT TITRES ***by IFA (IMMUNO FLUORESCENT ASSAY)***	**1:320**	

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

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Test Name	Value	Unit	Biological Reference interval
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			
Few	Auto-immune & Viral disease- Primary Biliay Cirrhosis & Chronic Active Hepatitis, Rarely Collagen Vascular disease		
Multiple	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%)		

Follow - Up:- Clinical correlation and/or repeat testing after 6-12 weeks and /or confirmation by ANA profile

*** End Of Report ***

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