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NAME	**: Mrs. GEETA**		
AGE/ GENDER	: 57 YRS/FEMALE	**PATIENT ID**	: 1507092
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012407290016**
REFERRED BY	:	**REGISTRATION DATE**	: 29/Jul/2024 08:37 AM
BARCODE NO.	: 01514038	**COLLECTION DATE**	: 29/Jul/2024 08:45AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 29/Jul/2024 05:47PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

CERULOPLASMIN

CERULOPLASMIN: SERUM *by NEPHELOMETRY*	38.37	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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CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 30/Jul/2024 11:19AM
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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

HEPATITIS B VIRUS CORE ANTIBODY (HBcAb): TOTAL

HEPATITIS B CORE ANTIBODY (HBcAb) TOTAL QUANTITATIVE *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	0.35	U/mL	< 0.85
HEPATITIS B CORE ANTIBODY (HBcAb) TOTAL RESULT *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	NON - REACTIVE		NON - REACTIVE

INTERPRETATION:

NEGATIVE	U/mL	< 0.85
EQUIVOCAL	U/mL	0.85 - 1.15
POSITIVE	U/mL	>1.15

NOTE:
1.Discrepant results may be observed in patients receiving mouse monoclonal antibodies for diagnosis or therapy
2.For heparinized patients, draw specimen prior to heparin therapy as presence of fibrin leads to erroneous results

COMMENTS:
1.Anti- HBc Total is the first antibody to appear usually 4-10 weeks after appearance of HBsAg, at the same time as clinical illness and persists for years or maybe lifetime.
2.It is almost always present during chronic HBV infection. It detects virtually all individuals who have been previously infected with HBV.
3.Detection of Anti HBc Total positive donors reduces incidence of post transmission Hepatitis and possibility of other viral infections like HIV due to frequency of dual infections.
4.This antibody may be seen in 2% of routine donors without any other serological marker and with normal liver enzyme levels. This indicates recovery from subclinical HBV infections.
5.Anti HBc Total is not protective and cannot be used to distinguish Acute from Chronic infection.

USES:
1.As a marker for HBV infection
2.As a screening test for blood donors

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Test Name	Value	Unit	Biological Reference interval

LIVER KIDNEY MICROSOMAL (LKM) - 1 ANTIBODY: ELISA

LIVER KIDNEY MICROSOMAL (LKM) ANTIBODY - ELISA : SERUM *by ELISA (ENZYME LINKED MMUNOSORBENT ASSAY)*	4	IU/mL	NEGATIVE: < 12 BORDERLINE:12.0 - 18.0 POSITIVE: > 18.0

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 spleenomegaly 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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CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 30/Jul/2024 05:41AM
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Test Name	Value	Unit	Biological Reference interval

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA	10.23	IU/mL	NEGATIVE: < 20.0 POSITIVE: > 20.0

by ELISA (ENZYME LINKED IMMUNOASSAY)

INTERPRETATION:

1.Anti-transglutaminase antibodies (ATA) are autoantibodies against the transglutaminase protein.
2.Antibodies to tissue transglutaminas are found in patients with several conditions, including coeliac disease, juvenile diabetes, inflammatory bowel disease, and various forms ofarthritis.
3.In coeliac disease, ATA are involved in the destruction of the villous extracellular matrix and target the destruction of intestinal villous epithelial cells by killer cells.
4.Deposits of anti-tTG in the intestinal epithelium predict coeliac disease.
5.Celiac disease (gluten-sensitive enteropathy, celiac sprue) results from an immune-mediated inflammatory process following ingestion of wheat, rye, or barley proteins that occurs in genetically susceptible individuals. The inflammation in celiac disease occurs primarily in the mucosa of the small intestine, which leads to villous atrophy.

CLINICAL MANIFESTATIONS RELATED TO GASTROINTESTINAL TRACT:

1.Abdominal pain
2.Malabsorption
3.Diarrhea and Constipation.

CLINICAL MANIFESTATION OF CELIAC DISEASE NOT RESTRICTED TO GIT:

1.Failure to grow (delayed puberty and short stature)
2.Iron deficiency anemia
3.Recurrent fetal loss
4.Osteoporosis and chronic fatigue
5.Recurrent aphthous stomatitis (canker sores)
6.Dental enamel hypoplasia, and dermatitis herpetiformis.
7.Patients with celiac disease may also present with neuropsychiatric manifestations including ataxia and peripheral neuropathy, and are at increased risk for development of non-Hodgkin lymphoma.
8.The disease is also associated with other clinical disorders including thyroiditis, type I diabetes mellitus, Down syndrome, and IgA deficiency.

NOTE:

1.The finding of tissue transglutaminase (tTG)-IgA antibodies is specific for celiac disease and possibly for dermatitis herpetiformis. For individuals with moderately to strongly positive results, a diagnosis of celiac disease is likely and the patient should undergo biopsy to confirm the diagnosis.
2.If patients strictly adhere to a gluten-free diet, the unit value of IgA-anti-tTG should begin to decrease within 6 to 12 months of onset of dietary therapy.

CAUTION:

1.This test should not be solely relied upon to establish a diagnosis of celiac disease. It should be used to identify patients who have an increased probability of having celiac disease and in whom a small intestinal biopsy is recommended.
2.Affected individuals who have been on a gluten-free diet prior to testing may have a negative result.

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Test Name	Value	Unit	Biological Reference interval

3.For individuals who test negative, IgA deficiency should be considered. If total IgA is normal and tissue transglutaminase (tTG)-IgA is negative there is a low probability of the patient having celiac disease and a biopsy may not be necessary.
4.If serology is negative or there is substantial clinical doubt remaining, then further investigation should be performed with endoscopy and bowel biopsy. This is especially important in patients with frank malabsorptive symptoms since many syndromes can mimic celiac disease. For the patient with frank malabsorptive symptoms, bowel biopsy should be performed regardless of serologic test results.
5.The antibody pattern in dermatitis herpetiformis may be more variable than in celiac disease; therefore, both endomysial and tTG antibody determinations are recommended to maximize the sensitivity of the serologic tests.

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Test Name	Value	Unit	Biological Reference interval

IMMUNOGLOBIN IgG

IMMUNOGLOBIN-G (IgG): SERUM *by NEPHLOMETRY*	**17.8^{H}**	**gm/L**	**7.0 - 16.0**

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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Test Name	Value	Unit	Biological Reference interval

SMITH (Sm) ANTIBODY IgG

SMITH (Sm) ANTIBODY IgG QUANTITATIVE *by ELISA (ENZYME LINKED IMMUNOASSAY)*	0.34		Negative : [<1.0 Index] Borderline : [1.0-1.2 Index] Positive : [>1.2 Index]
SMITH (Sm) ANTIBODY IgG RESULT *by ELISA (ENZYME LINKED IMMUNOASSAY)*	NEGATIVE (-ve)		NEGATIVE (-ve)

INTERPRETATION:

RESULT IN UNITS (U/mL)	REMARKS
< 12.00	NEGATIVE (-ve)
12.00 – 18.00	BORDERLINE
>18.00	POSITIVE (+ve)

COMMENTS:
Antibodies to Smith antigen are considered a highly specific marker for SLE. They usually occur in association with nuclear Ribonuclear proteins (nRNP). SLE patients with presence of Anti Sm antibodies usually have associated renal disease and / or disorders of central nervous system.

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Test Name	Value	Unit	Biological Reference interval

SPECIAL INVESTIGATIONS

PARIETAL CELL ANTIBODY: IFA

PARIETAL CELL ANTIBODY: SERUM NEGATIVE (-ve)

by IFA (IMMUNOFLUOROSCENCE)

INTERPRETATION:

NOTE:

Autoimmune reactivities are not by themselves diagnostic, but must be correlated with other laboratory & clinical findings

COMMENTS:

Parietal cell antibodies (APCA) are found predominantly in patients with Pernicious anemia (90%) and Autoimmune gastritis Type A (Chronic atrophic gastritis - 50%). These antibodies have also been found in patients with associated organ specific diseases like Insulin Dependent Diabetes mellitus, Hypothyroidism & Addison's disease. 2-15% of healthy individuals may also show these antibodies in low titres.

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Test Name	Value	Unit	Biological Reference interval

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

ANTI NUCLEAR ANTIBODY (ANA) - IFA, HEp2 *by IFA (IMMUNO FLUORESCENT ASSAY)*	NEGATIVE (-ve)		NEGATIVE (-ve)

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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Test Name	Value	Unit	Biological Reference interval
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%)		

*** End Of Report ***

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