Dr. Vinay Chopra
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NAME	**: Mr. ANSHUL SHYAM**		
AGE/ GENDER	: 31 YRS/MALE	**PATIENT ID**	: 1547597
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012407130051**
REFERRED BY	:	**REGISTRATION DATE**	: 13/Jul/2024 11:59 AM
BARCODE NO.	: 01513063	**COLLECTION DATE**	: 13/Jul/2024 12:15PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 13/Jul/2024 01:16PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

SGOT/SGPT PROFILE

SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	40.31	U/L	7.00 - 45.00
SGPT/ALT: SERUM ***by IFCC, WITHOUT PYRIDOXAL PHOSPHATE***	**70.99^{H}**	**U/L**	**0.00 - 49.00**
SGOT/SGPT RATIO *by CALCULATED, SPECTROPHOTOMETRY*	0.57		

INTERPRETATION

NOTE:- **To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.**
USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:-

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)

DECREASED:-

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

PROGNOSTIC SIGNIFICANCE:-

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6

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CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 14/Jul/2024 09:23AM
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Test Name	Value	Unit	Biological Reference interval

CERULOPLASMIN

CERULOPLASMIN: SERUM *by NEPHELOMETRY*	33.22	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

LIVER KIDNEY MICROSOMAL (LKM) - 1 ANTIBODY: ELISA

LIVER KIDNEY MICROSOMAL (LKM) ANTIBODY - ELISA : SERUM *by ELISA (ENZYME LINKED MMUNOSORBENT ASSAY)*	3.4	EU/ml	NEGATIVE: < 25.0 BORDERLINE:20.0 - 25.0 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 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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Test Name	Value	Unit	Biological Reference interval

ANTI NUCLEAR ANTIBODY/FACTOR (ANA/ANF)

ANTI NUCLEUR ANTIBODIES (ANA): SERUM *by ELISA (ENZYME LINKED IMMUNOASSAY)*	0.58	INDEX VALUE	NEGATIVE: < 1.0 BORDERLINE: 1.0 - 1.20 POSITIVE: > 1.20

INTERPRETATION:-

1.For diagnostic purposes, ANA value should be used as an adjuvant to other clinical and laboratory data available.
2.Measurement of antinuclear antibodies (ANAs) in serum is the most commonly performed screening test for patients suspected of having a systemic rheumatic disease, also referred to as connective tissue disease.
3.ANAs occur in patients with a variety of autoimmune diseases, both systemic and organ-specific. They are particularly common in the systemic rheumatic diseases, which include lupus erythematosus (LE), discoid LE, drug-induced LE, mixed connective tissue disease, Sjogren syndrome, scleroderma (systemic sclerosis), CREST (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia) syndrome, polymyositis/dermatomyositis, and rheumatoid arthritis.

NOTE:

1.The diagnosis of a systemic rheumatic disease is based primarily on the presence of compatible clinical signs and symptoms.
The results of tests for autoantibodies including ANA and specific autoantibodies are ancillary. Additional diagnostic criteria include consistent histopathology or specific radiographic findings. Although individual systemic rheumatic diseases are relatively uncommon, a great many patients present with clinical findings that are compatible with a systemic rheumatic disease ANA screening may be useful for ruling out the disease.
2.Secondary, disease specific auto antibodies maybe ordered for patients who are screen positive as ancillary aids for the diagnosis of specific auto-immune disorders.

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Test Name	Value	Unit	Biological Reference interval

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA	10.24	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*	14	g/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)*	0.36	U/mL	Negative : [<1.0 Index]
SMITH (Sm) ANTIBODY IgG RESULT *by ELISA (ENZYME LINKED IMMUNOASSAY)*	NON REACTIVE		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.

*** End Of Report ***

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