P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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NAME	**: Mr. AMANDEEP SINGH**		
AGE/ GENDER	: 35 YRS/MALE	**PATIENT ID**	: 1734074
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122501240025**
REFERRED BY	:	**REGISTRATION DATE**	: 24/Jan/2025 04:45 PM
BARCODE NO.	: 12506672	**COLLECTION DATE**	: 24/Jan/2025 04:55PM
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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

HEPATITIS C VIRUS (HCV) ANTIBODIES SCREENING

HEPATITIS C ANTIBODY (HCV) TOTAL RESULT *by IMMUNOCHROMATOGRAPHY*	NON - REACTIVE		

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

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA *by ELISA (ENZYME LINKED IMMUNOASSAY)*	5.95	IU/mL	NEGATIVE: < 20.0 POSITIVE: > 20.0

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.
3.For individuals who test negative, IgA deficiency should be considered. If total IgA is normal and tissue transglutaminase (tTG)-IgA is negative

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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

HEPATITIS B SURFACE ANTIGEN (HBsAg) SCREENING

HEPATITIS B SURFACE ANTIGEN (HBsAg) RESULT *by IMMUNOCHROMATOGRAPHY*	NON REACTIVE		

INTERPRETATION:-

1.HBsAG is the first serological marker of HBV infection to appear in the blood (approximately 30-60 days after infection and prior to the onset of clinical disease). It is also the last viral protein to disappear from blood and usually disappears by three months after infection in self limiting acute Hepatitis B viral infection.

2.Persistence of HBsAg in blood for more than six months implies chronic infection. It is the most common marker used for diagnosis of an acute Hepatitis B infection but has very limited role in assessing patients suffering from chronic hepatitis.

FALSE NEGATIVE RESULT SEEN IN:

1.Window period.

2.Infection with HBsAg mutant strains

3.Hepatitis B Surface antigen (HBsAg) is the earliest indicator of HBV infection. Usually it appears in 27 - 41 days (as early as 14 days).

4.Appears 7 - 26 days before biochemical abnormalities. Peaks as ALT rises. Persists during the acute illness. Usually disappears 12- 20 weeks after the onset of symptoms / laboratory abnormalities in 90% of cases.

5.Is the most reliable serologic marker of HBV infection. Persistence > 6 months defines carrier state. May also be found in chronic infection.Hepatitis B vaccination does not cause a positive HBsAg. Titers are not of clinical value.

NOTE:-

1.All reactive HBsAG Should be reconfirmed with neutralization test(HBsAg confirmatory test).

2.Anti - HAV IgM appears at the same time as symptoms in > 99% of cases, peaks within the first month, becomes nondetectable in 12 months (usually 6 months). Presence confirms diagnosis of recent acute infection.

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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 reticular/AMA (AC-21)	
END POINT TITRES *by IFA (IMMUNO FLUORESCENT ASSAY)*	1:100 to 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.

*** End Of Report ***

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