



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961 ✉ pkrijainhealthcare@gmail.com

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	: Mrs. SANGEETA	PATIENT ID	: 1742324
AGE/ GENDER	: 48 YRS/FEMALE	REG. NO./LAB NO.	: 122502010019
COLLECTED BY	:	REGISTRATION DATE	: 01/Feb/2025 02:32 PM
REFERRED BY	:	COLLECTION DATE	: 01/Feb/2025 03:29PM
BARCODE NO.	: 12506797	REPORTING DATE	: 01/Feb/2025 04:48PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE		
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) <i>by CALORIMETRIC</i>	10.9 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDEANCE</i>	4.18	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	32.5 ^L	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	77.9 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	26.1 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	33.6	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	15.2	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	44.6	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	18.64	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	28.35	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0


WHITE BLOOD CELLS (WBCS)


TOTAL LEUCOCYTE COUNT (TLC) <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	6810	/cmm	4000 - 11000
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DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	61	%	50 - 70
LYMPHOCYTES	34	%	20 - 40




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<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
EOSINOPHILS	0 ^L	%	1 - 6
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
MONOCYTES	5	%	2 - 12
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
BASOPHILS	0	%	0 - 1
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT	4154	/cmm	2000 - 7500
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
ABSOLUTE LYMPHOCYTE COUNT	2315	/cmm	800 - 4900
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
ABSOLUTE EOSINOPHIL COUNT	0 ^L	/cmm	40 - 440
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
ABSOLUTE MONOCYTE COUNT	340	/cmm	80 - 880
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT)	305000	/cmm	150000 - 450000
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
PLATELETCRIT (PCT)	0.32	%	0.10 - 0.36
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
MEAN PLATELET VOLUME (MPV)	10	fL	6.50 - 12.0
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
PLATELET LARGE CELL COUNT (P-LCC)	94000 ^H	/cmm	30000 - 90000
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
PLATELET LARGE CELL RATIO (P-LCR)	30.8	%	11.0 - 45.0
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
PLATELET DISTRIBUTION WIDTH (PDW)	15.7	%	15.0 - 17.0
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			




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CLINICAL CHEMISTRY/BIOCHEMISTRY

IRON PROFILE

IRON: SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	44.4	µg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	256.12	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM <i>by SPECTROPHOTOMETRY</i>	300.52	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY (FERENE)</i>	14.77^L	%	15.0 - 50.0
TRANSFERRIN: SERUM <i>by SPECTROPHOTOMETRY (FERENE)</i>	213.37	mg/dL	200.0 - 350.0

INTERPRETATION:-

VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:

1. Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia. i.e iron deficiency anemia, zinc deficiency anemia, anemia of chronic disease and thalassemia syndromes.
 2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BINDING CAPACITY (TIBC):

1. It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:

1. Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.



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FERRITIN

FERRITIN: SERUM <i>by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)</i>	10.31	ng/mL	4.63 - 204.0
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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 tarda
4. Ineffective erythropoiesis.

INCREASED FERRITIN WITHOUT IRON OVERLOAD:

1. Liver disorders (NASH) or viral hepatitis (B/C).
2. Inflammatory conditions (Ferritin is an 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 therefore 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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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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ANTI HUMAN IMMUNODEFICIENCY VIRUS (HIV) ANTIBODIES HIV (1 & 2) SCREENING

HIV 1/2 AND P24 ANTIGEN RESULT NON - REACTIVE
by IMMUNOCHROMATOGRAPHY

INTERPRETATION:-

- 1.AIDS is caused by at least 2 known types of HIV viruses, HIV-1 and HIV HIV-2.
- 2.This NACO approved immuno-chromatographic solid phase ELISA assay detects antibodies against both HIV-1 and HIV-2 viruses.
- 3.The test is used for routine serologic screening of patients at risk for HIV-1 or HIV-2 infection.
- 4.All screening ELISA assays for HIV antibody detection have high sensitivity but have low specificity.
- 5.At this laboratory, all positive samples are cross checked for positivity with two alternate assays prior to reporting.

NOTE:-

- 1.Confirmatory testing by Western blot is recommended for patients who are reactive for HIV by this assay.
- 2.Antibodies against HIV-1 and HIV-2 are usually not detectable until 6 to 12 weeks following exposure (window period) and are almost always detectable by 12 months.
- 3.The test is not recommended for children born to HIV infected mothers till the child turns two years old (as HIV antibodies may be transmitted passively to the child trans-placentally).

FALSE NEGATIVE RESULT SEEN IN:

- 1.Window period
- 2.Severe immuno-suppression including advanced AIDS.



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ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA <i>by ELISA (ENZYME LINKED IMMUNOASSAY)</i>	8.29	IU/mL	NEGATIVE: < 20.0 POSITIVE: > 20.0
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INTERPRETATION:

1. Anti-transglutaminase antibodies (ATA) are autoantibodies against the transglutaminase protein.
2. Antibodies to tissue transglutaminase are found in patients with several conditions, including coeliac disease, juvenile diabetes, inflammatory bowel disease, and various forms of arthritis.
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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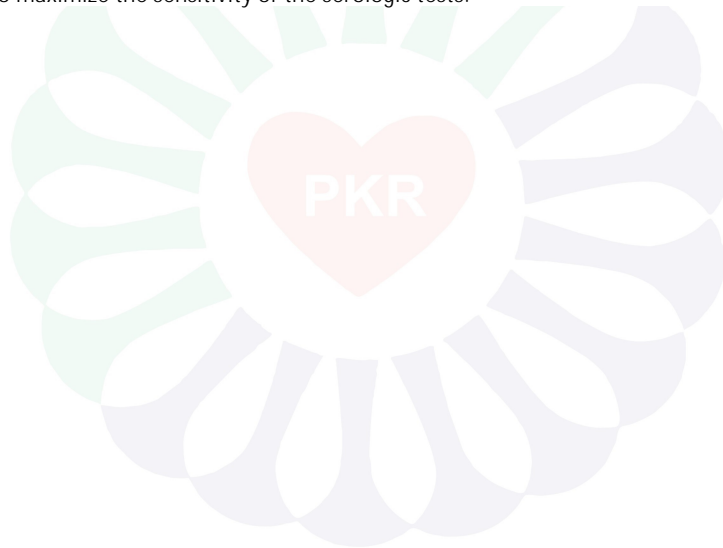
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there is a low probability of the patient having celiac disease and a biopsy may not be necessary.

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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HEPATITIS B SURFACE ANTIGEN (HBsAg) SCREENING

HEPATITIS B SURFACE ANTIGEN (HBsAg) NON - REACTIVE

RESULT

by IMMUNOCHROMATOGRAPHY

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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BARCODE NO.	: 12506797	REPORTING DATE	: 03/Feb/2025 03:37PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE		
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
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MICROBIOLOGY

CULTURE AEROBIC BACTERIA AND ANTIBIOTIC SENSITIVITY: URINE

CULTURE AND SUSCEPTIBILITY: URINE

DATE OF SAMPLE	01-02-2025
SPECIMEN SOURCE	URINE
INCUBATION PERIOD	48 HOURS
by AUTOMATED BROTH CULTURE	
CULTURE	STERILE
by AUTOMATED BROTH CULTURE	
ORGANISM	NO AEROBIC PYOGENIC ORGANISM GROWN AFTER 48 HOURS OF INCUBATION AT 37°C
by AUTOMATED BROTH CULTURE	

AEROBIC SUSCEPTIBILITY: URINE

INTERPRETATION:

1. In urine culture and sensitivity, presence of more than 100,000 organism per mL in midstream sample of urine is considered clinically significant. However in symptomatic patients , a smaller number of bacteria (100 to 10000/mL) may signify infection.
2. Colony count of 100 to 10000/ mL indicate infection, if isolate from specimen obtained by suprapubic aspiration or "in-and-out" catheterization or from patients with indwelling catheters.

SUSCEPTIBILITY:

1. A test interpreted as **SENSITIVE** implies that infection due to isolate may be appropriately treated with the dosage of an antimicrobial agent recommended for that type of infection and infecting species, unless otherwise indicated..
2. A test interpreted as **INTERMEDIATE** implies that the "infection due to the isolate may be appropriately treated in body sites where the drugs are physiologically concentrated or when a high dosage of drug can be used".
3. A test interpreted as **RESISTANT** implies that the "isolates are not inhibited by the usually achievable concentration of the agents with normal dosage, schedule and/or fall in the range where specific microbial resistance mechanism are likely (e.g. beta-lactamases), and clinical efficacy has not been reliable in treatment studies.

CAUTION:

Conditions which can cause a false Negative culture:

1. Patient is on antibiotics. Please repeat culture post therapy.
2. Anaerobic bacterial infection.
3. Fastidious aerobic bacteria which are not able to grow on routine culture media.
4. Besides all these factors, at least in 25-40 % of cases there is no direct correlation between in vivo clinical picture.
5. Renal tuberculosis to be confirmed by AFB studies.

*** End Of Report ***



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