



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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

NAME : Miss. ANJALI
AGE/ GENDER : 26 YRS/FEMALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12506994
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1755337
REG. NO./LAB NO. : 122502130012
REGISTRATION DATE : 13/Feb/2025 10:49 AM
COLLECTION DATE : 13/Feb/2025 11:34AM
REPORTING DATE : 13/Feb/2025 02:35PM

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>	7.3 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	4	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	25.3 ^L	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	63.4 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	18.2 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	28.7 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	21.4 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	50.6	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	15.85	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	33.83	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>	3490 ^L	/cmm	4000 - 11000
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DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	42 ^L	%	50 - 70
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LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	52 ^H	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	1	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	5	%	2 - 12
BASOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	%	0 - 1
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	1466 ^L	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	1815 ^L	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	35 ^L	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	174	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	/cmm	0 - 110
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	386000	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.33	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	9	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	75000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	19.3	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	15.2	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD




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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA
by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)

82.35

mg/dL


NORMAL: < 100.0
PREDIABETIC: 100.0 - 125.0
DIABETIC: > OR = 126.0


INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.




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LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.45	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.23	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.22	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	20.59	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	14.41	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.43	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	52.04	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	12.68	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.46	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.31	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.15 ^L	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2	RATIO	1.00 - 2.00

INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Reference 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 CHOLESTASIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)




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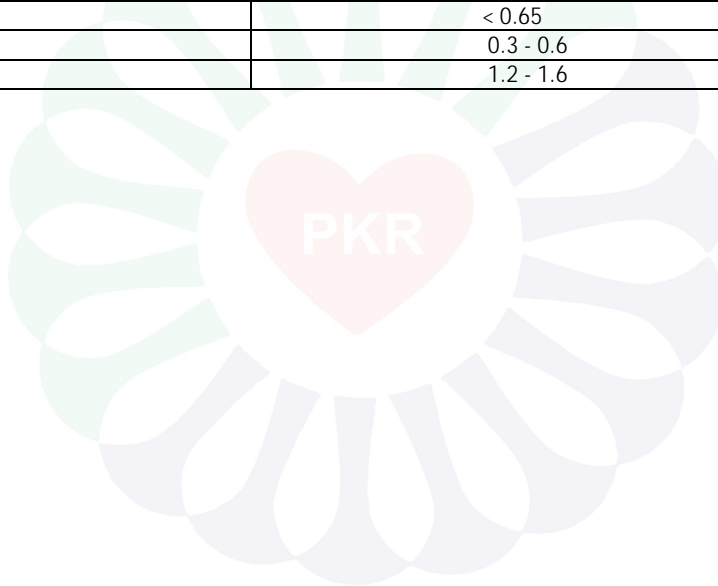
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DECREASED:


1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestasis: 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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ENDOCRINOLOGY

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 1.73 μ IU/mL 0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

AGE	REFERENCE RANGE (μ IU/mL)
0 – 5 DAYS	0.70 – 15.20
6 Days – 2 Months	0.70 – 11.00
3 – 11 Months	0.70 – 8.40
1 – 5 Years	0.70 – 7.00
6 – 10 Years	0.60 – 5.50
11 - 15	0.50 – 5.50
> 20 Years (Adults)	0.27 – 5.50
PREGNANCY	
1st Trimester	0.10 - 3.00
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

NOTE:- TSH levels are subjected to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50 %. Hence time of the day has influence on the measured serum TSH concentration.

USE:- TSH controls biosynthesis and release of thyroid hormones T4 & T3. It is a sensitive measure of thyroid function, especially useful in early or subclinical hypothyroidism, before the patient develops any clinical findings or goitre or any other thyroid function abnormality.

INCREASED LEVELS:

- 1.Primary or untreated hypothyroidism, may vary from 3 times to more than 100 times normal depending on degree of hypofunction.
- 2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
- 3.Hashimotos thyroiditis.
- 4.DRUGS: Amphetamines, Iodine containing agents and dopamine antagonist.
- 5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge.

DECREASED LEVELS:

- 1.Toxic multi-nodular goitre & Thyroiditis.
- 2.Over replacement of thyroid hormone in treatment of hypothyroidism.
- 3.Autonomously functioning Thyroid adenoma
- 4.Secondary pituitary or hypothalamic hypothyroidism
- 5.Acute psychiatric illness
- 6.Severe dehydration.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.



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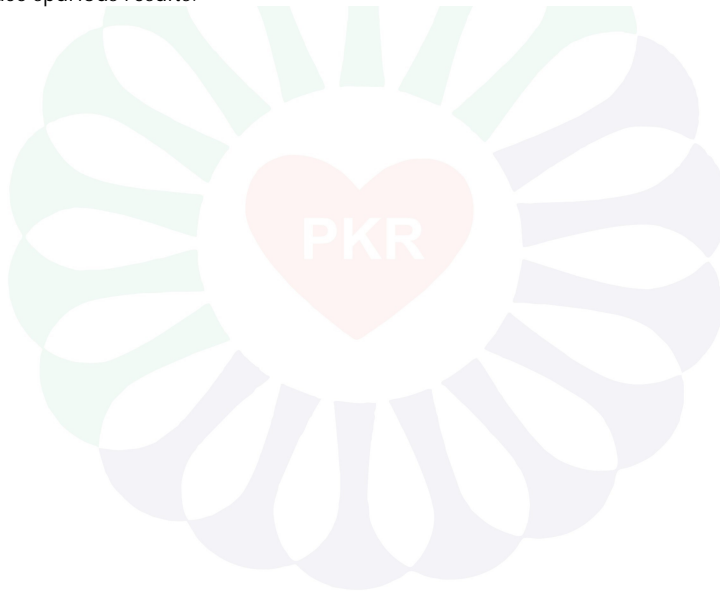
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8.Pregnancy: 1st and 2nd Trimester

LIMITATIONS:

- 1.TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.
- 2.Autoimmune disorders may produce spurious results.




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IMMUNOPATHOLOGY/SEROLOGY

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA by ELISA (ENZYME LINKED IMMUNOASSAY)	6.559	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.



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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, 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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C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: SERUM	0.49	mg/L	0.0 - 6.0
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by NEPHLOMETRY

INTERPRETATION:


1. C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.
2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant rejection, and to monitor these inflammatory processes.
4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,
5. Elevated values are consistent with an acute inflammatory process.

NOTE:

1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
2. Oral contraceptives may increase CRP levels.




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P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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NAME : Miss. ANJALI
AGE/ GENDER : 26 YRS/FEMALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12506994
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1755337
REG. NO./LAB NO. : 122502130012
REGISTRATION DATE : 13/Feb/2025 10:49 AM
COLLECTION DATE : 13/Feb/2025 11:34AM
REPORTING DATE : 13/Feb/2025 06:47PM

Test Name	Value	Unit	Biological Reference interval
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IMMUNOGLOBIN IgA

IMMUNOGLOBIN-A (IgA): SERUM
by NEPHLOMETRY


276.6	mg/dL	20.0 - 300.0
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INTERPRETATION:

1. Approximately 10 to 15% of total plasma immunoglobulins account for IgA. It contains 10% of carbohydrate and has mol. wt. 160,000 with half life of 6 days.
2. It serves to protect the skin and mucosa against microorganisms. It is capable of binding toxins and in combination with lysozyme develop antibacterial and antiviral activity.
3. IgA is the predominant immunoglobulin in the body secretion such as colostrum, saliva, and sweat. Secretory IgA provides defense against local infection and is important in binding food antigens in the gut.
4. Increased polyclonal IgA may occur in chronic liver diseases, autoimmune disorders (SLE, Rheumatoid arthritis) and sarcoidosis. Monoclonal IgA increases in IgA myeloma.
5. Decreased synthesis of IgA is observed in acquired and congenital immunodeficiency diseases. Reduced levels of IgA can be caused by protein losing gastroenteropathies and loss through skin from burns.

*** End Of Report ***




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