P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

0171-2532620, 8222896961 pkrjainhealthcare@gmail.com

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	**: Miss. MANSI**		
AGE/ GENDER	: 18 YRS/FEMALE	**PATIENT ID**	: 1554957
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122407200004**
REFERRED BY	:	**REGISTRATION DATE**	: 20/Jul/2024 08:33 AM
BARCODE NO.	: 12503694	**COLLECTION DATE**	: 20/Jul/2024 09:15AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 20/Jul/2024 12:21PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

HAEMATOLOGY

COMPLETE BLOOD COUNT (CBC)

Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) *by CALORIMETRIC*	**11.9[L]**	**gm/dL**	**12.0 - 16.0**
RED BLOOD CELL (RBC) COUNT *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	4.54	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**36.3[L]**	**%**	**37.0 - 50.0**
MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**79.8[L]**	**fL**	**80.0 - 100.0**
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**26.2[L]**	**pg**	**27.0 - 34.0**
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	32.8	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	15.4	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	47.2	fL	35.0 - 56.0
MENTZERS INDEX *by CALCULATED*	17.58	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX *by CALCULATED*	27.06	RATIO	BETA THALASSEMIA TRAIT: < = 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	6940	/cmm	4000 - 11000
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	56	%	50 - 70
LYMPHOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	34	%	20 - 40
EOSINOPHILS	1	%	1 - 6

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CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

NOT VALID FOR MEDICO LEGAL PURPOSE

440 Dated 17.5.2012 u/s 80 G OF INCOME TAX ACT. PAN NO. AAAAP1600.
REPORT ATTRACTS THE CONDITIONS PRINTED OVERLEAF (P.T.O.)

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Test Name	Value	Unit	Biological Reference interval
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
MONOCYTES	9	%	2 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	3886	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	**2360^{L}**	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE EOSINOPHIL COUNT	69	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE MONOCYTE COUNT	625	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.			
PLATELET COUNT (PLT)	265000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT)	0.29	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	11	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL COUNT (P-LCC)	**91000^{H}**	**/cmm**	**30000 - 90000**
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL RATIO (P-LCR)	34.5	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET DISTRIBUTION WIDTH (PDW)	16.2	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIVER FUNCTION TEST (COMPLETE)

Test Name	Value	Unit	Biological Reference interval
BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	1.01	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.34	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.67	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	21.45	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	17.83	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.2	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	72.51	U/L	50.00 - 370.00
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM *by SZASZ, SPECTROPHTOMETRY*	13.92	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	7.4	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	4.59	gm/dL	3.50 - 5.50
GLOBULIN: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	2.81	gm/dL	2.30 - 3.50
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.63	RATIO	1.00 - 2.00

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

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Test Name	Value	Unit	Biological Reference interval
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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Test Name	Value	Unit	Biological Reference interval
IRON PROFILE			
IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	70.13	µg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM *by FERROZINE, SPECTROPHOTOMETERY*	312.4	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM *by SPECTROPHOTOMETERY*	382.53	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM *by CALCULATED, SPECTROPHOTOMETERY (FERENE)*	18.33	%	15.0 - 50.0
TRANSFERRIN: SERUM *by SPECTROPHOTOMETERY (FERENE)*	271.6	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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Test Name	Value	Unit	Biological Reference interval

FERRITIN

FERRITIN: SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**9.2[L]**	**ng/mL**	**10.0 - 290.0**

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 tada
4. Ineffective erythropoiesis.

INCREASED FERRITIN WITHOUT IRON OVERLOAD:

1. Liver disorders (NASH) or viral hepatitis (B/C).
2. Inflammatory conditions (Ferritin is a 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 thererfore 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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Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM	3.938	µIU/mL	0.50 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

AGE	REFFERENCE RANGE (µIU/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 circardian 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 harmones 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 harmone in treatment of hypothyroidism.
3.Autonomously functioning Thyroid adenoma
4.Secondary pituatary or hypothalmic hypothyroidism
5.Acute psychiatric illness
6.Severe dehydration.

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Test Name	Value	Unit	Biological Reference interval

7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
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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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA	9.52	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

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Test Name	Value	Unit	Biological Reference interval

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 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 IgA			
IMMUNOGLOBIN-A (IgA): SERUM *by NEPHLOMETRY*	2.1	g/L	0.70 - 4.00 g/L

INTERPRETATION:

1.Approximately 10 to 15% of total plasma immunoglobulings account for IgA. It contains 10% of carbohdrate 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, salivai, and sweet. Secretary 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 ***

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)