

Dr. Vinay Chopra
MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

Dr. Yugam Chopra
MD (Pathology)
CEO & Consultant Pathologist

NAME : Mr. RISHABH
AGE/ GENDER : 23 YRS/MALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 01520427
CLIENT CODE. : KOS DIAGNOSTIC LAB
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1666328
REG. NO./LAB NO. : 012411090038
REGISTRATION DATE : 09/Nov/2024 12:03 PM
COLLECTION DATE : 09/Nov/2024 12:09PM
REPORTING DATE : 09/Nov/2024 04:28PM

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>	13.9	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	5.07 ^H	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	44.3	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	87.4	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	27.5	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	31.5 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	15.4	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	50.2	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	17.24	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	26.63	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>	9580	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) <i>by AUTOMATED 6 PART HEMATOLOGY ANALYZER</i>	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	NIL	%	< 10 %



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<u>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</u>			
NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	67	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	21	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	5	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	7	%	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>	6419	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	2012	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	479 ^H	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	671	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRANULOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	/cmm	0.0 - 999.0
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	113000 ^L	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.19	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	17 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	83000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	75.3 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	15.4	%	15.0 - 17.0




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NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED.




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PERIPHERAL BLOOD SMEAR

TEST NAME:

PERIPHERAL BLOOD FILM/SMEAR (PBF)

RED BLOOD CELLS (RBC'S):

RBCs mostly appear normocytic & normochromic.No polychromatic cells or normoblasts present.

WHITE BLOOD CELLS (WBC'S):

No immature leucocytes seen.

PLATELETS:

Platelets appear slightly reduced on smear.

HEMOPARASITES:

NOT SEEN.

IMPRESSION:

Normocytic normochromic picture.




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BARCODE NO.	: 01520427	REPORTING DATE	: 09/Nov/2024 05:46PM
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IMMUNOPATHOLOGY/SEROLOGY

HELICOBACTER PYLORI ANTIGEN DETECTION - STOOL

HELICOBACTER ANTIGEN DETECTION - STOOL by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	0.16	INDEX	NEGATIVE: <0.90 EQUIVOCAL: 0.90-1.10 POSITIVE: >=1.10
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INTERPRETATION:

CLINICAL BACKGROUND:

H pylori infection is associated with peptic ulcer disease (duodenal and gastric) and chronic active gastritis. H pylori infection is also an independent risk factor for gastric cancer and primary malignant lymphoma of the stomach. However, many people who are infected with H. pylori may not show any symptoms of the disease.

NOTE:

1. It is a chemiluminescent Immunoassay (CLIA) for detection of Helicobacter pylori antigen in faecal samples and can be used for diagnosis, therapeutic monitoring and to assess eradication of H. pylori infection post treatment.
2. It is a qualitative test.
3. A positive result (antigen detected) is indicative of H pylori presence in stool sample.
4. A negative result does not exclude the possibility of Helicobacter pylori infection.
5. Assay results should be utilized in conjunction with other clinical and laboratory data to assist the clinician in making individual patient management decisions.
6. Antimicrobials, proton pump inhibitors and bismuth preparations are known to suppress H.pylori and if ingested may give a false negative result.
7. Fecal specimens preserved in 10 % formalin, merthiolate formalin, sodium acetate formalin, or polyvinyl alcohol or specimens that are in transport media such as Cary Blair or C & S cannot be used.




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ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA by ELISA (ENZYME LINKED IMMUNOASSAY)	10.56	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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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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BARCODE NO.	: 01520427	REPORTING DATE	: 09/Nov/2024 01:48PM
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Test Name	Value	Unit	Biological Reference interval
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VITAMINS

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM **89^L** pg/mL 190.0 - 890.0
 by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:-

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol lgestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
 2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
 3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
 4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
 5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
 6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
 7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.
NOTE:A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.




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Test Name	Value	Unit	Biological Reference interval
VITAMIN B9/FOLIC ACID/FOLATE			
VITAMIN B9/FOLIC ACID/FOLATE: SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	3.6	ng/mL	DEFICIENT: < 3.37 INTERMEDIATE: 3.37 - 5.38 NORMAL: > 5.38

INTERPRETATION

RESULT IN ng/mL	REMARKS
0.35 – 3.37	DEFICIENT
3.38 – 5.38	INTERMEDIATE
5.39 – 100.00	NORMAL

NOTE:

1. Drugs like Methotrexate & Leucovorin interfere with folate measurement
2. To differentiate vitamin B12 & folate deficiency, measurement of Methyl malonic acid in urine & serum Homocysteine level is suggested
3. Risk of toxicity from folic acid is low as it is a water soluble vitamin regularly excreted in urine

COMMENTS:

1. Folate plays an important role in the synthesis of purine & pyrimidines in the body and is important for the maturation of erythrocytes.
2. It is widely available from plants and to a lesser extent organ meats, but more than half the folate content of food is lost during cooking.
3. Folate deficiency is commonly prevalent in alcoholic liver disease, pregnancy and the elderly. It may result from poor intestinal absorption, nutrition deficiency, excessive demand as in pregnancy or in malignancy and in response to certain drugs like Methotrexate & anticonvulsants.
4. Decreased Levels Megaloblastic anemia, Infantile hyperthyroidism, Alcoholism, Malnutrition, Scurvy, Liver disease, B12 deficiency, dietary amino acid excess, adult Celiac disease, Tropical Sprue, Crohn's disease, Hemolytic anemias, Carcinomas, Myelofibrosis, vitamin B6 deficiency, pregnancy, Whipple's disease, extensive intestinal resection and severe exfoliative dermatitis

*** End Of Report ***




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