CLIENT CODE.



KOS Diagnostic Lab

(A Unit of KOS Healthcare)



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

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

: 31/Aug/2024 10:43AM

NAME : Mr. KUMAR GAURAV

AGE/ GENDER : 44 YRS/MALE **PATIENT ID** : 1597175

COLLECTED BY : 012408310022 REG. NO./LAB NO.

REFERRED BY : ROTARY HOSPITAL (AMBALA CANTT) **REGISTRATION DATE** : 31/Aug/2024 09:56 AM BARCODE NO. :01516018 **COLLECTION DATE** : 31/Aug/2024 10:01AM

: KOS DIAGNOSTIC LAB **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit **Biological Reference interval**

HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

REPORTING DATE

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	12.6	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.76	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	39.8 ^L	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	83.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	26.4 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by calculated by automated hematology analyzer	31.6 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by calculated by automated hematology analyzer	14	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	43.8	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	17.56	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	24.52	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	7460	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by automated 6 part hematology analyzer	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by calculated by automated hematology analyzer DIFFERENTIAL LEUCOCYTE COUNT (DLC)	NIL	%	< 10 %
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	70	%	50 - 70



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Test Name	Value	Unit	Biological Reference interval
_YMPHOCYTES	21	%	20 - 40
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	21	/0	20 - 40
EOSINOPHILS	2	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
MONOCYTES	7	%	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	5222	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	/		
ABSOLUTE LYMPHOCYTE COUNT	1567	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE FOSINOPHIL COUNT	140	lomm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	149	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT	522	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	022	7 6111111	00 000
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
PLATELETS AND OTHER PLATELET PREDICTIVE MARKER	<u>RS.</u>		
PLATELET COUNT (PLT)	166000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT)	0.2	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	16 ^H	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC)	оооооН	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	83000 ^H	/CITIIII	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR)	64.5 ^H	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	04.0	,,	
PLATELET DISTRIBUTION WIDTH (PDW)	16.6	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			



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Test Name Value Unit Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.52	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.16	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.36	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	31.6	U/L	7.00 - 45.00
SGPT/ALT: SERUM	64.3 ^H	U/L	0.00 - 49.00
by IFCC, WITHOUT PYRIDOXAL PHOSPHATE AST/ALT RATIO: SERUM	0.49	RATIO	0.00 - 46.00
by CALCULATED, SPECTROPHOTOMETRY			
ALKALINE PHOSPHATASE: SERUM by Para nitrophenyl phosphatase by amino methyl propanol	79.58	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	16.17	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM	6.57	gm/dL	6.20 - 8.00
by BIURET, SPECTROPHOTOMETRY ALBUMIN: SERUM	3.62	gm/dL	3.50 - 5.50
by BROMOCRESOL GREEN	3.02	giii/uL	3.30 - 3.30
GLOBULIN: SERUM	2.95	gm/dL	2.30 - 3.50
by CALCULATED, SPECTROPHOTOMETRY		· ·	
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.23	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)	
DEODEACED	•		<u> </u>

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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age 4 of 12



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Test Name Value Unit Bi	
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	IRON PRO	FILE	
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	42.4 ^L	μg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY	218.98	μg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETERY	261.38	μg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	16.22	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)	185.58 ^L	mg/dL	200.0 - 350.0

INITEDDDETATION:

<u>IINTERPRETATION:-</u>			
VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	OTAL IRON BINDING CAPACITY: Decreased		Normal
% TRANSFERRIN SATURATION: Decreased		Decreased < 12-15 %	Normal
SERLIM 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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ENDOCRINOLOGY

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 4.153 μIU/mL 0.35 - 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		
PRE	GNANCY		
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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7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

8. Pregnancy: 1st and 2nd Trimester

LIMITATIONS:

CLIENT CODE.

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

IU/mL ANTI TISSUE TRANSGLUTAMINASE 11.2 NEGATIVE: < 20.0 ANTIBODY IgA POSITIVE: > 20.0

by ELISA (ENZYME LINKED IMMUNOASSAY)

- 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 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
- 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

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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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**

VITAMINS

REPORTING DATE

VITAMIN B12/COBALAMIN

252 VITAMIN B12/COBALAMIN: SERUM pg/mL 190.0 - 890.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:-

CLIENT CODE.

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 Igestion
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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NAME : Mr. KUMAR GAURAV

AGE/ GENDER : 44 YRS/MALE **PATIENT ID** : 1597175

COLLECTED BY : REG. NO./LAB NO. : 012408310022

REFERRED BY : ROTARY HOSPITAL (AMBALA CANTT) REGISTRATION DATE : 31/Aug/2024 09:56 AM

BARCODE NO. : 01516018 COLLECTION DATE : 31/Aug/2024 10:01AM

CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 31/Aug/2024 11:49AM

CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

CLINICAL PATHOLOGY FECAL CALPROTECTIN

FECAL CALPROTECTIN 10.4 μg/g < 50.0

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION

RESULT IN mg/kg FECES	REMARKS
< 25.0	NEGATIVE
25.0 – 50.0	BORDERLINE
>50.0	POSITIVE

NOTE:

1.To avoid potential false positive results, patients should abstain from using NSAIDs for at least two weeks prior to the test 2.It is recommended to repeat all borderline results if clinically indicated Comments Calprotectin is a calcium-binding protein found within neutrophils which influx into the bowel during inflammation.

Calprotectin is excreted in excess into the intestinal lumen during the inflammatory process and act as a marker for inflammatory diseases of the lower gastrointestinal tract. The levels of the protein are high in cases of Inflammatory bowel diseases (IBD) but not in non-inflammatory bowel diseases e.g. Irritable bowel syndrome (IBS), therefore this test can help to differentiate between the two diseases.

I ISES.

- 1.To differentiate between IBS and IBD
- 2.To monitor the effectiveness of IBD therapy
- 3.To detect IBD relapse



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





(A Unit of KOS Healthcare)



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

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

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STOOL ROUTINE AND MICROSCOPIC EXAMINATION

REPORTING DATE

PHYSICAL EXAMINATION

CLIENT CODE.

COLOUR / APPEARANCE BROWNISH YELLOWISH BROWN CONSISTENCY **SOFT** SEMI- FORMED/FORMED

PUS **ABSENT ABSENT MUCOUS PRESENT ABSENT BLOOD NEGATIVE (-ve)**

NEGATIVE (-ve) **NOT SEEN PARASITES NOT SEEN**

MICROSCOPIC EXAMINATION

NEGATIVE (-ve) 0 - 5 **PUS CELLS** /HPF by MICROSCOPY

RED BLOOD CELLS (RBCs) NEGATIVE (-ve) /HPF 0 - 3 by MICROSCOPY

NOT SEEN NOT SEEN

by MICROSCOPY **CYSTS NOT SEEN NOT SEEN**

by MICROSCOPY NO DARTING MOTILITY SEEN

STOOL FOR VIBRIO CHOLERA by MICROSCOPY

STOOL FOR FAT GLOBULES **NOT SEEN NOT SEEN**

End Of Report ***



by MICROSCOPY

CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

