

(A Unit of KOS Healthcare)



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

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

**NAME** : Mr. KUDRAT VERMA

**PATIENT ID AGE/ GENDER** : 16 YRS/MALE : 1641172

**COLLECTED BY** : SURJESH REG. NO./LAB NO. :012410110050

REFERRED BY : CENTRAL PHOENIX CLUB (AMBALA CANTT) **REGISTRATION DATE** : 11/Oct/2024 07:09 PM

BARCODE NO. :01518721 **COLLECTION DATE** : 12/Oct/2024 02:26AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 11/Oct/2024 07:34PM

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

**Test Name** Value Unit **Biological Reference interval** 

## **HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)**

### **RED BLOOD CELLS (RBCS) COUNT AND INDICES**

HAEMOGLOBIN (HB) by CALORIMETRIC	11.9 <sup>L</sup>	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	5.36 <sup>H</sup>	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV)	37.9	%	35.0 - 49.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER MEAN CORPUSCULAR VOLUME (MCV)	70.7 <sup>L</sup>	fL	80.0 - 100.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	22.2 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	31.4 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	16	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD)  by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	42.2	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	13.19	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	21.1	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	10800	/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	NIL	%	< 10 %
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by flow cytometry by Sf cube & microscopy	64	%	50 - 70



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LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	27	%	20 - 40
EOSINOPHILS  by Flow cytometry by SF cube & microscopy	4	%	1 - 6
MONOCYTES  by Flow cytometry by SF cube & microscopy	5	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY  ABSOLUTE LEUKOCYTES (WBC) COUNT	0	%	0 - 1
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6912	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT  by Flow cytometry by SF cube & microscopy	2916	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by Flow cytometry by Sf cube & microscopy	432	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY  PLATELETS AND OTHER PLATELET PREDICTIVE MARKE	540 <b>RS.</b>	/cmm	80 - 880
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	411000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.32	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	8	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	55000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	13.4	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	15.8	%	15.0 - 17.0



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Test Name Value Unit Biological Reference interval

# CLINICAL CHEMISTRY/BIOCHEMISTRY LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.21	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.06	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.15	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	15.5	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	9.8	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.58	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para nitrophenyl phosphatase by amino methyl propanol	67.01	U/L	50.00 - 370.00
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	25.27	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.01	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.2	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	1.49	RATIO	1.00 - 2.00

#### <u>INTERPRETATION</u>

*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



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Test Name	Value	Unit	Biological Reference interval
INTRAHEPATIC CHOLESTATIS		> 1.5	
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS		> 1.3 (Slightly Increased)	
DEODEAGED			

#### 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:

NO CITO OTO CICILII TO MICE.			
NORMAL	< 0.65		
GOOD PROGNOSTIC SIGN	0.3 - 0.6		
POOR PROGNOSTIC SIGN	1.2 - 1.6		



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: 12/Oct/2024 10:46AM

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## IMMUNOPATHOLOGY/SEROLOGY ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

REPORTING DATE

IU/mL ANTI TISSUE TRANSGLUTAMINASE 11.21 NEGATIVE: < 20.0 ANTIBODY IgA POSITIVE: > 20.0

by ELISA (ENZYME LINKED IMMUNOASSAY)

CLIENT CODE.

- 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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### TYPHOID COMBO SCREEN (TYPHOID ANTIGEN, IgG AND IgM): SERUM

TYPHOID ANTIGEN - SERUM NEGATIVE (-ve) NEGATIVE (-ve)

by ICT (IMMUNOCHROMATOGRAPHY)

TYPHI DOT ANTIBODY IgG POSITIVE (+ve) NEGATIVE (-ve) by ICT (IMMUNOCHROMATOGRAPHY)

TYPHI DOT ANTIBODY IgM WEAK POSITIVE (+ve) NEGATIVE (-ve)

by ICT (IMMUNOCHROMATOGRAPHY)

#### INTERPRETATION:

Typhoid fever is a life threatening illness caused by the bacterium Salmonella typhus. The infection is acquired typically by ingestion. On reaching the gut, the bacilli attach themselves to the epithelial cells of the intestinal villi and penetrate the lamina and submucosa. They are then phagocytosed there by polymorphs and mesenteric lymph nodes, where they multiply and, via the thoracic duct, enter the blood stream. A transient bacteremia follows, during which the bacilli are seeded in the liver, gall bladder, spleen, bone marrow, lymph nodes, and kidneys, where further multiplication takes place. Towards the end of the incubation period, there occurs a massive bacteremia from these sites, heralding the onset of the clinical symptoms.

The diagnosis of typhoid consists of isolation of the bacilli and the demonstration of antibodies. The isolation of the bacilli is very time consuming and antibody detection is not very specific. Other tests include the Widal reaction. The advantage of this test is that it takes only 10-20 minutes and requires only a small amount of stool/serum/plasma to perform. It is the easiest and most specific method for detecting S. typhi infection.

RELATIVE SENSTIVITY OF TYPHOID ANTIGEN DETECTION: 98.7% RELATIVE SPECIFICITY OF TYPHOID ANTIGEN DETECTION: 97.4%

#### **DETECTABLE IGM RESPONSE:**

ONSET OF FEVER	PERCENT POSITIVE
4 - 6 DAYS	43.5
6 - 9 DAYS	92.9
> 9 DAYS	99.5

1.This is a solid phase, immunochromatographic ELISA assay that detects specific IgM and IgG Antibodies against the OUTER MEMBRAN PROTEIN(OMP) of the Salmonella species. IgM antibodies appear in the serum 2-3 days post infection and are indicative of a recent infection while the IgG antibodies appear later and are useful for presumptive diagnosis of Enteric fever if the patient presents more than a week after onset of symptoms.

2. This is a useful screening assay for the early detection of Enteric fever and has a high sensitivity. However the test has moderate specificity and false positive results may be obtained in the following situations:

- · Antibodies against Salmonella may cross react with other antibodies.
- Unrelated infections may lead to production of specific Salmonella antibodies if the patient has previously been exposed to Salmonella infection (ANAMNESTIC RESPONSE).



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NOTE:-Rapid blood culture performed during ft week of infection is highly recommended for confirmation of all IgM positive results. In case the patient has presented after the first week of infection, a thorough clinical correlation and confirmatory Widal test must be performed to establish the diagnosis.



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### **C-REACTIVE PROTEIN (CRP)**

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: 23.81<sup>H</sup> mg/L 0.0 - 6.0

**SERUM** 

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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### MICROBIOLOGY

### CULTURE AEROBIC BACTERIA AND ANTIBIOTIC SENSITIVITY (CONVENTIONAL): BLOOD

### **BLOOD CULTURE AND SUSCEPTIBILITY**

DATE OF SAMPLE 12-10-2024 SPECIMEN SOURCE **BLOOD** 

72 HOURS (3 SUBCULTURES) INCUBATION PERIOD

**CULTURE STERILE** 

by AUTOMATED BROTH CULTURE

NO AEROBIC PYOGENIC ORGANISM GROWN AFTER 72 HOURS OF INCUBATION AT ORGANISM

37\*C

### by AUTOMATED BROTH CULTURE **AEROBIC SUSCEPTIBILITY BLOOD**

- 1. A test interpreted as SENSTITIVE 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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