

Dr. Vinay Chopra
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 Chairman & Consultant Pathologist

Dr. Yugam Chopra
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NAME	: Master. KAVIAR SOOD	PATIENT ID	: 1810259
AGE/ GENDER	: 7 YRS/MALE	REG. NO./LAB NO.	: 012503280044
COLLECTED BY	: SURJESH	REGISTRATION DATE	: 28/Mar/2025 06:26 PM
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	COLLECTION DATE	: 28/Mar/2025 06:26PM
BARCODE NO.	: 01527943	REPORTING DATE	: 28/Mar/2025 06:43PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

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>	11.5 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	4.45	Millions/cmm	3.50 - 5.50
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	34.5 ^L	%	35.0 - 49.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	77.5 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	25.8 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	33.3	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	14.1	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	41.2	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	17.42	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	73.64	RATIO	BETA THALASSEMIA TRAIT: <= 74.1 IRON DEFICIENCY ANEMIA: >= 74.1

WHITE BLOOD CELLS (WBCS)

TOTAL LEUCOCYTE COUNT (TLC) <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	5520	/cmm	5000 - 15000
NUCLEATED RED BLOOD CELLS (nRBCS) <i>by AUTOMATED 6 PART HEMATOLOGY ANALYZER</i>	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) %	NIL	%	< 10 %




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by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER			
<u>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</u>			
NEUTROPHILS	64	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
LYMPHOCYTES	28	%	20 - 45
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
EOSINOPHILS	0L	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
MONOCYTES	8	%	3 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT	3533	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	1546	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE EOSINOPHIL COUNT	0L	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE MONOCYTE COUNT	442	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT)	259000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT)	0.24	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	9	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL COUNT (P-LCC)	52000	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL RATIO (P-LCR)	20	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET DISTRIBUTION WIDTH (PDW)	15.7	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			




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IMMUNOPATHOLOGY/SEROLOGY

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

TYPHOID ANTIGEN - SERUM <i>by ICT (IMMUNOCHROMATOGRAPHY)</i>	NEGATIVE (-ve)	NEGATIVE (-ve)
TYPHI DOT ANTIBODY IgG <i>by ICT (IMMUNOCHROMATOGRAPHY)</i>	NEGATIVE (-ve)	NEGATIVE (-ve)
TYPHI DOT ANTIBODY IgM <i>by ICT (IMMUNOCHROMATOGRAPHY)</i>	NEGATIVE (-ve)	NEGATIVE (-ve)

INTERPRETATION:

Typhoid fever is a life threatening illness caused by the bacterium *Salmonella typhi*. 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 SENSITIVITY 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 MEMBRANE 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.




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. Unrelated infections may lead to production of specific Salmonella antibodies if the patient has previously been exposed to Salmonella infection (**ANAMNESTIC RESPONSE**).

NOTE:-Rapid blood culture performed during 1st 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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM	50.4	ng/mL	DEFICIENCY: < 20.0
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)			INSUFFICIENCY: 20.0 - 30.0
			SUFFICIENCY: 30.0 - 100.0
			TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFERRED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

1. Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
2. 25-OH--Vitamin D represents the main body reservoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
3. Vitamin D plays a primary role in the maintenance of calcium homeostasis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (PTH).
4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

1. Lack of sunshine exposure.
2. Inadequate intake, malabsorption (celiac disease)
3. Depressed Hepatic Vitamin D 25- hydroxylase activity
4. Secondary to advanced Liver disease
5. Osteoporosis and Secondary Hyperparathyroidism (Mild to Moderate deficiency)
6. Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:

1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphosphatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:- Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interfere with Vitamin D absorption.

*** End Of Report ***




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