

Dr. Vinay Chopra
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NAME	: Dr. DEEPIKA	PATIENT ID	: 1806758
AGE/ GENDER	: 35 YRS/FEMALE	REG. NO./LAB NO.	: 012503260036
COLLECTED BY	:	REGISTRATION DATE	: 26/Mar/2025 10:16 AM
REFERRED BY	:	COLLECTION DATE	: 26/Mar/2025 01:02PM
BARCODE NO.	: 01527796	REPORTING DATE	: 26/Mar/2025 01:12PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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SWASTHYA WELLNESS PANEL: 1.0
COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES


HAEMOGLOBIN (HB) <i>by CALORIMETRIC</i>	11.9 ^L	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	4.35	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	37.3 ^L	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	85.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	27.3	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	31.9 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	14.7	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	47.1	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	19.68	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	90.51	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>	7660	/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) %	NIL	%	< 10 %




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by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER			
<u>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</u>			
NEUTROPHILS	70	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
LYMPHOCYTES	23	%	20 - 40
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
EOSINOPHILS	1	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
MONOCYTES	6	%	2 - 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	5362	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	1762	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE EOSINOPHIL COUNT	77	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE MONOCYTE COUNT	460	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT)	235000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT)	0.34	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	14 ^H	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL COUNT (P-LCC)	131000 ^H	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL RATIO (P-LCR)	55.6 ^H	%	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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ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	56^H	mm/1st hr	0 - 20
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by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

INTERPRETATION:

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

CONDITION WITH LOW ESR

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis), and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

NOTE:

1. ESR and C - reactive protein (C-RP) are both markers of inflammation.
2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
3. **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.**
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it




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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)


GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	76.15	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
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INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.




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Test Name	Value	Unit	Biological Reference interval
LIPID PROFILE : BASIC			
CHOLESTEROL TOTAL: SERUM <i>by CHOLESTEROL OXIDASE PAP</i>	172.53	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM <i>by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)</i>	116.59	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM <i>by SELECTIVE INHIBITION</i>	56.3	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	92.91	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	116.23	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	23.32	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	461.65	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	3.06	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0





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Test Name	Value	Unit	Biological Reference interval
LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.65	RATIO	MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.07 ^L	RATIO	3.00 - 5.00

INTERPRETATION:

- Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
- As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
- Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
- NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL.
- Additional testing for Apolipoprotein B, hsCRP, Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement.




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LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	1.1	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.3	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.8	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	16.65	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	9.63	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.73	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	122	U/L	40.0 - 150.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	16	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.97	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.02	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.95	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.36	RATIO	1.00 - 2.00

INTERPRETATION


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
INTRAHEPATIC CHOLESTATIS	> 1.5




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Test Name	Value	Unit	Biological Reference interval
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 cholestasis: 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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KIDNEY FUNCTION TEST (COMPLETE)

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	15.38	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	0.67	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	7.19	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	10.73	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	22.96	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	4.21	mg/dL	2.50 - 6.80
CALCIUM: SERUM <i>by ARSENAZO III, SPECTROPHOTOMETRY</i>	9.33	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM <i>by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY</i>	4.05	mg/dL	2.30 - 4.70

ELECTROLYTES

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	138.1	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.25	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	103.57	mmol/L	90.0 - 110.0

ESTIMATED GLOMERULAR FILTRATION RATE

ESTIMATED GLOMERULAR FILTRATION RATE (eGFR): SERUM <i>by CALCULATED</i>	116.8
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INTERPRETATION:

To differentiate between pre- and post renal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased glomerular filtration rate.




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Test Name	Value	Unit	Biological Reference interval
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- Catabolic states with increased tissue breakdown.
- GI haemorrhage.
- High protein intake.
- Impaired renal function plus
- Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
- Urine reabsorption (e.g. ureter colostomy)
- Reduced muscle mass (subnormal creatinine production)
- Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

- Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
- Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN :

- Acute tubular necrosis.
- Low protein diet and starvation.
- Severe liver disease.
- Other causes of decreased urea synthesis.
- Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
- Inherited hyperammonemias (urea is virtually absent in blood).
- SIADH (syndrome of inappropriate antidiuretic hormone) due to tubular secretion of urea.
- Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

- Phenacimide therapy (accelerates conversion of creatine to creatinine).
- Rhabdomyolysis (releases muscle creatinine).
- Muscular patients who develop renal failure.


INAPPROPRIATE RATIO:

- Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
- Cephalosporin therapy (interferes with creatinine measurement).

ESTIMATED GLOMERULAR FILTRATION RATE:

CKD STAGE	DESCRIPTION	GFR (mL/min/1.73m ²)	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with normal or high GFR	>90	Presence of Protein , Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	




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Test Name	Value	Unit	Biological Reference interval
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COMMENTS:

1. Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
3. In patients, with eGFR creatinine between 45-59 ml/min/1.73 m² (G3) and without any marker of Kidney damage, It is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fulfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
7. **A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).**

ADVICE:
 KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated




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Test Name	Value	Unit	Biological Reference interval
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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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Test Name	Value	Unit	Biological Reference interval
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C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE:	26.88^H	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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM	20.5 ^L	ng/mL	DEFICIENCY: < 20.0
			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

- 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.
- 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.
- 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).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

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

INCREASED:

- 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.




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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	248	pg/mL	190.0 - 890.0
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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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CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

PHYSICAL EXAMINATION


QUANTITY RECIEVED	10	ml	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
COLOUR	PALE YELLOW		PALE YELLOW
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
TRANSPARANCY	HAZY		CLEAR
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SPECIFIC GRAVITY	1.01		1.002 - 1.030
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			


CHEMICAL EXAMINATION

REACTION	ACIDIC		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
PROTEIN	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SUGAR	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
pH	<=5.0		5.0 - 7.5
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
BILIRUBIN	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
NITRITE	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.			
UROBILINOGEN	Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
KETONE BODIES	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
BLOOD	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
ASCORBIC ACID	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			

MICROSCOPIC EXAMINATION




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RED BLOOD CELLS (RBCs) <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)	/HPF	0 - 3
PUS CELLS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	3-4	/HPF	0 - 5
EPITHELIAL CELLS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	5-8	/HPF	ABSENT
CRYSTALS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	ABSENT		ABSENT

*** End Of Report ***




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