

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

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

NAME : Mr. MANGAT RAM
AGE/ GENDER : 61 YRS/MALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 01512710
CLIENT CODE. : KOS DIAGNOSTIC LAB
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1541559
REG. NO./LAB NO. : 012407080002
REGISTRATION DATE : 08/Jul/2024 07:06 AM
COLLECTION DATE : 08/Jul/2024 08:34AM
REPORTING DATE : 08/Jul/2024 08:50AM

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) by CALORIMETRIC	15	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	5.57 ^H	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	47.5	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	85.2	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	26.9 ^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	14.4	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	46.1	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	15.3	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	22	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	6450	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL	%	< 10 %

DIFFERENTIAL LEUCOCYTE COUNT (DLC)




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NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	66	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	24	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	2	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	8	%	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>	4257	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	1548	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	129	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	516	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	/cmm	0 - 110
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	163000	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.23	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	14 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	88000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	54 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	16.5	%	15.0 - 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			



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ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR) 8 mm/1st hr 0 - 20

by MODIFIED WESTERGREN AUTOMATED METHOD

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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Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	97.61	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>	181.9	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>	129.92	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>	40.03	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>	115.89	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>	141.87 ^H	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>	25.98	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	493.72	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	4.54 ^H	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.9	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0



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Test Name	Value	Unit	Biological Reference interval
TRIGLYCERIDES/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	3.25	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>	1.72 ^H	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.39	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.33 ^H	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	26.8	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	26.5	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.01	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	64.81	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	26.52	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	7.07	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.35	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.72	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.6	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
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)




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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>	32.78	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	1.12	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	15.32	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	13.68	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	29.27	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	5.39	mg/dL	3.60 - 7.70
CALCIUM: SERUM <i>by ARSENAZO III, SPECTROPHOTOMETRY</i>	9.84	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM <i>by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY</i>	2.87	mg/dL	2.30 - 4.70

ELECTROLYTES

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	139.4	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.27	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	104.55	mmol/L	90.0 - 110.0

ESTIMATED GLOMERULAR FILTRATION RATE

ESTIMATED GLOMERULAR FILTRATION RATE (eGFR): SERUM <i>by CALCULATED</i>	74.7
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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.
2. Catabolic states with increased tissue breakdown.




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3. GI haemorrhage.
4. High protein intake.
5. Impaired renal function plus
6. 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).
7. Urine reabsorption (e.g. ureter colostomy)
8. Reduced muscle mass (subnormal creatinine production)
9. Certain drugs (e.g. tetracycline, glucocorticoids)

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

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

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

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

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

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

INAPPROPRIATE RATIO:

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
2. 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	




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


 DR.YUGAM CHOPRA
 CONSULTANT PATHOLOGIST
 MBBS, MD (PATHOLOGY)



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

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

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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




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


DR.YUGAM CHOPRA
 CONSULTANT PATHOLOGIST
 MBBS, MD (PATHOLOGY)



Dr. Vinay Chopra
 MD (Pathology & Microbiology)
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IMMUNOPATHOLOGY/SEROLOGY

RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM

RHEUMATOID (RA) FACTOR QUANTITATIVE:	4.9	IU/mL	NEGATIVE: < 18.0
SERUM			BORDERLINE: 18.0 - 25.0
by NEPHLOMETRY			POSITIVE: > 25.0

INTERPRETATION:-

RHEUMATOID FACTOR (RA):

1. Rheumatoid factors (RF) are antibodies that are directed against the Fc fragment of IgG altered in its tertiary structure.
2. Over 75% of patients with rheumatoid arthritis (RA) have an IgM antibody to IgG immunoglobulin. This autoantibody (RF) is diagnostically useful although it may not be etiologically related to RA.
3. Inflammatory Markers such as ESR & C-Reactive protein (CRP) are normal in about 60 % of patients with positive RA.
4. The titer of RF correlates poorly with disease activity, but those patients with high titers tend to have more severe disease course.
5. The test is useful for diagnosis and prognosis of rheumatoid arthritis.


RHEUMATOID ARTHRITIS:

1. Rheumatoid Arthritis is a systemic autoimmune disease that is multi-functional in origin and is characterized by chronic inflammation of the membrane lining (synovium) joints which leads to progressive joint destruction and in most cases to disability and reduction of quality life.
2. The disease spreads from small to large joints, with greatest damage in early phase.
3. The diagnosis of RA is primarily based on clinical, radiological & immunological features. The most frequent serological test is the measurement of RA factor.

CAUTION (FALSE POSTIVE):-

1. RA factor is not specific for Rheumatoid arthritis, as it is often present in healthy individuals with other autoimmune diseases and chronic infections.
2. Non rheumatoid and rheumatoid arthritis (RA) populations are not clearly separate with regard to the presence of rheumatoid factor (RF) (15% of RA patients have a nonreactive titer and 8% of nonrheumatoid patients have a positive titer).
3. Patients with various nonrheumatoid diseases, characterized by chronic inflammation may have positive tests for RF. These diseases include systemic lupus erythematosus, polymyositis, tuberculosis, syphilis, viral hepatitis, infectious mononucleosis, and influenza.
4. Anti-CCP have been discovered in joints of patients with RA, but not in other form of joint disease. Anti-CCP2 is HIGHLY SENSITIVE (71%) & more specific (98%) than RA factor.
5. Upto 30 % of patients with Seronegative Rheumatoid arthritis also show Anti-CCP antibodies.
6. The positive predictive value of Anti-CCP antibodies for Rheumatoid Arthritis is far greater than Rheumatoid factor.




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


DR. YUGAM CHOPRA
 CONSULTANT PATHOLOGIST
 MBBS, MD (PATHOLOGY)



Dr. Vinay Chopra
 MD (Pathology & Microbiology)
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 MD (Pathology)
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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM	68.1	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

- 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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 CONSULTANT PATHOLOGIST
 MBBS, MD (PATHOLOGY & MICROBIOLOGY)


 DR.YUGAM CHOPRA
 CONSULTANT PATHOLOGIST
 MBBS, MD (PATHOLOGY)



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

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

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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM 330 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 ingestion
4. Hepatocellular injury	4. Contraceptive Hormones
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.




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


DR. YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY)



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

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 MD (Pathology)
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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	CLEAR		CLEAR
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SPECIFIC GRAVITY	1.02		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	5.5		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		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
ASCORBIC ACID	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			

MICROSCOPIC EXAMINATION




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


 DR.YUGAM CHOPRA
 CONSULTANT PATHOLOGIST
 MBBS, MD (PATHOLOGY)



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


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 MD (Pathology)
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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>	1-2	/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 ***




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


DR.YUGAM CHOPRA
 CONSULTANT PATHOLOGIST
 MBBS, MD (PATHOLOGY)

