

(A Unit of KOS Healthcare)



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

NAME : Miss. BHAWNA

AGE/ GENDER : 14 YRS/FEMALE **PATIENT ID** : 1542732

COLLECTED BY : REG. NO./LAB NO. : 012407080063

 REFERRED BY
 : 08/Jul/2024 07:34 PM

 BARCODE NO.
 : 01512771
 COLLECTION DATE
 : 09/Jul/2024 07:30 PM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 09/Jul/2024 09:48 AM

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	11.5 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.12	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	35.1	%	35.0 - 49.0
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	27.9	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by calculated by automated hematology analyzer	32.7	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	14.6	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	46.4	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	20.68	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	30.18	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	9760	/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) %	NIL	%	< 10 %



MICROSCOPY

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by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER &



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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	45 ^L	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES	H	%	20 - 40
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	48 ^H	70	20 - 40
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	0	70	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	4392	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	4685	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT	98	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	90	/CITIITI	40 - 440
ABSOLUTE MONOCYTE COUNT	586	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY		, 5	
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE IMMATURE GRANULOCYTE COUNT	0	/cmm	0.0 - 999.0
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKEI	oc.		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	96000 ^L	/cmm	150000 - 450000
PLATELETCRIT (PCT)	0.13	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	18 ^H	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	60000	/cmm	30000 - 90000
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	00000	/CITIITI	20000 - 90000
PLATELET LARGE CELL RATIO (P-LCR)	80.5 ^H	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET DISTRIBUTION WIDTH (PDW)	15.8	%	15.0 - 17.0



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Test Name Value Unit Biological Reference interval

by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED.



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

Test Name Value Unit **Biological Reference interval**

ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)

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.

- ESR and C reactive protein (C-RP) are both markers of inflammation.
 Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
 CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.
 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

CLINICAL CHEMISTRY/BIOCHEMISTRY URIC ACID

URIC ACID: SERUM 4.79 mg/dL 2.50 - 6.80

by URICASE - OXIDASE PEROXIDASE

1.GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.

2.Uric Acid is the end product of purine metabolism. Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

- 1. Idiopathic primary gout.
- 2. Excessive dietary purines (organ meats, legumes, anchovies, etc).
- 3. Cytolytic treatment of malignancies especially leukemais & lymphomas.
- 4. Polycythemai vera & myeloid metaplasia.
- 5.Psoriasis.
- 6. Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCREATION (BY KIDNEYS)

- 1. Alcohol ingestion.
- 2. Thiazide diuretics.
- 3.Lactic acidosis.
- 4. Aspirin ingestion (less than 2 grams per day).
- 5. Diabetic ketoacidosis or starvation.
- 6.Renal failure due to any cause etc.

DECREASED:-

(A).DUE TO DIETARY DEFICIENCY

- 1. Dietary deficiency of Zinc, Iron and molybdenum.
- 2.Fanconi syndrome & Wilsons disease.
- 3. Multiple sclerosis.
- 4.Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCREATION

1.Drugs:-Probenecid, sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosterroids and ACTH, anti-coagulants and estrogens etc.



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Test Name Value Unit **Biological Reference interval**

IMMUNOPATHOLOGY/SEROLOGY **C-REACTIVE PROTEIN (CRP)**

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: 0.11 mg/L 0.0 - 6.0

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

3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant

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

Test Name Value Unit **Biological Reference interval**

RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM

RHEUMATOID (RA) FACTOR QUANTITATIVE: 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 2. Over 75% of patients with free ination artiffus (RA) have an igivi antibody to IgG immunoglobulin. This autoantibody (RF) is diagnouseful 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 ARTHIRITIS:

 1. Pheymatoid Arthritis is a systemic systemic

- 1. Rheumatoid Arthiritis is a systemic autoimmune disease that is multi-functional in origin and is characterized by chronic inflammation of the membrane lining (synovium) joints which ledas to progressive joint destruction and in most cases to disability and reduction of quality life.

 2. The disease spredas 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. PA factor is not specific for Phosometrial arthirities as it is often present in healthy individuals with other autoimmuno diseases and chronic infect.

- 1. RA factor is not specific for Rheumatoid arthiritis, as it is often present in healthy individuals with other autoimmune diseases and chronic infections. 2. Non 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 arthiritis also show Anti-CCP antibodies.
- 6. The positive predictive value of Anti-CCP antibodies for Rheumatoid Arthiritis is far greater than Rheumatoid factor.



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

Test Name Value Unit **Biological Reference interval**

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

21.1^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
PREFFERED 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 resevoir 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 homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (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 Hyperparathroidism (Mild to Moderate deficiency)
- 6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.
- 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 hyperphophatemia.

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 interefere with Vitamin D absorption.

*** End Of Report ***



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