

(A Unit of KOS Healthcare)



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

NAME : Mrs. MANJU BUCHAR

AGE/ GENDER : 67 YRS/FEMALE PATIENT ID : 1820466

COLLECTED BY : SURJESH REG. NO./LAB NO. : 012504070039

 REFERRED BY
 : 07/Apr/2025 10:05 AM

 BARCODE NO.
 : 01528516
 COLLECTION DATE
 : 07/Apr/2025 10:37AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 07/Apr/2025 10:49AM

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

RED DECOD CELLS (RDCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) by CALORIMETRIC	9.5 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	3.8	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	31.1 ^L	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	81.8	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	25 ^L	pg	27.0 - 34.0
$\begin{array}{l} \text{MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC)} \\ \textit{by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER} \end{array}$	30.6 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	16.2 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	49.2	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	21.53	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	114.1	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	4820	/cmm	4000 - 11000
	3 777		

NIL

NIL



NUCLEATED RED BLOOD CELLS (nRBCS)

by AUTOMATED 6 PART HEMATOLOGY ANALYZER
NUCLEATED RED BLOOD CELLS (nRBCS) %

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

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS MD (PATHOLOGY)



0.00 - 20.00

< 10 %



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by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER			
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	54	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	30	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	10	%	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	2603	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1446	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	289	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	482	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIV	E MARKERS.		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	193000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.25	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	13 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	90000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	46.5 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW)	15.8	%	15.0 - 17.0



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CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)



KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana



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



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0171-2643898, +91 99910 43898 | care@koshealthcare.com | www.koshealthcare.com



KOS Diagnostic Lab (A Unit of KOS Healthcare)



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 : 07/Apr/2025 11:16AM

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PERIPHERAL BLOOD SMEAR

TEST NAME:

PERIPHERAL BLOOD FILM/SMEAR (PBF)

RED BLOOD CELLS (RBC'S):

Mild anisocytosis with microcytes.RBCs reveal mild hypochromia.No polychromatic cells or normoblasts noted.

WHITE BLOOD CELLS (WBC'S):

No immature leucocytes seen.

PLATELETS:

Platelets appear adequate.

HEMOPARASITES:

NOT SEEN.

IMPRESSION:

Mild microcytic hypochromic picture.



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 : 07/Apr/2025 12:25PM

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Test Name Value Unit Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

IRON PROFILE

IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	30.3 ^L	μg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM	371.06^{H}	μg/dL	150.0 - 336.0
by FERROZINE, SPECTROPHOTOMETERY			
TOTAL IRON BINDING CAPACITY (TIBC)	401.36	μg/dL	230 - 430
:SERUM by SPECTROPHOTOMETERY			
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	7.55^{L}	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)	284.97	mg/dL	200.0 - 350.0

INTERPRETATION:-

WIERFRETATION.			
VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:

- 1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia, anemia of chronic disease and thalassemia syndromes.
- 2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BÍNDING CAPACITY (TÍBC):

1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:

1.Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.



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Value Unit Test Name **Biological Reference interval**

FERRITIN

FERRITIN: SERUM 28.28 4.63 - 204.0 ng/mL

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy.

DECREASED:

- 1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.
- 2. Hypothyroidism.3. Vitamin-C deficiency

INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

- 1. Hemochromatosis or hemosiderosis.
- 2. Wilson Disease

INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

- 1. Transfusion overload
- 2. Excess dietary Iron
- 3. Porphyria Cutanea tada
- 4. Ineffective erythropoiesis

INCREASED FERRITIN WITHOUT IRON OVERLOAD:

- Liver disorders (NASH) or viral hepatitis (B/C).
 Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
- 3. Leukaemia, hodgkin's disease.
- 4. Alcohol excess.
- 5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma, synthesis of ferritin by tumour cells.

 6. Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can thererfore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive proteins to rule out any inflammatory conditions.

2. Patients with iron déficiency anaemia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.



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VITAMINS VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM 439 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 Igestion		
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.

*** End Of Report ***



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