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<b>NAME</b>	: Mrs. SEEMA SHARMA	<b>PATIENT ID</b>	: 1602651
<b>AGE/ GENDER</b>	: 59 YRS/FEMALE	<b>REG. NO./LAB NO.</b>	: <b>012409050037</b>
<b>COLLECTED BY</b>	: SURJESH	<b>REGISTRATION DATE</b>	: 05/Sep/2024 10:54 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 05/Sep/2024 10:58AM
<b>BARCODE NO.</b>	: 01516340	<b>REPORTING DATE</b>	: 05/Sep/2024 11:20AM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
<b>CLIENT ADDRESS</b>	: 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**

<b>HAEMOGLOBIN (HB)</b> <i>by CALORIMETRIC</i>	10.6 <sup>L</sup>	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	4.38	Millions/cmm	3.50 - 5.00
<b>PACKED CELL VOLUME (PCV)</b> <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	35.1 <sup>L</sup>	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	80.1	fL	80.0 - 100.0
<b>MEAN CORPUSCULAR HAEMOGLOBIN (MCH)</b> <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	24.3 <sup>L</sup>	pg	27.0 - 34.0
<b>MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC)</b> <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	30.2 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	16	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	49.1	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	18.29	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	29.38	RATIO	BETA THALASSEMIA TRAIT: <= 65.0 IRON DEFICIENCY ANEMIA: > 65.0

**WHITE BLOOD CELLS (WBCS)**

TOTAL LEUCOCYTE COUNT (TLC) <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	6340	/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) % <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	NIL	%	< 10 %

**DIFFERENTIAL LEUCOCYTE COUNT (DLC)**

<b>NEUTROPHILS</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	73 <sup>H</sup>	%	50 - 70
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<b>LYMPHOCYTES</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	13 <sup>L</sup>	%	20 - 40
<b>EOSINOPHILS</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0 <sup>L</sup>	%	1 - 6
<b>MONOCYTES</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	14 <sup>H</sup>	%	2 - 12
<b>BASOPHILS</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	%	0 - 1
<b><u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u></b>			
<b>ABSOLUTE NEUTROPHIL COUNT</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	4628	/cmm	2000 - 7500
<b>ABSOLUTE LYMPHOCYTE COUNT</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	824 <sup>L</sup>	/cmm	800 - 4900
<b>ABSOLUTE EOSINOPHIL COUNT</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0 <sup>L</sup>	/cmm	40 - 440
<b>ABSOLUTE MONOCYTE COUNT</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	888 <sup>H</sup>	/cmm	80 - 880
<b>ABSOLUTE BASOPHIL COUNT</b> <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	/cmm	0 - 110
<b><u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u></b>			
<b>PLATELET COUNT (PLT)</b> <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	385000	/cmm	150000 - 450000
<b>PLATELET CRIT (PCT)</b> <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.35	%	0.10 - 0.36
<b>MEAN PLATELET VOLUME (MPV)</b> <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	9	fL	6.50 - 12.0
<b>PLATELET LARGE CELL COUNT (P-LCC)</b> <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	75000	/cmm	30000 - 90000
<b>PLATELET LARGE CELL RATIO (P-LCR)</b> <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	19.4	%	11.0 - 45.0
<b>PLATELET DISTRIBUTION WIDTH (PDW)</b> <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	15.5	%	15.0 - 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			



  
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**IMMUNOPATHOLOGY/SEROLOGY**

**DENGUE FEVER COMBO SCREENING - (NS1 ANTIGEN, IgG AND IgM)**

DENGUE NS1 ANTIGEN - SCREENING <i>by ICT (IMMUNOCHROMATOGRAPHY)</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
DENGUE ANTIBODY IgG - SCREENING <i>by ICT (IMMUNOCHROMATOGRAPHY)</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
DENGUE ANTIBODY IgM - SCREENING <i>by ICT (IMMUNOCHROMATOGRAPHY)</i>	NEGATIVE (-ve)		NEGATIVE (-ve)

**INTERPRETATION:-**

- This is a solid phase immunochromatographic ELISA test for the qualitative detection of the specific IgG and IgM antibodies against the Dengue virus.
- The IgM antibodies take a minimum of 5-10 days in primary infection and 4-5 days in secondary infections to test positive and hence are suitable for the diagnosis of dengue fever only when the fever is approximately one week old.
- The IgG antibodies develop at least two weeks after exposure to primary infection and subsequently remain positive for the rest of the life. A positive result is incapable of differentiating a current infection from a past infection.
- The Dengue NS-1 antigen test is most suited for early diagnosis (within the first week of exposure).



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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**VITAMINS**

**VITAMIN D/25 HYDROXY VITAMIN D3**

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM <i>by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)</i>	30.5	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
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**INTERPRETATION:**

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

\*\*\* End Of Report \*\*\*



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