

MAHESH L DESHPANDE
Male/51 Years

Reg. Date : **29/03/2023**
Lab. No **482509-18**
Sample No
9052

Ref. Dr.

Dr. POOJA D TANDEL M D MEDICINE
VIBRANT HOSPITAL VAP1

HEMATOLOGY REPORT

Test	Result	Unit	Ref. Range
Haemoglobin:	12.8	g/dL	13.0 - 17.0 g/dL
Total Leucocyte Count:	8120	X 10 ³ / μ L	4000 - 10000 /uL
Differential Count			
Neutrophils:	84	%	40-80
Eosinophils:	01	%	1.0-6.0
Basophils:	00	%	<1-2
Lymphocytes:;	13	%	M: 20-40; F: 20-40
Monocytes:	02	%	2-10
Neutrophils Absolute Count:	6.85	X 10 ³ / μ L	2.0-7.0
Eosinophils Absolute Count:	0.04	X 10 ³ / μ L	0.02-0.50
Basophils Absolute Count:	0.02	X 10 ³ / μ L	0.02-0.10
Lymphocytes Absolute Count:	1.02	X 10 ³ / μ L	1.0-3.0
Monocytes Absolute Count:	0.19	X 10 ³ / μ L	0.2-1.0
Total RBC Count:	4.24	X 10 ⁶ / μ L	M: 4.5-5.5; F: 3.9-4.8
Hematocrit (HCT):	36.1	%	42 - 52 %
MCV:	85.1	fL	83 - 101
MCH:	30.3	pg	27-32
MCHC:	35.5	g/dL	31.5 - 34.5
RDW-SD:	42.9	fL	39 - 46
RDW-CV:	12.1	%	11.6 - 14.0
Platelets Count:	254000	/ μ L	150000 - 400000
Plateletcrit (PCT):	0.226	%	
Mean Platelet Volume	8.9	fL	
Malariaial Parasite	M.P. are not seen		

Method: Fully automated bidirectional interfaced analyser (6 Part Differential **SYSMEX XN-1000**).

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Dr. Mehul SOLANKY
M.D.(Path & Bact)



IMMUNO-DIAGNOSTIC & PATHOLOGY LABORATORY

Halar Road Cross Lane, Besides L.I.C. Bldg.
Valsad-396 001. Ph.: (02632) 243280. Mo.: 99250 49280

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MALARIAL ANTIGEN DETECTION TEST

Malarial Antigen Detection Test :

P. Vivax Antigen Detection Test : Negative

P. Falciparum Antigen Detection Test : Negative

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BIOCHEMISTRY REPORT

Test	Result	Unit	Ref. Range
S.G.P.T. (ALT):	20.99	IU/L	10 - 40 IU/L
S.Creatinine:	0.81	mg/dL	0.60 - 1.30 mg/dL

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DENGUE ANTIGEN & ANTIBODY TEST

Test : Dengue IgM / IgG Antibody

Method : Immunochromatography

Result :

Dengue IgM : Negative

Dengue IgG : Negative

DENGUE NS1 ANTIGEN Negative

EXPECTED VALUE

The NS1 is expected to be detected 1 day after the onset of fever and persists up to 9 days in both Primary & Secondary dengue infection. But if anti NS1 Antibodies is produced, detection of NS1 is inhibited. **Primary Dengue** is characterized by the presence of detectable IgM 3- 5 days after the onset of infection. **Secondary Dengue** is characterized by the elevation of Specific IgG after the onset of infection and in the majority of the cases this is accompanied by elevation of IgM.

INTERPRETATION AND LIMITATIONS OF THE ASSAY

- 1) Method - Rapid Solid Phase Immunocromatographic test.
- 2) The test detects the presence of Dengue NS1 Antigen & IgM & IgG Antibodies to dengue virus in the specimen and should not be used as the sole criteria for diagnosis of DENGUE virus infection.
- 3) In early infections & some secondary infections, Detectable lvels of IgM Antibodies may be low. Some patients may not produce detectable levels of Antiboies within the first Seven too Ten days after infection. Hence negative reasult can not exclude a recent early infection and must be retested after 3- 5 days after the firs test.
- 4) Serological cross reactivity across the fFavivirus group (Dengue virus, Japnes encephlitis, St Louis encephlitis, West Nile encephlitis and Yellow fever) is common.
- 5) As with all diagnostic tests, all results must be corelated with the other clinical findings. If the test result is negative and clinical symptoms persis, additional follow up testing using other clinical methods
- 6) This is a screening test only, Therefore, Isolation of Virus, Antigen detection in fixed tissue, RT-PCR & Serological test like haemagglutination test must be used to obtain a confirmation of Dengue Virus infection. is recomended.

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URINE REPORT

Test	Result	Unit	Ref. Range
<u>PHYSICAL EXAMINATION</u>			
QUANTITY	30	mL	500 - 2000/24 HRS
COLOUR	PALE YELLOW		
APPEARANCE	Clear	Clear	
REACTION(PH)	6.0		4.6 - 8.0
SPECIFIC GRAVITY	1.025		1.005 - 1.030
<u>CHEMICAL EXAMINATION</u>			
URINE ALBUMIN	Nil		Absent
URINE SUGAR(Qualitative)	Nil		Absent
KETONES	Absent		Absent
BILE SALTS	Absent		Absent
BILE PIGMENTS	Absent		Absent
UROBILOGEN	Normal		Normal
BLOOD	Absent		Absent
<u>MICROSCOPIC EXAMINATION</u>			
PUS CELLS/HPF	2 - 3 / HPF	/HPF	1 - 5
RED BLOOD CELLS/HPF	Absent	/HPF	0 - 2/hpf
EPITHELIAL CELLS/HPF	1 - 2 / HPF	/HPF	
CASTS/LPF	Absent		
CRYSTALS	Absent		
BACTERIA	ABSENT		
YEAST	Absent		
NOTE			
REMARK			

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S.TYPHI. SPECIFIC IgM ANTIBODY DETECTION TEST

Result:

S.Typhi IgM : Negative

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C - REACTIVE PROTEIN TITRE

Test	Result	Unit	Ref. Range
CRP :	<u>11.34</u>	mg/L	Adult < 6.0 mg/L Newborn upto 3 weeks < 4.1 mg/L Infants & Children < 2.8 mg/L
Method	BY IMMUNOTURBIDOMETRIC METHOD.		

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SERUM VITAMIN-D LEVEL

Test	Result	Unit	Ref. Range
S. VITAMIN D Level : (25-Hydroxy vitamin D)	18.6	ng/ml	11.1 - 42.9 ng/mL > 30 nG/mL IS DESIRABLE

INTERPRETATION

- Vitamin D is a fat-soluble steroid hormone precursor that is mainly produced in the skin by exposure to sunlight.
 - Vitamin D is biologically inert and must undergo two successive hydroxylations in the liver and kidney to become the biologically active 1,25-dihydroxyvitamin D.
 - The two most important forms of vitamin D are vitamin D₃ (cholecalciferol) and vitamin D₂ (ergocalciferol).
 - In contrast to vitamin D₃, the human body cannot produce vitamin D₂ which is taken up with fortified food or given by supplements.
 - In human plasma vitamin D₃ and D₂ are bound to the vitamin D binding protein and transported to the liver where both are hydroxylated to form vitamin D (25-OH), i.e. 25-hydroxyvitamin D.
 - **It is commonly agreed that vitamin D (25-OH) is the metabolite to determine the overall vitamin D status as it is the major storage form of vitamin D in the human body.**
- This primary circulating form of vitamin D is biologically inactive with levels approximately 1000-fold greater than the circulating 1,25-dihydroxyvitamin D. The half-life of circulating vitamin D (25-OH) is 2-3 weeks.
- Most of the vitamin D (25-OH), measurable in serum, is vitamin D₃ (25-OH) whereas vitamin D₂ (25-OH) reaches measurable levels only in patients taking vitamin D₂ supplements. Vitamin D₂ is considered to be less effective.
 - Vitamin D is essential for bone health. In children, severe deficiency leads to bone-malformation, known as rickets. Milder degrees of insufficiency are believed to cause reduced efficiency in the utilization of dietary calcium.
 - Vitamin D deficiency causes muscle weakness; in elderly, the risk of falling has been attributed to the effect of vitamin D on muscle function.
 - Vitamin D deficiency is a common cause of secondary hyperparathyroidism.
 - Elevations of PTH levels, especially in elderly vitamin D deficient adults can result in osteomalacia, increased bone turnover, reduced bone mass and risk of bone fractures.
 - Low vitamin D (25-OH) concentrations are also associated with lower bone mineral density.
 - In conjunction with other clinical data, the results may be used as an aid in the assessment of bone metabolism.
 - So far, vitamin D has been shown to affect expression of over 200 different genes.
 - Insufficiency has been linked to diabetes, different forms of cancer, cardiovascular disease, autoimmune diseases and innate immunity.

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