



CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHÍ

NEW DELHI 110030 8800465156

ACCESSION NO: 0080WL004375

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO

DRAWN

:47 Years

AGE/SEX

RECEIVED: 14/12/2023 08:56:43 REPORTED :15/12/2023 17:11:06

Biological Reference Interval **Test Report Status** Results Units <u>Final</u>

| MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE BLOOD COUNTS, EDTA WHOLE BLOOD | HAEMATOLOGY - CBC | | | | | |
|---|---|--------------|--------------|---------|--|--|
| HEMOGLOBIN (HB) 15.3 13.0 - 17.0 9/dL METHOD : CYANNETHEMOCLOBIN METHOD METHOD : CYANNETHEMOCLOBIN METHOD RED BLOOD CELL (RBC) COUNT 5.05 4.5 - 5.5 mil/μL WHITE BLOOD CELL (WBC) COUNT 6.10 4.0 - 10.0 thou/μL PLATELET COUNT 206 150 - 410 thou/μL PLATELET COUNT 206 150 - 410 thou/μL RBC AND PLATELET INDICES HEMATOCRIT (PCV) 44.7 40.0 - 50.0 % MEAN CORPUSCULAR VOLUME (MCV) 88.4 83.0 - 101.0 fL METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM MEAN CORPUSCULAR HEMOGLOBIN (MCH) 30.3 27.0 - 32.0 pg METHOD : CALCULATED PARAMETER MEAN CORPUSCULAR HEMOGLOBIN (MCH) 34.3 31.5 - 34.5 g/dL CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER MEAN CORPUSCULAR HEMOGLOBIN 17.5 11.6 - 14.0 % METHOD : CALCULATED PARAMETER MENTZER INDEX 17.5 | MEDI WHEEL FULL BODY HEALTH CHECK UP A | BOVE 40 MALE | | | | |
| RED BLOOD CELL (RBC) COUNT 5.05 4.5 - 5.5 mil/µL WHITE BLOOD CELL (WBC) COUNT 6.10 4.0 - 10.0 thou/µL PLATELET COUNT 206 150 - 410 thou/µL RBC AND PLATELET INDICES HEMATOCRIT (PCV) 44.7 40.0 - 50.0 % MEAN CORPUSCULAR VOLUME (MCV) 88.4 83.0 - 101.0 fL METHOD: DERIVED PARAMETER FROM RBC HISTOGRAM MEAN CORPUSCULAR HEMOGLOBIN (MCH) 30.3 27.0 - 32.0 pg METHOD: CALCULATED PARAMETER MEAN CORPUSCULAR HEMOGLOBIN (MCH) 34.3 31.5 - 34.5 g/dL CONCENTRATION (MCHC) METHOD: CALCULATED PARAMETER MEAN CORPUSCULAR HEMOGLOBIN 12.9 11.6 - 14.0 % METHOD: CALCULATED PARAMETER MENTZER INDEX 17.5 MEAN PLATELET VOLUME (MPV) 8.0 6.8 - 10.9 fL METHOD: DERIVED PARAMETER FROM RBC HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 1.0 - 6.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 0 1 0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 0 1 0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 | BLOOD COUNTS,EDTA WHOLE BLOOD | | | | | |
| RED BLOOD CELL (RBC) COUNT 5.05 | HEMOGLOBIN (HB) | 15.3 | 13.0 - 17.0 | g/dL | | |
| WHITE BLOOD CELL (WBC) COUNT 6.10 4.0 - 10.0 thou/μL PLATELET COUNT 206 150 - 410 thou/μL RBC AND PLATELET INDICES Thou/μL Thou/μL HEMATOCRIT (PCV) 44.7 40.0 - 50.0 % MEAN CORPUSCULAR VOLUME (MCV) 88.4 83.0 - 101.0 fL MEAN CORPUSCULAR HEMOGLOBIN (MCH) 30.3 27.0 - 32.0 pg MEAN CORPUSCULAR HEMOGLOBIN (MCH) 34.3 31.5 - 34.5 g/dL CONCENTRATION (MCHC) METHOD: CALCULATED PARAMETER g/dL MEAN CORPUSCULAR HEMOGLOBIN (MCHC) 12.9 11.6 - 14.0 % METHOD: CALCULATED PARAMETER METHOD: | METHOD: CYANMETHEMOGLOBIN METHOD | | | | | |
| PLATELET COUNT | RED BLOOD CELL (RBC) COUNT | 5.05 | 4.5 - 5.5 | mil/μL | | |
| REC AND PLATELET INDICES HEMATOCRIT (PCV) | WHITE BLOOD CELL (WBC) COUNT | 6.10 | 4.0 - 10.0 | thou/µL | | |
| HEMATOCRIT (PCV) | PLATELET COUNT | 206 | 150 - 410 | thou/µL | | |
| MEAN CORPUSCULAR VOLUME (MCV) 88.4 83.0 - 101.0 fL METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM MEAN CORPUSCULAR HEMOGLOBIN (MCH) 30.3 27.0 - 32.0 pg METHOD : CALCULATED PARAMETER MEAN CORPUSCULAR HEMOGLOBIN 34.3 31.5 - 34.5 g/dL CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER RED CELL DISTRIBUTION WIDTH (RDW) 12.9 11.6 - 14.0 % METHOD : CALCULATED PARAMETER MENTZER INDEX 17.5 MEAN PLATELET VOLUME (MPV) 8.0 6.8 - 10.9 fL METHOD : DERIVED PARAMETER ROM PLATELET HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROPHILS 50 40 80 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 2.0 - 10.0 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD : LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EASOPHILS 0 0 0 - 7.0 thou/µL | RBC AND PLATELET INDICES | | | | | |
| METHOD: DERIVED PARAMETER FROM RBC HISTOGRAM MEAN CORPUSCULAR HEMOGLOBIN (MCH) 30.3 27.0 - 32.0 pg METHOD: CALCULATED PARAMETER MEAN CORPUSCULAR HEMOGLOBIN 34.3 31.5 - 34.5 g/dL CONCENTRATION (MCHC) METHOD: CALCULATED PARAMETER RED CELL DISTRIBUTION WIDTH (RDW) 12.9 11.6 - 14.0 % METHOD: CALCULATED PARAMETER MENTZER INDEX 17.5 MEAN PLATELET VOLUME (MPV) 8.0 6.8 - 10.9 fL METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROPHILS 50 40 80 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES MONOCYTES 3 1.0 - 6.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | HEMATOCRIT (PCV) | 44.7 | 40.0 - 50.0 | % | | |
| METHOD: CALCULATED PARAMETER MEAN CORPUSCULAR HEMOGLOBIN 34.3 CONCENTRATION (MCHC) METHOD: CALCULATED PARAMETER RED CELL DISTRIBUTION WIDTH (RDW) 12.9 METHOD: CALCULATED PARAMETER MENTZER INDEX 17.5 MEAN PLATELET VOLUME (MPV) 8.0 6.8 - 10.9 METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROPHILS 50 40 - 80 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | ` , | 88.4 | 83.0 - 101.0 | fL | | |
| CONCENTRATION (MCHC) METHOD: CALCULATED PARAMETER RED CELL DISTRIBUTION WIDTH (RDW) 12.9 11.6 - 14.0 % METHOD: CALCULATED PARAMETER MENTZER INDEX 17.5 MEAN PLATELET VOLUME (MPV) 8.0 6.8 - 10.9 fL METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROPHILS 50 40 80 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | 30.3 | 27.0 - 32.0 | pg | | |
| METHOD: CALCULATED PARAMETER MENTZER INDEX 17.5 MEAN PLATELET VOLUME (MPV) METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROPHILS METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES MONOCYTES MONOCYTES METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | CONCENTRATION (MCHC) | 34.3 | 31.5 - 34.5 | g/dL | | |
| MENTZER INDEX 17.5 MEAN PLATELET VOLUME (MPV) 8.0 6.8 - 10.9 fL METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROPHILS 50 40 - 80 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE V 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE V V V BASOPHILS 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE V V ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | RED CELL DISTRIBUTION WIDTH (RDW) | 12.9 | 11.6 - 14.0 | % | | |
| MEAN PLATELET VOLUME (MPV) 8.0 6.8 - 10.9 fL METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROPHILS 50 40 - 80 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | | | | | |
| METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM WBC DIFFERENTIAL COUNT NEUTROPHILS 50 40 - 80 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | MENTZER INDEX | | | | | |
| WBC DIFFERENTIAL COUNT NEUTROPHILS 50 40 - 80 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | , | 8.0 | 6.8 - 10.9 | fL | | |
| NEUTROPHILS METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 BASOPHILS 0 0 0 - 1 METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | | | | | |
| METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | | 4000 | 0/ | | |
| LYMPHOCYTES 40 20 - 40 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | | 40 - 80 | % | | |
| METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE MONOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | | 20 - 40 | 0/0 | | |
| MONOCYTES 7 2.0 - 10.0 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | | 20 - 40 | 70 | | |
| METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE EOSINOPHILS 3 1.0 - 6.0 % BASOPHILS 0 0 1 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | | 2.0 - 10.0 | % | | |
| BASOPHILS 0 0 0 - 1 % METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | | IMPEDENCE | | | | |
| METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | EOSINOPHILS | 3 | 1.0 - 6.0 | % | | |
| ABSOLUTE NEUTROPHIL COUNT 3.05 2.0 - 7.0 thou/µL | BASOPHILS | 0 | 0 - 1 | % | | |
| | METHOD: LIGHT ABSORBANCE OF CYTCHEMICAL STAINED CELLS IMPEDENCE | | | | | |
| ABSOLUTE LYMPHOCYTE COUNT 2.44 1.0 - 3.0 thou/µL | ABSOLUTE NEUTROPHIL COUNT | 3.05 | 2.0 - 7.0 | thou/µL | | |
| | ABSOLUTE LYMPHOCYTE COUNT | 2.44 | 1.0 - 3.0 | thou/μL | | |



Remember

Dr.Pranjali Vasisht LAB HEAD

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DR.CHANDNI GARG

Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India Tel: 9111591115, Fax:

CIN - U74899PB1995PLC045956

CONSULTANT PATHOLOGIST







CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

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:47 Years

| | <u> </u> | <u> </u> | |
|-----------------------------------|----------|----------------------|----------------|
| Test Report Status <u>Final</u> | Results | Biological Reference | Interval Units |
| | | | |
| ABSOLUTE MONOCYTE COUNT | 0.43 | 0.2 - 1.0 | thou/μL |
| ABSOLUTE EOSINOPHIL COUNT | 0.18 | 0.02 - 0.50 | thou/μL |
| ABSOLUTE BASOPHIL COUNT | 0 Low | 0.02 - 0.10 | thou/μL |
| METHOD: CALCULATED PARAMETER | | | |
| NEUTROPHIL LYMPHOCYTE RATIO (NLR) | 1.2 | | |

METHOD: CALCULATED PARAMETER

Interpretation(s)
BLOOD COUNTS,EDTA WHOLE BLOOD-The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading RBC AND PLATELET INDICES-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13)

from Beta thalassaemia trait

(<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT-The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to show mild disease.
(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients A.-P. Yang, et al. International Immunopharmacology 84 (2020) 106504

This ratio element is a calculated parameter and out of NABL scope.

Chardni gary

DR.CHANDNI GARG CONSULTANT PATHOLOGIST Personalis

Dr.Pranjali Vasisht LAB HEAD





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Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India Tel: 9111591115, Fax:







CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030

8800465156

ACCESSION NO: 0080WL004375

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO

AGE/SEX DRAWN

RECEIVED: 14/12/2023 08:56:43

:47 Years

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Test Report Status Biological Reference Interval <u>Final</u> Results Units

HAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD

0 - 14E.S.R

mm at 1 hr

METHOD: MODIFIED WESTERGREN

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE **BLOOD**

HBA1C 5.5 Non-diabetic Adult < 5.7 %

Pre-diabetes 5.7 - 6.4

Diabetes diagnosis: > or = 6.5Therapeutic goals: < 7.0

Action suggested: > 8.0 (ADA Guideline 2021)

ESTIMATED AVERAGE GLUCOSE(EAG) 111.2 < 116.0 mg/dL

Interpretation(s)

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD-**TEST DESCRIPTION**:Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of clear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

ESR is not diagnostic it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition.CRP is superior to ESR because it is more sensitive and reflects a more rapid change.

TEST INTERPRETATION

Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy, Estrogen medication, Aging

Finding a very accelerated ESR(>100 mm/hour) in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).

In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm /hr(95 if anemic). ESR returns to normal 4th week post partum.

Decreased in: Polycythermia vera, Sickle cell anemia

LIMITATIONS

False elevated ESR: Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia

False Decreased: Poikilocytosis, (SickleCells, spherocytes), Microcytosis, Low fibrinogen, Very high WBC counts, Drugs (Quinine,

salicylates)

REFERENCE :

- 1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition 2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin 3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis,10th edition. GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-Used For:
- 1. Evaluating the long-term control of blood glucose concentrations in diabetic patients.
- 2. Diagnosing diabetes.
- 3. Identifying patients at increased risk for diabetes (prediabetes).

The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to determine whether a patients metabolic control has remained continuously within the target range.





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Dr. Praniali Vasisht LAB HEAD

DR.CHANDNI GARG CONSULTANT PATHOLOGIST





Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India Tel: 9111591115, Fax:







CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

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:47 Years

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Test Report Status Results **Biological Reference Interval** <u>Final</u> Units

- 1. eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
- eAG gives an evaluation of blood glucose levels for the last couple of months.
 eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c 46.7

- **HbA1c Estimation can get affected due to :**1. Shortened Erythrocyte survival : Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss, hemolytic anemia) will falsely lower HbA1c test results. Fructosamine is recommended in these patients which indicates diabetes control over 15 days. 2. Vitamin C & E are reported to falsely lower test results. (possibly by inhibiting glycation of hemoglobin.
- 3. Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia, uremia, hyperbilirubinemia, chronic alcoholism, chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods, falsely increasing results.

 4. Interference of hemoglobinopathies in HbA1c estimation is seen in
- a) Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
- b) Heterozygous state detected (D10 is corrected for HbS & HbC trait.)
- c) HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy

Pourculit

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CONSULTANT PATHOLOGIST

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Page 4 Of 16



Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India

Tel: 9111591115, Fax: CIN - U74899PB1995PLC045956



Patient Ref. No. 775000005752046





CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030

8800465156

ACCESSION NO: 0080WL004375

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO

AGE/SEX :47 Years

DRAWN

RECEIVED: 14/12/2023 08:56:43 REPORTED :15/12/2023 17:11:06

Test Report Status Results **Biological Reference Interval** Units <u>Final</u>

IMMUNOHAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

TYPE B **ABO GROUP**

METHOD: SLIDE AGGLUTINATION

RH TYPE **POSITIVE**

METHOD: SLIDE AGGLUTINATION

Interpretation(s)
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

Disclaimer: "Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same.

The test is performed by both forward as well as reverse grouping methods.

Resealed

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CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

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BIOCHEMISTRY

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR) 108 High 74 - 106 mg/dL

METHOD: HEXOKINASE

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR) Non-Diabetes mg/dL 128

70 - 140

METHOD: HEXOKINASE

LIPID PROFILE WITH CALCULATED LDL

CHOLESTEROL, TOTAL 247 High < 200 Desirable mg/dL

200 - 239 Borderline High

>/= 240 High

METHOD: CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE TRIGLYCERIDES 213 High

< 150 Normal mg/dL 150 - 199 Borderline High

200 - 499 High >/= 500 Very High

METHOD: ENZYMATIC ASSAY

HDL CHOLESTEROL 38 Low < 40 Low mg/dL

>/=60 High

METHOD: DIRECT MEASURE - PEG

CHOLESTEROL LDL 166 High < 100 Optimal mg/dL

100 - 129

Near or above optimal

130 - 159 Borderline High 160 - 189 High >/= 190

Very High

METHOD: CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE

NON HDL CHOLESTEROL 38 Desirable: Less than 130 mg/dL

> Above Desirable: 130 - 159 Borderline High: 160 - 189

High: 190 - 219

Very high: > or = 220

METHOD: CALCULATED PARAMETER

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Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India Tel: 9111591115, Fax:







Male

PATIENT NAME: DR SRIVASTAVA ASHISH MOHAN REF. DOCTOR: SELF

CODE/NAME & ADDRESS: C000138383

ARCOFEMI HEALTHCARE LTD (MEDIWHEEL
F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHÍ

NEW DELHI 110030

8800465156

ACCESSION NO: 0080WL004375

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO : AGE/SEX : DRAWN :

AWN :

:47 Years

RECEIVED : 14/12/2023 08:56:43 REPORTED :15/12/2023 17:11:06

| rest report status filial results biological reference interval only | Test Report Status | Final | Results E | Biological Reference Interval | Units |
|--|--------------------|-------|-----------|-------------------------------|-------|
|--|--------------------|-------|-----------|-------------------------------|-------|

VERY LOW DENSITY LIPOPROTEIN 42.6 High Desirable value: mg/dL

10 - 35

METHOD: CALCULATED PARAMETER

CHOL/HDL RATIO 6.5 High 3.3-4.4 Low Risk 4.5-7.0 Average Risk

7.1-11.0 Moderate Risk > 11.0 High Risk

METHOD: CALCULATED PARAMETER

LDL/HDL RATIO 4.4 High 0.5 - 3.0 Desirable/Low Risk

3.1 - 6.0 Borderline/Moderate

Risk

>6.0 High Risk

METHOD: CALCULATED PARAMETER

Interpretation(s)

Serum lipid profile is measured for cardiovascular risk prediction. Lipid Association of India recommends LDL-C as primary target and Non HDL-C as co-primary treatment target.

Risk Stratification for ASCVD (Atherosclerotic cardiovascular disease) by Lipid Association of India

| Risk Category | | | | | |
|--|---|---|--|--|--|
| Extreme risk group | A.CAD with > 1 feature of high risk group | A.CAD with > 1 feature of high risk group | | | |
| | B. CAD with > 1 feature of Very high risk g | group or recurrent ACS (within 1 year) despite LDL-C < or = | | | |
| | 50 mg/dl or polyvascular disease | 50 mg/dl or polyvascular disease | | | |
| Very High Risk | Established ASCVD 2. Diabetes with 2 r | major risk factors or evidence of end organ damage 3. | | | |
| | Familial Homozygous Hypercholesterolemi | a | | | |
| High Risk | 1. Three major ASCVD risk factors. 2. Diabetes with 1 major risk factor or no evidence of end organ | | | | |
| | damage. 3. CKD stage 3B or 4. 4. LDL >190 mg/dl 5. Extreme of a single risk factor. 6. Coronary | | | | |
| | Artery Calcium - CAC >300 AU. 7. Lipoprotein a >/= 50mg/dl 8. Non stenotic carotid plaque | | | | |
| Moderate Risk | 2 major ASCVD risk factors | | | | |
| Low Risk | 0-1 major ASCVD risk factors | | | | |
| Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors | | | | | |
| Age > or = 45 years in males and > or = 55 years in females Current Cigarette smoking or tobacco use | | | | | |
| Family history of premature ASCVD 4. High blood pressure | | | | | |
| 5. Low HDL | | | | | |

Newer treatment goals and statin initiation thresholds based on the risk categories proposed by LAI in 2020.

| ٠ | server treatment Bours und stutin in | inclination currently follows | ca on the risk entegori | es proposed by an | it im avavi |
|---|--------------------------------------|--|--|-----------------------|-----------------|
| | Risk Group | Treatment Goals | | Consider Drug Therapy | |
| | | LDL-C (mg/dl) | Non-HDL (mg/dl) | LDL-C (mg/dl) | Non-HDL (mg/dl) |
| | Extreme Risk Group Category A | <50 (Optional goal | < 80 (Optional goal | >OR = 50 | >OR = 80 |
| | | < OR = 30) | <or 60)<="" =="" td=""><td></td><td></td></or> | | |
| | Extreme Risk Group Category B | <or 30<="" =="" td=""><td><or 60<="" =="" td=""><td>> 30</td><td>>60</td></or></td></or> | <or 60<="" =="" td=""><td>> 30</td><td>>60</td></or> | > 30 | >60 |
| | Very High Risk | <50 | <80 | >OR= 50 | >OR= 80 |
| | High Risk | <70 | <100 | >OR= 70 | >OR= 100 |

Consult

Dr. Pranjali Vasisht

LAB HEAD

Chaidni Jary

DR.CHANDNI GARG
CONSULTANT PATHOLOGIST



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Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India Tel: 9111591115, Fax:







Units

PATIENT NAME: DR SRIVASTAVA ASHISH MOHAN REF. DOCTOR: SELF

CODE/NAME & ADDRESS: C000138383

ARCOFEMI HEALTHCARE LTD (MEDIWHEEL
F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030 8800465156 ACCESSION NO : 0080WL004375

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO : AGE/SEX DRAWN

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:47 Years

| Test Report Status | <u>Final</u> | Results | Biological Reference Interval |
|---------------------------|--------------|---------|--------------------------------------|
|---------------------------|--------------|---------|--------------------------------------|

| Moderate Risk | <100 | <130 | >OR= 100 | >OR= 130 |
|---------------|------|------|-----------|----------|
| Low Risk | <100 | <130 | >OR= 130* | >OR= 160 |

^{*}After an adequate non-pharmacological intervention for at least 3 months.

References: Management of Dyslipidaemia for the Prevention of Stroke: Clinical Practice Recommendations from the Lipid Association of India, Current Vascular Pharmacology, 2022, 20, 134-155.

LIVER FUNCTION PROFILE, SERUM

| BILIRUBIN, TOTAL METHOD: DIAZONIUM ION, BLANKED (ROCHE) | 0.89 | UPTO 1.2 | mg/dL |
|---|-----------|---|-------|
| BILIRUBIN, DIRECT METHOD: DIAZOTIZATION | 0.21 | 0.00 - 0.30 | mg/dL |
| BILIRUBIN, INDIRECT METHOD: CALCULATED PARAMETER | 0.68 High | 0.00 - 0.60 | mg/dL |
| TOTAL PROTEIN METHOD: BIURET | 7.9 | 6.6 - 8.7 | g/dL |
| ALBUMIN METHOD: BROMOCRESOL GREEN | 5.3 High | 3.97 - 4.94 | g/dL |
| GLOBULIN | 2.6 | 2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04 | g/dL |
| METHOD: CALCULATED PARAMETER | | | |
| ALBUMIN/GLOBULIN RATIO METHOD: CALCULATED PARAMETER | 2.0 | 1.0 - 2.0 | RATIO |
| ASPARTATE AMINOTRANSFERASE(AST/SGOT) | 22 | 0 - 40 | U/L |
| ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD: UV WITHOUT PYRIDOXAL-5 PHOSPHATE | 38 | 0 - 41 | U/L |
| ALKALINE PHOSPHATASE METHOD: PNPP - AMP BUFFER | 64 | 40 - 129 | U/L |
| GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD: GAMMA GLUTAMYLCARBOXY 4NITROANILIDE | 23 | 8 - 61 | U/L |
| LACTATE DEHYDROGENASE METHOD: LACTATE -PYRUVATE | 159 | 135 - 225 | U/L |
| BLOOD UREA NITROGEN (BUN), SERUM | | | |
| BLOOD UREA NITROGEN METHOD: UREASE - UV | 8 | 6 - 20 | mg/dL |
| CREATININE, SERUM | | | |
| CREATININE | 1.02 | 0.70 - 1.20 | mg/dL |



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CONSULTANT PATHOLOGIST





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CODE/NAME & ADDRESS: C000138383

ARCOFEMI HEALTHCARE LTD (MEDIWHEEL
F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHÍ

NEW DELHI 110030 8800465156 ACCESSION NO : **0080WL004375**

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO : AGE/SEX :47 Years

DRAWN :

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| | | _ | |
|----------------------------------|-------------|----------------------------|---------------|
| Test Report Status <u>Final</u> | Results | Biological Reference I | nterval Units |
| | | | |
| METHOD: ALKALINE PICRATE-KINETIC | | | |
| BUN/CREAT RATIO | | | |
| BUN/CREAT RATIO | 7.84 | 5.00 - 15.00 | |
| METHOD: CALCULATED PARAMETER | | | |
| URIC ACID, SERUM | | | |
| URIC ACID | 7.7 High | 3.4 - 7.0 | mg/dL |
| METHOD: URICASE, COLORIMETRIC | | | |
| TOTAL PROTEIN, SERUM | | | |
| TOTAL PROTEIN | 7.9 | 6.6 - 8.7 | g/dL |
| METHOD : BIURET | | | |
| ALBUMIN, SERUM | | | |
| ALBUMIN | 5.3 High | 3.97 - 4.94 | g/dL |
| METHOD: BROMOCRESOL GREEN | | | |
| GLOBULIN | | | |
| GLOBULIN | 2.6 | 2.0 - 4.0 | g/dL |
| | | Neonates - | |
| | | Pre Mature: 0.29 - 1.04 | |
| METHOD: CALCULATED PARAMETER | | 0.23 | |
| ELECTROLYTES (NA/K/CL), SE | RUM | | |
| SODIUM, SERUM | 140 | 136 - 145 | mmol/L |
| METHOD : ISE INDIRECT | | | |
| POTASSIUM, SERUM | 4.25 | 3.5 - 5.1 | mmol/L |
| METHOD: ISE INDIRECT | | | |
| CHLORIDE, SERUM | 104 | 98 - 107 | mmol/L |
| METHOD: ISE INDIRECT | | | |
| Interpretation(s) | | | |
| Sodium | Potassium C | hloride |] |



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View Details





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CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

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Test Report Status Results **Biological Reference Interval Final** Units

| Decreased in: CCF, cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (5SRI), antipsychotics. Increased in: Dehydration (excessivesweating, severe womiting or diarrhea), diabetes mellitus, diabetes mellitus, diabetesinsipidus, hyperaldosteronism, lacidosis, dehydration, renal failure, adiabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics. Increased in: Dehydration (excessivesweating, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics. Increased in: Dehydration (excessivesweating, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics. Increased in: Domyord sill, type religions, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diabetic ketoacidosis, diabetic ketoacidosis, dehetic ketoacidosis, denetic dehetic ketoacidosis, dehetic ketoacidosis, dehetic | | | |
|--|---------------------------------------|--|--|
| sweating, salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (SSRI), antipsychotics. Increased in: Dehydration (excessivesweating, severe womiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, Cushing's severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. RTA types I and II, hyperaldosteronism, Cushing's syndrome, swantic diuresis (e.g., hyperaldosteronism, Cushing's diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, Gushing's syndrome, swantic diuresis (e.g., diabetic, chronic respiratory acidosis, diabetic, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics. Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison's disease, RTA type IV, hyperaldosteronism, detrement with sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics. Increased in: Paral failure, Addison's disease, RTA type IV, hyperaldosteronism, delayer syndrome, swantical periodic paralysis, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, | Decreased In:CCF,cirrhosis, | Decreased in: Low potassium | |
| nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (SSRI), antipsychotics. Increased in: Dehydration (excessivesweating, severe vomitting or diarrhea), diabetes mater intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/t for each 100 mg/dL increase in blood glucose. Interferences interferences in blood glucose. Interferences interference | vomiting, diarrhea, excessive | intake,prolonged vomiting or diarrhea, | renal failure combined with salt |
| nephrotic syndrome, water intoxication, SIADH. Drugs: hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics. Increased in: Dehydration (excessiveseating, severe vomiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. syndrome, osmotic diuresis (e.g., hyperaldosic sq.g., hyperaldosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics. licorice, oral contraceptives. syndrome, osmotic diuresis (e.g., hyperaldisis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics. Increased in: Dehydration (excessiveseweating, severe lissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison's disease, RTA type IV, salt-losing nephropathy, porphyria, expension of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chromic laxative, corticosteroids, diuretics. Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, hemal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, potassium salts, potassium-sparing diuretics. Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, hyperadenocoticism. overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, potassium salts, potassium-sparing diuretics. Increased in: Renal failure, nephrotic saline, diarrhea (Loss of HCO3-), respiratory alkalosis, potassium salts, potassium-sparing diuretics. Increased in: Renal failure, nephrotic | sweating, salt-losing | RTA types I and II, | deprivation, over-treatment with |
| intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (SSRI), antipsychotics. Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sedium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics. Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, hyperaldosteronism, inadequate hyperkalemic familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics. Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, nephrotic syndrome, RTA,dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis. Drugs: chronic laxative, corticosteroids, diuretics. Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, novertreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis. Drugs: chronic laxative, corticosteroids, lovertreased in: Renal failure, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperalcosteronism, metabolic alkalosis. Intereased in: Massive hemolysis, soverticosteronism, metabolic alkalosis, hyperalcosteronism, deviced in: Renal failure, novertreatment with saline, hyperparathyroidism, diarrhea, lovertreatment with saline, hyp | nephropathy, adrenal insufficiency, | hyperaldosteronism, Cushing's | diuretics, chronic respiratory acidosis, |
| thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (55RI), antipsychotics. Increased in: Dehydration (excessives weating, severe vomiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics. periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics. Increased in: Massive hemolysis, acidosis. Drugs: hemolysis, acidosis, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. | nephrotic syndrome, water | syndrome,osmotic diuresis (e.g., | diabetic ketoacidosis, excessive |
| chlorpropamide, carbamazepine, anti depressants (SSRI), antipsychotics. Increased in: Dehydration (excessivesweating, severe water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. (transient). Drugs: Adrenergic agents, diuretics. (extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics. Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison' s disease, RTA type IV, hyperaldosteronism, inadequate paralysis. Drugs: potassium salts, potassium salts, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. (transient). Drugs: Adrenergic agents, diuretics. Increased in: Massive hemolysis, severe laxative, corticosteroids, diuretics. Increased in: Massive hemolysis, syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | intoxication, SIADH. Drugs: | hyperglycemia), alkalosis, familial | sweating, SIADH, salt-losing |
| diuretics. diuretics. diuretics. diuretics. diuretics. adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chromic laxative, corticosteroids, diuretics. Increased in: Dehydration (excessivesweating, severe severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sedium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. diuretics. Increased in: Massive hemolysis, severe laxative, corticosteronism, diavetics. Increased in: Massive hemolysis, severe laxative, corticosteronism, diavetics. Increased in: Massive hemolysis, severe laxative, corticosteronism, diavetics. Increased in: Massive hemolysis, severe lipemolysis, severe tissue damage, rhabdomyolysis, syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperparathyroidism, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole. Interferences: Severe lipemia or hyperparatences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | thiazides, diuretics, ACE inhibitors, | periodic paralysis,trauma | nephropathy, porphyria, expansion of |
| Increased in: Dehydration (excessivesweating, severe womiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sedium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq./L for each 100 mg/dL increase in blood glucose. Increased in: Massive hemolysis, lavasive, hemolysis, lavabetes insipidus, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperparathyroidism, beta-blockers, ACE inhibitors, high- delayed separation of serum, prolonged first clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium hyperpaldosteronism, metabolic laxative, corticosteroids, diuretics. Increased in: Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, balacies, hyperparathyroidism, balacies, hyperparathyroidism, beta-blockers, ACE inhibitors, hyperparathyroidism, overtreatment with saline, hyperparathyroidism, balacies, hyperparathyroidism, balacies, syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, balacies, syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, beta-blockers, ACE inhibitors, high- diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism, high- diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, alkalosis, hyperadrenoc | chlorpropamide,carbamazepine,anti | (transient). Drugs: Adrenergic agents, | extracellular fluid volume, |
| Increased in: Dehydration (excessivesweating, severe somiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Intereased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, syndrome, RTA, dehydration, overtreatment with syndrome, RTA, dehydration, overtreatment with syndrome, acidosis from diabetes syndrome, RTA, dehydration, overtreatment with syndrome, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | depressants (SSRI), antipsychotics. | diuretics. | adrenalinsufficiency, |
| Increased in: Dehydration (excessivesweating, severe severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, hyperaldosteronism, inadequate water intake. Drugs: steroids, paralysis. Drugs: potassium salts, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Increased in: Massive hemolysis, severe damage, rhabdomyolysis, acidosis, dehydration, renal failure, syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | | | hyperaldosteronism, metabolic |
| Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Interferences is severe lipemia or hyperproteinement, if serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Interferences is severe lipemia or hyperproteinement, if serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Interferences is severe lipemia or hyperproteinement, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Interferences is severe tissue damage, rhabdomyolysis, severe tissue damage, rhabdomyolysis, schehydration, renal failure, spurdome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrencocriticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Test is helpful in assessing normal and increased anion gap metabolic acidosis and in diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrencocriticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Test is helpful in assessing normal and increased anion gap metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrencocriticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, and | | | alkalosis. Drugs: chronic |
| (excessivesweating, severe vomiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sedium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison' s disease, RTA type IV, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Severe lipemia or hyperproteinemi, if sedium analysis involves a dilution step can cause spurious results. The serum sodium drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | | | laxative,corticosteroids, diuretics. |
| womiting or diarrhea), diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sedium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. acidosis, dehydration, renal failure, Addison's disease, RTA type IV, hyperaldic insipidus, metabolic acidosis from diarrhea (Loss of HCOa-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | Increased in: Dehydration | Increased in: Massive hemolysis, | Increased in: Renal failure, nephrotic |
| mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Addison's disease, RTA type IV, hyperaldose, Saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Addison's disease, RTA type IV, hyperadice, Insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. Interferences: Hemolysis of sample, delayed separation of serum, assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum characterism). | (excessivesweating, severe | severe tissue damage, rhabdomyolysis, | syndrome, RTA, dehydration, |
| hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium sa | vomiting or diarrhea), diabetes | acidosis, dehydration, renal failure, | overtreatment with |
| water intake. Drugs: steroids, licorice, oral contraceptives. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium drawing, and prolonged tourniquet may falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. paralysis. Drugs: potassium salts, po | mellitus, diabetesinsipidus, | Addison' s disease, RTA type IV, | saline,hyperparathyroidism, diabetes |
| licorice, oral contraceptives. potassium- sparing diuretics, NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium drawing, and prolonged tourniquet placement. Very high WBC/PLT counts mg/dL increase in blood glucose. potassium- sparing diuretics, NSAIDs, beta-blockers, ACE inhibitors, high-dose inhibitors, high-dose trimethoprim-sulfamethoxazole. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis of sample, delayed separation of serum, prolonged fist clenching during blood gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy | hyperaldosteronism, inadequate | hyperkalemic familial periodic | insipidus, metabolic acidosis from |
| beta-blockers, ACE inhibitors, high- dose trimethoprim-sulfamethoxazole. Interferences: Severe lipemia or hyperproteinemi, if sedium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium Drugs: acetazolamide,androgens, hydrochlorothiazide,salicylates. Interferences: Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy | water intake. Drugs: steroids, | paralysis. Drugs: potassium salts, | diarrhea (Loss of HCO3-), respiratory |
| dose trimethoprim-sulfamethoxazole. hydrochlorothiazide, salicylates. Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. dose trimethoprim-sulfamethoxazole. hydrochlorothiazide, salicylates. Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy | licorice, oral contraceptives. | potassium- sparing diuretics, NSAIDs, | alkalosis, hyperadrenocorticism. |
| Interferences: Severe lipemia or hyperproteinemi, if sodium analysis delayed separation of serum, involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | | beta-blockers, ACE inhibitors, high- | Drugs: acetazolamide, androgens, |
| hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | | dose trimethoprim-sulfamethoxazole. | hydrochlorothiazide,salicylates. |
| involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose. prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium chloride) from that due to malignancy | Interferences: Severe lipemia or | Interferences: Hemolysis of sample, | Interferences:Test is helpful in |
| spurious results. The serum sodium falls about 1.6 mEq/L for each 100 placement. Very high WBC/PLT counts mg/dL increase in blood glucose. may cause spurious. Plasma potassium chloride) from that due to malignancy | hyperproteinemi, if sodium analysis | delayed separation of serum, | assessing normal and increased anion |
| falls about 1.6 mEq/L for each 100 placement. Very high WBC/PLT counts hyperparathyroidism (high serum chloride) from that due to malignancy | involves a dilution step can cause | prolonged fist clenching during blood | gap metabolic acidosis and in |
| mg/dL increase in blood glucose. | spurious results. The serum sodium | drawing, and prolonged tourniquet | distinguishing hypercalcemia due to |
| | falls about 1.6 mEq/L for each 100 | placement. Very high WBC/PLT counts | hyperparathyroidism (high serum |
| levels are normal. (Normal serum chloride) | mg/dL increase in blood glucose. | may cause spurious. Plasma potassium | chloride) from that due to malignancy |
| | | levels are normal. | (Normal serum chloride) |

GLUCOSE FASTING, FLUORIDE PLASMA-TEST DESCRIPTION

Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and sothat no glucose is excreted in the

Increased in:Diabetes mellitus, Cushing's syndrome (10 – 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.

Decreased in:Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency,hypopituitarism,diffuse liver disease,

malignancy(adrenocortical,stomach,fibrosarcoma),infant of a diabetic mother,enzyme deficiency diseases(e.g.galactosemia),Drugs-insulin,ethanol,propranolol

sulfonylureas,tolbutamide,and other oral hypoglycemic agents.

NOTE: While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values),there is wide fluctuation within

individuals.Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.

High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.

GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin

treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc. Additional test HbA1c LIVER FUNCTION PROFILE, SERUM-

Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. **Elevated levels** results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors &Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.

AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver, liver cancer, kidney failure, hemolytic anemia, pancreatitis, hemochromatosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. ALT is found mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis, sometimes due to a viral infection, is chemia to the liver, chronic



LAB HEAD

Dr. Praniali Vasisht

Chardii Jary

DR.CHANDNI GARG CONSULTANT PATHOLOGIST Page 10 Of 16





Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India Tel: 9111591115, Fax:







CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030

8800465156

ACCESSION NO : 0080WL004375

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO

DRAWN

AGE/SEX

RECEIVED: 14/12/2023 08:56:43

:47 Years

REPORTED :15/12/2023 17:11:06

Test Report Status Results **Biological Reference Interval Final** Units

hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Pagets disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilsons disease.

GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, biliary system and pancreas. Conditions that increase serum GGT are obstructive liver disease, high alcohol consumption and use of enzyme-inducing drugs etc.

Total Protein also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin. Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstroms disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic

syndrome, Protein-losing enteropathy etc. **Albumin** is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc

BLOOD UREA NITROGEN (BUN), SERUM-Causes of Increased levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism) Causes of decreased level include Liver disease, SIADH.

CREATININE, SERUM-Higher than normal level may be due to: Blockage in the urinary tract, Kidney problems, such as kidney damage or failure, infection, or reduced blood flow, Loss of body fluid (dehydration), Muscle problems, such as breakdown of muscle fibers, Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to: Myasthenia Gravis, Muscuophy

URIC ACID, SERUM-Causes of Increased levels: Dietary(High Protein Intake, Prolonged Fasting, Rapid weight loss), Gout, Lesch nyhan syndrome, Type 2 DM, Metabolic

syndrome Causes of decreased levels-Low Zinc intake, OCP, Multiple Sclerosis

TOTAL PROTEIN, SERUM-is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin. **Higher-than-normal levels may be due to:** Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstroms disease Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

ALBUMIN, SERUM-Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc.

Dr. Praniali Vasisht

LAB HEAD

DR.CHANDNI GARG

CONSULTANT PATHOLOGIST

Chardni Jary

Page 11 Of 16



Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India Tel: 9111591115, Fax:







Male

PATIENT NAME: DR SRIVASTAVA ASHISH MOHAN REF. DOCTOR: SELF

CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030

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CLINICAL PATH - URINALYSIS

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

PHYSICAL EXAMINATION, URINE

COLOR PALE YELLOW

APPEARANCE CLEAR

CHEMICAL EXAMINATION, URINE

PH 7.5 4.7 - 7.5

METHOD: REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD

SPECIFIC GRAVITY 1.003 - 1.035

METHOD: REFLECTANCE SPECTROPHOTOMETRY (PKA CHANGE OF PRETREATED POLY ELECTROLYTES)

NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY (PROTEIN-ERROR-OF-INDICATORS PRINCIPLE)

NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY(GLUCOSE OXIDAE/PEROXIDASE METHOD)

NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY (SODIUM NITROPRUSSIDE REACTION)

BLOOD NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY (PEROXIDASE METHOD)

BILIRUBIN NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY (DIAZO REACTION)

UROBILINOGEN NORMAL **NORMAL**

METHOD: REFLECTANCE SPECTROPHOTOMETRY - EHRLICH REACTION

NITRITE NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY, CONVERSION OF NITRATE TO NITRITE

NOT DETECTED LEUKOCYTE ESTERASE NOT DETECTED

MICROSCOPIC EXAMINATION, URINE

/HPF NOT DETECTED NOT DETECTED RED BLOOD CELLS

METHOD: MICROSCOPIC EXAMINATION

PUS CELL (WBC'S) /HPF 1-2 0-5

METHOD: MICROSCOPIC EXAMINATION

/HPF 0-5 EPITHELIAL CELLS 0 - 1

METHOD: MICROSCOPIC EXAMINATION

CASTS NOT DETECTED **CRYSTALS** NOT DETECTED



Dr. Pranjali Vasisht

LAB HEAD

DR.CHANDNI GARG

Page 12 Of 16

CONSULTANT PATHOLOGIST













Male

PATIENT NAME: DR SRIVASTAVA ASHISH MOHAN REF. DOCTOR: SELF

CODE/NAME & ADDRESS : C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

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NEW DELHI 110030 8800465156

ACCESSION NO: 0080WL004375

PATIENT ID : DRSRM30087680

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Test Report Status Results Biological Reference Interval Units <u>Final</u>

METHOD: MICROSCOPIC EXAMINATION

NOT DETECTED NOT DETECTED **BACTERIA**

METHOD: MICROSCOPIC EXAMINATION

YEAST NOT DETECTED NOT DETECTED

Interpretation(s)

The following table describes the probable conditions, in which the analytes are present in urine

| Presence of | Conditions |
|-------------------------|---|
| Proteins | Inflammation or immune illnesses |
| Pus (White Blood Cells) | Urinary tract infection, urinary tract or kidney stone, tumors or any kind |
| | of kidney impairment |
| Glucose | Diabetes or kidney disease |
| Ketones | Diabetic ketoacidosis (DKA), starvation or thirst |
| Urobilinogen | Liver disease such as hepatitis or cirrhosis |
| Blood | Renal or genital disorders/trauma |
| Bilirubin | Liver disease |
| Erythrocytes | Urological diseases (e.g. kidney and bladder cancer, urolithiasis), urinary |
| - | tract infection and glomerular diseases |
| Leukocytes | Urinary tract infection, glomerulonephritis, interstitial nephritis either |
| - | acute or chronic, polycystic kidney disease, urolithiasis, contamination by |
| | genital secretions |
| Epithelial cells | Urolithiasis, bladder carcinoma or hydronephrosis, ureteric stents or |
| | bladder catheters for prolonged periods of time |
| | |
| Granular Casts | Low intratubular pH, high urine osmolality and sodium concentration, |
| | interaction with Bence-Jones protein |
| Hyaline casts | Physical stress, fever, dehydration, acute congestive heart failure, renal |
| | diseases |
| Calcium oxalate | Metabolic stone disease, primary or secondary hyperoxaluria, intravenous |
| | infusion of large doses of vitamin C, the use of vasodilator naftidrofuryl |
| | oxalate or the gastrointestinal lipase inhibitor orlistat, ingestion of |
| | ethylene glycol or of star fruit (Averrhoa carambola) or its juice |
| Uric acid | arthritis |
| Bacteria | Urinary infectionwhen present in significant numbers & with pus cells. |
| Trichomonas vaginalis | Vaginitis, cervicitis or salpingitis |

Remember

Dr.Pranjali Vasisht

LAB HEAD

Chardni gary

DR.CHANDNI GARG CONSULTANT PATHOLOGIST Page 13 Of 16





Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India Tel: 9111591115, Fax:







CODE/NAME & ADDRESS: C000138383

ARCOFEMI HEALTHCARE LTD (MEDIWHEEL
F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030 8800465156 ACCESSION NO: 0080WL004375

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO : AGE/SEX :47 Years Male

DRAWN :

RECEIVED : 14/12/2023 08:56:43 REPORTED :15/12/2023 17:11:06

Test Report Status Final Results Biological Reference Interval Units

CLINICAL PATH - STOOL ANALYSIS

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

PHYSICAL EXAMINATION, STOOL

CONSISTENCY

SAMPLE NOT RECEIVED

Dr. Nidhi Garg Lab Consultant

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View Details

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SPECIALISED CHEMISTRY - HORMONE

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

THYROID PANEL, SERUM

80.00 - 200.00 ng/dL T3 116.60 METHOD: COMPETITIVE (ECLIA)

7.80 5.10 - 14.10 **T4** μg/dL

METHOD: COMPETITIVE (ECLIA)

0.270 - 4.200TSH (ULTRASENSITIVE) 2.440 μIU/mL

METHOD: SANDWICH (ECLIA) Interpretation(s)

Triiodothyronine T3, Thyroxine T4, and Thyroid Stimulating Hormone TSH are thyroid hormones which affect almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate.

Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.

Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hyperthyroidism, TSH levels are low, Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3.Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hyperthyroidism, and measurement of TT4 is more useful in the diagnosis of hypothyroidism. Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active. It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.

| Sr. No. | TSH | Total T4 | FT4 | Total T3 | Possible Conditions |
|---------|------------|----------|--------|----------|--|
| 1 | High | Low | Low | Low | (1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3) |
| | | | | | Post Thyroidectomy (4) Post Radio-Iodine treatment |
| 2 | High | Normal | Normal | Normal | (1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid |
| | | | | | hormone replacement therapy (3) In cases of Autoimmune/Hashimoto |
| | | | | | thyroiditis (4). Isolated increase in TSH levels can be due to Subclinical |
| | | | | | inflammation, drugs like amphetamines, Iodine containing drug and |
| | | | | | dopamine antagonist e.g. domperidone and other physiological reasons. |
| 3 | Normal/Low | Low | Low | Low | (1) Secondary and Tertiary Hypothyroidism |
| 4 | Low | High | High | High | (1) Primary Hyperthyroidism (Graves Disease) (2) Multinodular Goitre |
| | | | | | (3)Toxic Nodular Goitre (4) Thyroiditis (5) Over treatment of thyroid |
| | | | | | hormone (6) Drug effect e.g. Glucocorticoids, dopamine, T4 |
| | | | | | replacement therapy (7) First trimester of Pregnancy |
| 5 | Low | Normal | Normal | Normal | (1) Subclinical Hyperthyroidism |
| 6 | High | High | High | High | (1) TSH secreting pituitary adenoma (2) TRH secreting tumor |
| 7 | Low | Low | Low | Low | (1) Central Hypothyroidism (2) Euthyroid sick syndrome (3) Recent |
| | | | | | treatment for Hyperthyroidism |

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REF. DOCTOR: SELF **PATIENT NAME: DR SRIVASTAVA ASHISH MOHAN**

CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

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NEW DELHI 110030 8800465156

ACCESSION NO: 0080WL004375

PATIENT ID : DRSRM30087680

CLIENT PATIENT ID: ABHA NO

AGE/SEX DRAWN

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Test Report Status Results Biological Reference Interval **Final** Units

| 8 | Normal/Low | Normal | Normal | High | (1) T3 thyrotoxicosis (2) Non-Thyroidal illness |
|---|------------|--------|--------|--------|--|
| 9 | Low | High | High | Normal | (1) T4 Ingestion (2) Thyroiditis (3) Interfering Anti TPO antibodies |

REF: 1. TIETZ Fundamentals of Clinical chemistry 2.Guidlines of the American Thyroid association during pregnancy and Postpartum, 2011. NOTE: It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.TSH is not affected by variation in thyroid - binding protein. TSH has a diurnal rhythm, with peaks at 2:00 - 4:00 a.m. And troughs at 5:00 - 6:00 p.m. With ultradian variations.

> **End Of Report** Please visit www.agilusdiagnostics.com for related Test Information for this accession

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- 2. All tests are performed and reported as per the turnaround time stated in the AGILUS Directory of Services.
- 3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.
- 4. A requested test might not be performed if:
 - i. Specimen received is insufficient or inappropriate
 - ii. Specimen quality is unsatisfactory
 - iii. Incorrect specimen type
 - iv. Discrepancy between identification on specimen container label and test requisition form

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Agilus Diagnostics Ltd

Fortis Hospital, Sector 62, Phase VIII, Mohali 160062

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