

PATIENT NAME : MAMTA BIST

REF. DOCTOR : SELF

B 1205 Vasundhara chs ltd building no 6 shastri
nagar siddharth hospital road
400104

ACCESSION NO : **0002WK032382**
PATIENT ID : MAMTF31128527
CLIENT PATIENT ID:
ABHA NO :

AGE/SEX : 37 Years Female
DRAWN : 25/11/2023 08:48:53
RECEIVED : 25/11/2023 08:50:50
REPORTED : 27/11/2023 14:43:05

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|--------------------|-------|---------|-------------------------------|-------|

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

XRAY-CHEST

IMPRESSION NO SIGNIFICANT PLEUROPARENCHYMAL ABNORMALITY DETECTED

ECG

ECG WITHIN NORMAL LIMITS

MEDICAL HISTORY

RELEVANT PRESENT HISTORY HYPERTENSION SINCE 1 1/2 YRS.
HEEL PAIN ON AND OFF.
COLD AND COUGH SINCE 1 WEEK MEDICATION TAKEN.

RELEVANT PAST HISTORY PULMONARY TB 10 YRS BACK TREATED FULLY

RELEVANT PERSONAL HISTORY NOT SIGNIFICANT

MENSTRUAL HISTORY (FOR FEMALES) REGULAR

LMP (FOR FEMALES) 13/11/2023

RELEVANT FAMILY HISTORY HYPERTESION / DIABETES

HISTORY OF MEDICATIONS ANTI HYPERTENSTION.
AYURVEDIC FOR HEEL PAIN

ANTHROPOMETRIC DATA & BMI

| | | |
|------------------|------|----------|
| HEIGHT IN METERS | 1.65 | mts |
| WEIGHT IN KGS. | 81.7 | Kgs |
| BMI | 30 | kg/sqmts |

BMI & Weight Status as follows
Below 18.5: Underweight
18.5 - 24.9: Normal
25.0 - 29.9: Overweight
30.0 and Above: Obese

GENERAL EXAMINATION

Dr. J N Shukla ,MBBS, AFIH
Consultant Physician

Dr. Swati Karmarkar,
MD,DNB,DMRD
Consultant Radiologist

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Agilus Diagnostics Ltd
Prime Square Building,Plot No 1,Gaiwadi Industrial Estate,S.V. Road,Goregaon (W)
Mumbai, 400062
Maharashtra, India
Tel : 9111591115, 022 - 67801212
CIN - U74899PB1995PLC045956



Patient Ref. No. 2000012092810

PATIENT NAME : MAMTA BIST

REF. DOCTOR : SELF

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| | | | |
|---|---|--|--|
| MENTAL / EMOTIONAL STATE | NORMAL | | |
| GENERAL APPEARANCE / NUTRITIONAL STATUS | HEALTHY | | |
| BUILT / SKELETAL FRAMEWORK | AVERAGE | | |
| FACIAL APPEARANCE | NORMAL | | |
| SKIN | PALE WITH DRYNESS | | |
| UPPER LIMB | NORMAL | | |
| LOWER LIMB | NORMAL | | |
| NECK LYMPHATICS / SALIVARY GLANDS | NOT ENLARGED OR TENDER | | |
| THYROID GLAND | NOT ENLARGED | | |
| CAROTID PULSATION | NORMAL | | |
| TEMPERATURE | NORMAL | | |
| PULSE | 72/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID BRUIT | | |
| RESPIRATORY RATE | NORMAL | | |

CARDIOVASCULAR SYSTEM

| | | |
|--------------|-----------------------|-------|
| BP | 120/80 MM HG (SUPINE) | mm/Hg |
| APEX BEAT | NORMAL | |
| HEART SOUNDS | NORMAL | |
| MURMURS | ABSENT | |

RESPIRATORY SYSTEM

| | |
|-------------------------|--------------------|
| SIZE AND SHAPE OF CHEST | NORMAL |
| MOVEMENTS OF CHEST | SYMMETRICAL |
| BREATH SOUNDS QUALITY | VESICULAR (NORMAL) |
| ADDED SOUNDS | ABSENT |

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PER ABDOMEN

| | |
|------------|--------------|
| APPEARANCE | NORMAL |
| LIVER | NOT PALPABLE |
| SPLEEN | NOT PALPABLE |
| HERNIA | NORMAL |

CENTRAL NERVOUS SYSTEM

| | |
|----------------------|--------|
| HIGHER FUNCTIONS | NORMAL |
| CRANIAL NERVES | NORMAL |
| CEREBELLAR FUNCTIONS | NORMAL |
| SENSORY SYSTEM | NORMAL |
| MOTOR SYSTEM | NORMAL |
| REFLEXES | NORMAL |

MUSCULOSKELETAL SYSTEM

| | |
|--------|--------|
| SPINE | NORMAL |
| JOINTS | NORMAL |

BASIC EYE EXAMINATION

| | |
|--|---------------------------|
| CONJUNCTIVA | NORMAL |
| EYELIDS | NORMAL |
| EYE MOVEMENTS | NORMAL |
| CORNEA | NORMAL |
| DISTANT VISION RIGHT EYE WITHOUT GLASSES | WITHIN NORMAL LIMIT (6/6) |
| DISTANT VISION LEFT EYE WITHOUT GLASSES | WITHIN NORMAL LIMIT (6/6) |
| NEAR VISION RIGHT EYE WITHOUT GLASSES | WITHIN NORMAL LIMIT (N6) |
| NEAR VISION LEFT EYE WITHOUT GLASSES | WITHIN NORMAL LIMIT (N6) |



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COLOUR VISION NORMAL (17/17)

BASIC ENT EXAMINATION

| | |
|--------------------|-------------------------|
| EXTERNAL EAR CANAL | NORMAL |
| TYMPANIC MEMBRANE | NORMAL |
| NOSE | NO ABNORMALITY DETECTED |
| SINUSES | MILD CONGESTION |
| THROAT | NO ABNORMALITY DETECTED |
| TONSILS | NOT ENLARGED |

SUMMARY

| | |
|------------------------------------|---|
| RELEVANT HISTORY | NOT SIGNIFICANT |
| RELEVANT GP EXAMINATION FINDINGS | NOT SIGNIFICANT |
| RELEVANT LAB INVESTIGATIONS | LOW HEMOGLOBIN (11.5) RAISED EOSINOPHILS (10) RAISED CHOLESTEROL (229) RAISED LDL CHOLESTEROL (156) |
| RELEVANT NON PATHOLOGY DIAGNOSTICS | USG- EARLY HEPATOSTEATOSIS |
| REMARKS / RECOMMENDATIONS | LOW HEMOGLOBIN , RAISED EOISNOPHILS, ALTRED BLOOD LIPID, STOOL - OCCULT BLOOD TRACE ADV- MONITOR BLOOD PRESSURE ADV- REDUCE SATURATED FAT IN FOOD ADV- FIBER RICH DIET ADV- VITAMIN D TEST FOLLOW UP WITH PHYSICIAN FOR - RAISED LIPID PROFILE |

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MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

EARLY HEPATOSTEATOSIS.

TMT OR ECHO

CLINICAL PROFILE

2 DECHO DONE : IMPRESSION.

-GOOD LV SYSTOLIC FUNCTION AT REST. NO RWMA

-LVEF 55-60%.

-ALL VALVES STRUCTURALLY NORMAL.

-NO EVIDENCE OF PE/CLOT/VEGETATION

Interpretation(s)

MEDICAL

HISTORY-*****:
THIS REPORT CARRIES THE SIGNATURE OF OUR LABORATORY DIRECTOR. THIS IS AN INVIOABLE FEATURE OF OUR LAB MANAGEMENT SOFTWARE. HOWEVER, ALL
EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.

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MC-5718

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HAEMATOLOGY - CBC**MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE****BLOOD COUNTS, EDTA WHOLE BLOOD**

| | | | |
|--|-----------------|-------------|---------------|
| HEMOGLOBIN (HB) METHOD : CYANIDE FREE DETERMINATION | 11.5 Low | 12.0 - 15.0 | g/dL |
| RED BLOOD CELL (RBC) COUNT METHOD : FLUORESCENCE FLOW CYTOMETRY | 4.41 | 3.8 - 4.8 | mil/ μ L |
| WHITE BLOOD CELL (WBC) COUNT METHOD : ELECTRICAL IMPEDANCE | 6.61 | 4.0 - 10.0 | thou/ μ L |
| PLATELET COUNT METHOD : ELECTRONIC IMPEDENCE & MICROSCOPY | 220 | 150 - 410 | thou/ μ L |

RBC AND PLATELET INDICES

| | | | |
|--|------------------|--------------|------|
| HEMATOCRIT (PCV) METHOD : CALCULATED PARAMETER | 36.3 | 36 - 46 | % |
| MEAN CORPUSCULAR VOLUME (MCV) METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM | 82.3 Low | 83.0 - 101.0 | fL |
| MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER | 26.2 Low | 27.0 - 32.0 | pg |
| MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER | 31.8 | 31.5 - 34.5 | g/dL |
| RED CELL DISTRIBUTION WIDTH (RDW) METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM | 13.8 | 11.6 - 14.0 | % |
| MENTZER INDEX | 18.7 | | |
| MEAN PLATELET VOLUME (MPV) METHOD : DERIVED PARAMETER FROM PLATELET HISTOGRAM | 14.4 High | 6.8 - 10.9 | fL |

WBC DIFFERENTIAL COUNT

| | | | |
|---|----|---------|---|
| NEUTROPHILS METHOD : FLUORESCENCE FLOW CYTOMETRY | 54 | 40 - 80 | % |
| LYMPHOCYTES METHOD : FLUORESCENCE FLOW CYTOMETRY | 29 | 20 - 40 | % |
| MONOCYTES | 7 | 2 - 10 | % |

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Consultant Pathologist

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| METHOD : FLUORESCENCE FLOW CYTOMETRY | | | | |
| EOSINOPHILS | | 10 High | 1 - 6 | % |
| METHOD : FLUORESCENCE FLOW CYTOMETRY | | | | |
| BASOPHILS | | 0 | 0 - 1 | % |
| METHOD : FLUORESCENCE FLOW CYTOMETRY | | | | |
| ABSOLUTE NEUTROPHIL COUNT | | 3.57 | 2.0 - 7.0 | thou/ μ L |
| METHOD : CALCULATED PARAMETER | | | | |
| ABSOLUTE LYMPHOCYTE COUNT | | 1.92 | 1.0 - 3.0 | thou/ μ L |
| METHOD : CALCULATED PARAMETER | | | | |
| ABSOLUTE MONOCYTE COUNT | | 0.46 | 0.2 - 1.0 | thou/ μ L |
| METHOD : CALCULATED PARAMETER | | | | |
| ABSOLUTE EOSINOPHIL COUNT | | 0.66 High | 0.02 - 0.50 | thou/ μ L |
| METHOD : CALCULATED PARAMETER | | | | |
| ABSOLUTE BASOPHIL COUNT | | 0 Low | 0.02 - 0.10 | thou/ μ L |
| METHOD : CALCULATED PARAMETER | | | | |
| NEUTROPHIL LYMPHOCYTE RATIO (NLR) | | 1.8 | | |
| METHOD : CALCULATED | | | | |

MORPHOLOGY

RBC PREDOMINANTLY NORMOCYTIC NORMOCHROMIC
WBC EOSINOPHILIA PRESENT
PLATELETS ADEQUATE

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 to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.

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.

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Consultant Pathologist

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HAEMATOLOGY**MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE****ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD**

| | | | |
|-------|----|-----------|------------|
| E.S.R | 12 | = or < 12 | mm at 1 hr |
|-------|----|-----------|------------|

METHOD : MODIFIED WESTERGREN METHOD BY AUTOMATED ANALYSER

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

| | | |
|-------|-----|--|
| HBA1C | 5.4 | Non-diabetic Adult < 5.7 % Pre-diabetes 5.7 - 6.4 Diabetes diagnosis: > or = 6.5 Therapeutic goals: < 7.0 Action suggested : > 8.0 (ADA Guideline 2021) |
|-------|-----|--|

METHOD : ION- EXCHANGE HPLC

| | | | |
|--------------------------------|-------|-------|-------|
| ESTIMATED AVERAGE GLUCOSE(EAG) | 108.3 | < 116 | 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, Vasculitides, 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: Polycythemia 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:

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Consultant Pathologist

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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.

1. eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
2. eAG gives an evaluation of blood glucose levels for the last couple of months.
3. 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 addition 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

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Test Report Status Final**Results****Biological Reference Interval** **Units****IMMUNOHAEMATOLOGY****MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE****ABO GROUP & RH TYPE, EDTA WHOLE BLOOD**

ABO GROUP

A

METHOD : HAEMAGGLUTINATION (AUTOMATED)

RH TYPE

POSITIVE

METHOD : HAEMAGGLUTINATION (AUTOMATED)

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.

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Tel : 9111591115, 022 - 67801212
CIN - U74899PB1995PLC045956

**Patient Ref. No. 2000012092810**



MC-5718

PATIENT NAME : MAMTA BIST**REF. DOCTOR : SELF**B 1205 Vasundhara chs ltd building no 6 shastri
nagar siddharth hospital road
400104ACCESSION NO : **0002WK032382**
PATIENT ID : MAMTF31128527
CLIENT PATIENT ID:
ABHA NO :AGE/SEX : 37 Years Female
DRAWN : 25/11/2023 08:48:53
RECEIVED : 25/11/2023 08:50:50
REPORTED : 27/11/2023 14:43:05

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BIOCHEMISTRY**MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE****GLUCOSE FASTING,FLUORIDE PLASMA**

| | | |
|---------------------------|----|--|
| FBS (FASTING BLOOD SUGAR) | 91 | Normal <100 mg/dL Impaired fasting glucose:100 to 125 Diabetes mellitus: > = 126 (on more than 1 occassion) (ADA guidelines 2021) |
|---------------------------|----|--|

METHOD : SPECTROPHOTOMETRY HEXOKINASE

GLUCOSE, POST-PRANDIAL, PLASMA

| | | |
|---------------------------------|----|--|
| PPBS(POST PRANDIAL BLOOD SUGAR) | 83 | Normal <140 mg/dL Impaired glucose tolerance:140 to 199 Diabetes mellitus : > = 200 (on more than 1 occassion) ADA guideline 2021 |
|---------------------------------|----|--|

METHOD : SPECTROPHOTOMETRY HEXOKINASE

LIPID PROFILE WITH CALCULATED LDL

| | | |
|--------------------|-----------------|---|
| CHOLESTEROL, TOTAL | 229 High | Desirable : < 200 mg/dL Borderline : 200 - 239 High : > / = 240 |
|--------------------|-----------------|---|

METHOD : SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC - CHOLETSEROL OXIDASE, ESTERASE, PEROXIDASE

| | | |
|---------------|-----|--|
| TRIGLYCERIDES | 134 | Normal: < 150 mg/dL Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500 |
|---------------|-----|--|

METHOD : SPECTROPHOTOMETRY, ENZYMATIC ENDPOINT WITH GLYCEROL BLANK

| | | |
|-----------------|----|---|
| HDL CHOLESTEROL | 46 | At Risk: < 40 mg/dL Desirable: > or = 60 |
|-----------------|----|---|

METHOD : SPECTROPHOTOMETRY, HOMOGENEOUS DIRECT ENZYMATIC COLORIMETRIC

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Chief Of Lab - Mumbai Reference Lab**Dr. Apeksha Sharma**
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| CHOLESTEROL LDL | | 156 High | Optimal : < 100 Near optimal/above optimal : 100-129 Borderline high : 130-159 High : 160-189 Very high : = 190 | mg/dL |
| METHOD : CALCULATED PARAMETER | | | | |
| NON HDL CHOLESTEROL | | 183 High | Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220 | mg/dL |
| METHOD : CALCULATED PARAMETER | | | | |
| VERY LOW DENSITY LIPOPROTEIN | | 27.0 | < or = 30.0 | mg/dL |
| METHOD : CALCULATED PARAMETER | | | | |
| CHOL/HDL RATIO | | 5.0 High | Low Risk : 3.3 - 4.4 Average Risk : 4.5 - 7.0 Moderate Risk : 7.1 - 11.0 High Risk : > 11.0 | |
| METHOD : CALCULATED PARAMETER | | | | |
| LDL/HDL RATIO | | 3.6 High | Desirable/Low Risk : 0.5 - 3.0 Borderline/Moderate Risk : 3.1 - 6.0 High Risk : > 6.0 | |
| 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 B. CAD with > 1 feature of Very high risk group or recurrent ACS (within 1 year) despite LDL-C < or = 50 mg/dl or polyvascular disease |
| Very High Risk | 1. Established ASCVD 2. Diabetes with 2 major risk factors or evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia |
| 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 |

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| | |
|--|---|
| Moderate Risk | 2 major ASCVD risk factors |
| Low Risk | 0-1 major ASCVD risk factors |
| Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors | |
| 1. Age > or = 45 years in males and > or = 55 years in females | 3. Current Cigarette smoking or tobacco use |
| 2. 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.

| 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 < OR = 30) | < 80 (Optional goal <OR = 60) | >OR = 50 | >OR = 80 |
| Extreme Risk Group Category B | <OR = 30 | <OR = 60 | > 30 | >60 |
| Very High Risk | <50 | <80 | >OR= 50 | >OR= 80 |
| High Risk | <70 | <100 | >OR= 70 | >OR= 100 |
| 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 | 0.23 | Upto 1.2 | mg/dL |
| METHOD : SPECTROPHOTOMETRY, COLORIMETRIC -DIAZO METHOD | | | |
| BILIRUBIN, DIRECT | 0.11 | < or = 0.3 | mg/dL |
| METHOD : SPECTROPHOTOMETRY, JENDRASSIK & GROFF - DIAZOTIZATION | | | |
| BILIRUBIN, INDIRECT | 0.12 | 0.0 - 0.9 | mg/dL |
| METHOD : CALCULATED PARAMETER | | | |
| TOTAL PROTEIN | 7.4 | 6.0 - 8.0 | g/dL |
| METHOD : SPECTROPHOTOMETRY, COLORIMETRIC -BIURET, REAGENT BLANK, SERUM BLANK | | | |
| ALBUMIN | 4.5 | 3.97 - 4.94 | g/dL |
| METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING | | | |
| GLOBULIN | 2.9 | 2.0 - 3.5 | g/dL |
| METHOD : CALCULATED PARAMETER | | | |
| ALBUMIN/GLOBULIN RATIO | 1.6 | 1.0 - 2.1 | RATIO |
| METHOD : CALCULATED PARAMETER | | | |
| ASPARTATE AMINOTRANSFERASE(AST/SGOT) | 14 | Upto 32 | U/L |
| METHOD : SPECTROPHOTOMETRY, WITHOUT PYRIDOXAL PHOSPHATE ACTIVATION(P5P) - IFCC | | | |
| ALANINE AMINOTRANSFERASE (ALT/SGPT) | 10 | Upto 33 | U/L |
| METHOD : SPECTROPHOTOMETRY, WITHOUT PYRIDOXAL PHOSPHATE ACTIVATION(P5P) - IFCC | | | |
| ALKALINE PHOSPHATASE | 65 | 35 - 104 | U/L |
| METHOD : SPECTROPHOTOMETRY, PNPP, AMP BUFFER - IFCC | | | |
| GAMMA GLUTAMYL TRANSFERASE (GGT) | 15 | < 40 | U/L |

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METHOD : SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC - G-GLUTAMYL-CARBOXY-NITROANILIDE - IFCC
LACTATE DEHYDROGENASE 130 < 223 U/L
 METHOD : SPECTROPHOTOMETRY, LACTATE TO PYRUVATE - UV-IFCC

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN 9 6 - 20 mg/dL
 METHOD : SPECTROPHOTOMETRY, UREASE -COLORIMETRIC

CREATININE, SERUM

CREATININE 0.83 0.60 - 1.10 mg/dL
 METHOD : SPECTROPHOTOMETRY, JAFFE'S ALKALINE PICRATE KINETIC - RATE BLANKED - IFCC-IDMS STANDARDIZED

BUN/CREAT RATIO

BUN/CREAT RATIO 11.30 8 - 15
 METHOD : CALCULATED PARAMETER

URIC ACID, SERUM

URIC ACID 5.4 2.4 - 5.7 mg/dL
 METHOD : SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC- URICASE

TOTAL PROTEIN, SERUM

TOTAL PROTEIN 7.4 6.0 - 8.0 g/dL
 METHOD : SPECTROPHOTOMETRY, COLORIMETRIC -BIURET, REAGENT BLANK, SERUM BLANK

ALBUMIN, SERUM

ALBUMIN 4.5 3.97 - 4.94 g/dL
 METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING

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GLOBULIN

| | | | |
|-------------------------------|-----|-----------|------|
| GLOBULIN | 2.9 | 2.0 - 3.5 | g/dL |
| METHOD : CALCULATED PARAMETER | | | |

ELECTROLYTES (NA/K/CL), SERUM

| | | | |
|-----------------------|------|-----------|--------|
| SODIUM, SERUM | 139 | 136 - 145 | mmol/L |
| METHOD : ISE INDIRECT | | | |
| POTASSIUM, SERUM | 4.30 | 3.5 - 5.1 | mmol/L |
| METHOD : ISE INDIRECT | | | |
| CHLORIDE, SERUM | 104 | 98 - 106 | mmol/L |
| METHOD : ISE INDIRECT | | | |

Interpretation(s)

| Sodium | Potassium | Chloride |
|---|---|--|
| 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 (SSRI), antipsychotics. | Decreased in: Low potassium intake, prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome, osmotic diuresis (e.g., hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics. | Decreased in: Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenal insufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics. |
| Increased in: Dehydration [excessive sweating, severe vomiting or diarrhea], diabetes mellitus, diabetes insipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives. | Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison's disease, RTA type IV, hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium-sparing diuretics, NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole. | Increased in: Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO ₃ ⁻), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates. |

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REPORTED : 27/11/2023 14:43:05**Test Report Status Final Results Biological Reference Interval Units****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:** 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 levels are normal.**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 (Normal serum chloride)**Interpretation(s)****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 so that no glucose is excreted in the urine.

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 Glycosuria, 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 Glycosuria, 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, ischemia to the liver, chronic 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.**Dr. Deepak Sanghavi, M.D (Path)**
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Test Report Status Final**Results****Biological Reference Interval** **Units****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. Deepak Sanghavi, M.D (Path)**
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CLINICAL PATH - URINALYSIS**MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE****PHYSICAL EXAMINATION, URINE**

| | |
|------------|-------------|
| COLOR | PALE YELLOW |
| APPEARANCE | CLEAR |

CHEMICAL EXAMINATION, URINE

| | | |
|--------------------|--------------|---------------|
| PH | 6.0 | 5.00 - 7.50 |
| SPECIFIC GRAVITY | 1.015 | 1.010 - 1.030 |
| PROTEIN | NOT DETECTED | NOT DETECTED |
| GLUCOSE | NOT DETECTED | NOT DETECTED |
| KETONES | NOT DETECTED | NOT DETECTED |
| BLOOD | NOT DETECTED | NOT DETECTED |
| BILIRUBIN | NOT DETECTED | NOT DETECTED |
| UROBILINOGEN | NOT DETECTED | |
| NITRITE | NOT DETECTED | NOT DETECTED |
| LEUKOCYTE ESTERASE | NOT DETECTED | NOT DETECTED |

MICROSCOPIC EXAMINATION, URINE

| | | | |
|------------------|--------------|--------------|------|
| RED BLOOD CELLS | NOT DETECTED | NOT DETECTED | /HPF |
| PUS CELL (WBC'S) | 0-1 | 0-5 | /HPF |
| EPITHELIAL CELLS | 0-1 | 0-5 | /HPF |
| CASTS | NOT DETECTED | | |
| CRYSTALS | NOT DETECTED | | |
| BACTERIA | NOT DETECTED | NOT DETECTED | |
| YEAST | NOT DETECTED | NOT DETECTED | |

METHOD : URINE ROUTINE & MICROSCOPY EXAMINATION BY INTEGRATED AUTOMATED SYSTEM

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Mumbai, 400062
Maharashtra, India
Tel : 9111591115, 022 - 67801212
CIN - U74899PB1995PLC045956



Patient Ref. No. 2000012092810



MC-5718

PATIENT NAME : MAMTA BIST**REF. DOCTOR : SELF**B 1205 Vasundhara chs ltd building no 6 shastri
nagar siddharth hospital road
400104ACCESSION NO : **0002WK032382**
PATIENT ID : MAMTF31128527
CLIENT PATIENT ID:
ABHA NO :AGE/SEX : 37 Years Female
DRAWN : 25/11/2023 08:48:53
RECEIVED : 25/11/2023 08:50:50
REPORTED : 27/11/2023 14:43:05**Test Report Status** Final **Results** **Biological Reference Interval** **Units****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 infection when present in significant numbers & with pus cells. |
| Trichomonas vaginalis | Vaginitis, cervicitis or salpingitis |

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CYTOLOGY**MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE****PAPANICOLAOU SMEAR**

| | |
|-------------------------|---|
| TEST METHOD | CONVENTIONAL GYNEC CYTOLOGY |
| SPECIMEN TYPE | TWO CERVICAL SMEARS RECEIVED (2CW-30892). |
| REPORTING SYSTEM | 2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY |
| SPECIMEN ADEQUACY | SMEARS ARE SATISFACTORY FOR EVALUATION. |
| MICROSCOPY | THE SMEARS SHOW MAINLY INTERMEDIATE SQUAMOUS CELLS, FEW SUPERFICIAL SQUAMOUS CELLS, OCCASIONAL CLUSTERS OF ENDOCERVICAL CELLS AND FEW POLYMORPHS. |
| INTERPRETATION / RESULT | NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY |

Comments

Suggestions / Guidelines: (REF: THE BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY,2014, 3rd Edition)
PAP RE-TESTING AT 3 YEARS

- 1) Please note papanicolaou smear study is a screening procedure for cervical cancer with inherent false negative results, hence should be interpreted with caution.
- 2) No cytologic evidence of hpv infection in the smears studied.
- 3) Primary screening of papanicolaou smears is carried out by cytotechnologist with 100% rescreening and reporting by surgical pathologist.

V. Swathi

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CLINICAL PATH - STOOL ANALYSIS**MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE****PHYSICAL EXAMINATION,STOOL**

| | | | |
|----------------|--------------|--------------|--|
| COLOUR | BROWN | | |
| CONSISTENCY | SEMI FORMED | | |
| MUCUS | NOT DETECTED | NOT DETECTED | |
| VISIBLE BLOOD | ABSENT | ABSENT | |
| ADULT PARASITE | NOT DETECTED | | |

METHOD : MICROSCOPIC EXAMINATION

CHEMICAL EXAMINATION,STOOL

| | | | |
|--------------|-------|--------------|--|
| STOOL PH | 6.0 | | |
| OCCULT BLOOD | TRACE | NOT DETECTED | |

METHOD : MODIFIED GUAIAC METHOD

MICROSCOPIC EXAMINATION,STOOL

| | | | |
|-------------------------|--------------|--------------|------|
| PUS CELLS | 0-1 | | /hpf |
| RED BLOOD CELLS | 0 - 1 | NOT DETECTED | /HPF |
| CYSTS | NOT DETECTED | NOT DETECTED | |
| OVA | NOT DETECTED | | |
| LARVAE | NOT DETECTED | NOT DETECTED | |
| TROPHOZOITES | NOT DETECTED | NOT DETECTED | |
| FAT | ABSENT | | |
| CHARCOT LEYDEN CRYSTALS | ABSENT | | |

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Stool routine analysis is only a screening test for disorders of gastrointestinal tract like infection, malabsorption, etc. The following table describes the probable conditions, in which the analytes are present in stool.

| PRESENCE OF | CONDITION |
|------------------------|---|
| Pus cells | Pus in the stool is an indication of infection |
| Red Blood cells | Parasitic or bacterial infection or an inflammatory bowel condition such as ulcerative colitis |
| Parasites | Infection of the digestive system. Stool examination for ova and parasite detects presence of parasitic infestation of gastrointestinal tract. Various forms of parasite that can be detected include cyst, trophozoite and larvae. One negative result does not rule out the possibility of parasitic infestation. Intermittent shedding of parasites warrants examinations of multiple specimens tested on consecutive days. Stool specimens for parasitic examination should be collected before initiation of anti-diarrheal therapy or antiparasitic therapy. This test does not detect presence of opportunistic parasites like Cyclospora, Cryptosporidia and Isospora species. Examination of Ova and Parasite has been carried out by direct and concentration techniques. |
| Mucus | Mucus is a protective layer that lubricates, protects & reduces damage due to bacteria or viruses. |
| Charcot-Leyden crystal | Parasitic diseases. |
| Ova & cyst | Ova & cyst indicate parasitic infestation of intestine. |
| Frank blood | Bleeding in the rectum or colon. |
| Occult blood | Occult blood indicates upper GI bleeding. |
| Macrophages | Macrophages in stool are an indication of infection as they are protective cells. |
| Epithelial cells | Epithelial cells that normally line the body surface and internal organs show up in stool when there is inflammation or infection. |
| Fat | Increased fat in stool maybe seen in conditions like diarrhoea or malabsorption. |
| pH | Normal stool pH is slightly acidic to neutral. Breast-fed babies generally have an acidic stool. |

ADDITIONAL STOOL TESTS :

- Stool Culture**:- This test is done to find cause of GI infection, make decision about best treatment for GI infection & to find out if treatment for GI infection worked.
- Fecal Calprotectin**: It is a marker of intestinal inflammation. This test is done to differentiate Inflammatory Bowel Disease (IBD) from Irritable Bowel Syndrome (IBS).
- Fecal Occult Blood Test (FOBT)**: This test is done to screen for colon cancer & to evaluate possible cause of unexplained anaemia.
- Clostridium Difficile Toxin Assay**: This test is strongly recommended in healthcare associated bloody or watery diarrhoea, due to overuse of broad spectrum antibiotics which alter the normal GI flora.
- Biofire (Film Array) GI PANEL**: In patients of Diarrhoea, Dysentery, Rice watery Stool, FDA approved, Biofire Film Array Test (Real Time Multiplex PCR) is strongly recommended as it identifies organisms, bacteria, fungi, virus, parasite and other opportunistic pathogens, Vibrio cholera infections only in 3 hours. Sensitivity 96% & Specificity 99%.

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6. **Rota Virus Immunoassay:** This test is recommended in severe gastroenteritis in infants & children associated with watery diarrhoea, vomiting & abdominal cramps. Adults are also affected. It is highly contagious in nature.

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SPECIALISED CHEMISTRY - HORMONE**MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE****THYROID PANEL, SERUM**

| | | | |
|----|------|------------------------------|-------|
| T3 | 99.7 | Non-Pregnant Women | ng/dL |
| | | 80.0 - 200.0 | |
| | | Pregnant Women | |
| | | 1st Trimester: 105.0 - 230.0 | |
| | | 2nd Trimester: 129.0 - 262.0 | |
| | | 3rd Trimester: 135.0 - 262.0 | |

METHOD : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY

| | | | |
|----|------|-----------------------------|-------|
| T4 | 6.14 | Non-Pregnant Women | µg/dL |
| | | 5.10 - 14.10 | |
| | | Pregnant Women | |
| | | 1st Trimester: 7.33 - 14.80 | |
| | | 2nd Trimester: 7.93 - 16.10 | |
| | | 3rd Trimester: 6.95 - 15.70 | |

METHOD : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY

| | | | |
|----------------------|-------|-------------------------------|--------------|
| TSH (ULTRASENSITIVE) | 3.340 | NonPregnant Women | 0.27- µIU/mL |
| | | 4.20 | |
| | | Pregnant Women (As per | |
| | | American Thyroid Association) | |
| | | 1st Trimester 0.100 - 2.500 | |
| | | 2nd Trimester 0.200 - 3.000 | |
| | | 3rd Trimester 0.300 - 3.000 | |

METHOD : SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY

Comments

Important Note: Please note the change in Biological Reference Interval of TSH for Pregnant Women.

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.

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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 |
| 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. Guidelines of the American Thyroid association during pregnancy and Postpartum, 2011.

TSH in pregnancy

There's reduction in both the lower and the upper limit of maternal TSH relative to the non-pregnant TSH reference range. This is because of elevated levels of serum hCG that directly stimulates the TSH receptor, thereby increasing thyroid hormone production. The largest decrease in serum TSH is observed during the first trimester. Thereafter, serum TSH and its reference range gradually increases in the second and third trimesters, but nonetheless remains lower than in non-pregnant women.

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 accessionDr. Deepak Sanghavi, M.D (Path)
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