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| | | | |
|---|---|---|---|
| **NAME** | **: Mrs. NARESH KUMARI WALIA** | | |
| **AGE/ GENDER** | : 71 YRS/FEMALE | **PATIENT ID** | : 1604959 |
| **COLLECTED BY** | : SURJESH | **REG. NO./LAB NO.** | **: 012409070017** |
| **REFERRED BY** | : | **REGISTRATION DATE** | : 07/Sep/2024 08:50 AM |
| **BARCODE NO.** | : 01516470 | **COLLECTION DATE** | : 07/Sep/2024 08:52AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 07/Sep/2024 09:18AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

## SWASTHYA WELLNESS PANEL: 1.5

### COMPLETE BLOOD COUNT (CBC)

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **RED BLOOD CELLS (RBCS) COUNT AND INDICES** | | | |
| **HAEMOGLOBIN (HB)** *by CALORIMETRIC* | **11[L]** | **gm/dL** | **12.0 - 16.0** |
| RED BLOOD CELL (RBC) COUNT *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE* | 4.11 | Millions/cmm | 3.50 - 5.00 |
| **PACKED CELL VOLUME (PCV)** *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER* | **34.2[L]** | **%** | **37.0 - 50.0** |
| MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER* | 83.2 | fL | 80.0 - 100.0 |
| **MEAN CORPUSCULAR HAEMOGLOBIN (MCH)** *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER* | **26.7[L]** | **pg** | **27.0 - 34.0** |
| MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER* | 32.1 | g/dL | 32.0 - 36.0 |
| RED CELL DISTRIBUTION WIDTH (RDW-CV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER* | 14.1 | % | 11.00 - 16.00 |
| RED CELL DISTRIBUTION WIDTH (RDW-SD) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER* | 43.9 | fL | 35.0 - 56.0 |
| MENTZERS INDEX *by CALCULATED* | 20.24 | RATIO | BETA THALASSEMIA TRAIT: < 13.0<br>IRON DEFICIENCY ANEMIA: >13.0 |
| GREEN & KING INDEX *by CALCULATED* | 28.47 | RATIO | BETA THALASSEMIA TRAIT:<= 65.0<br>IRON DEFICIENCY ANEMIA: > 65.0 |
| **WHITE BLOOD CELLS (WBCS)** | | | |
| TOTAL LEUCOCYTE COUNT (TLC) *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 5720 | /cmm | 4000 - 11000 |
| NUCLEATED RED BLOOD CELLS (nRBCS) *by AUTOMATED 6 PART HEMATOLOGY ANALYZER* | NIL | | 0.00 - 20.00 |
| NUCLEATED RED BLOOD CELLS (nRBCS) % *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER* | NIL | % | < 10 % |
| **DIFFERENTIAL LEUCOCYTE COUNT (DLC)** | | | |
| NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 57 | % | 50 - 70 |

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| LYMPHOCYTES<br>*by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 27 | % | 20 - 40 |
| **EOSINOPHILS**<br>***by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*** | **9 H** | **%** | **1 - 6** |
| MONOCYTES<br>*by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 7 | % | 2 - 12 |
| BASOPHILS<br>*by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 0 | % | 0 - 1 |
| **ABSOLUTE LEUKOCYTES (WBC) COUNT** | | | |
| ABSOLUTE NEUTROPHIL COUNT<br>*by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 3260 | /cmm | 2000 - 7500 |
| ABSOLUTE LYMPHOCYTE COUNT<br>*by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 1544 | /cmm | 800 - 4900 |
| **ABSOLUTE EOSINOPHIL COUNT**<br>***by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*** | **515 H** | **/cmm** | **40 - 440** |
| ABSOLUTE MONOCYTE COUNT<br>*by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 400 | /cmm | 80 - 880 |
| ABSOLUTE BASOPHIL COUNT<br>*by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY* | 0 | /cmm | 0 - 110 |
| **PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.** | | | |
| PLATELET COUNT (PLT)<br>*by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE* | 314000 | /cmm | 150000 - 450000 |
| PLATELETCRIT (PCT)<br>*by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE* | 0.31 | % | 0.10 - 0.36 |
| MEAN PLATELET VOLUME (MPV)<br>*by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE* | 10 | fL | 6.50 - 12.0 |
| PLATELET LARGE CELL COUNT (P-LCC)<br>*by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE* | 78000 | /cmm | 30000 - 90000 |
| PLATELET LARGE CELL RATIO (P-LCR)<br>*by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE* | 24.9 | % | 11.0 - 45.0 |
| PLATELET DISTRIBUTION WIDTH (PDW)<br>*by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE* | 16 | % | 15.0 - 17.0 |

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 07/Sep/2024 02:54PM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

## GLYCOSYLATED HAEMOGLOBIN (HBA1C)

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD<br>*by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)* | 6 | % | 4.0 - 6.4 |
| ESTIMATED AVERAGE PLASMA GLUCOSE<br>*by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)* | 125.5 | mg/dL | 60.00 - 140.00 |

***INTERPRETATION:***

| AS PER AMERICAN DIABETES ASSOCIATION (ADA): | | |
|---|---|---|
| **REFERENCE GROUP** | **GLYCOSYLATED HEMOGLOGIB (HBAIC) in %** | |
| Non diabetic Adults >= 18 years | <5.7 | |
| At Risk (Prediabetes) | 5.7 – 6.4 | |
| Diagnosing Diabetes | >= 6.5 | |
| Therapeutic goals for glycemic control | **Age > 19 Years** | |
| | Goals of Therapy: | < 7.0 |
| | Actions Suggested: | >8.0 |
| | **Age < 19 Years** | |
| | Goal of therapy: | <7.5 |

**COMMENTS:**

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients.

*2.Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAIc. Converse is true for a diabetic previously under good control but now poorly controlled.*

*3.Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropiate.*

*4.*High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications

5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution , given the pathological processes including anemia,increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## ERYTHROCYTE SEDIMENTATION RATE (ESR)

| | | | |
|---|---|---|---|
| ERYTHROCYTE SEDIMENTATION RATE (ESR) | 16 | mm/1st hr | 0 - 20 |

*by MODIFIED WESTERGREN AUTOMATED METHOD*

**INTERPRETATION:**

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

**CONDITION WITH LOW ESR**

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis) , and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

**NOTE:**

1. ESR and C - reactive protein (C-RP) are both markers of inflammation.
2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
3. **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.**
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## CLINICAL CHEMISTRY/BIOCHEMISTRY

### GLUCOSE FASTING (F)

| | | | |
|---|---|---|---|
| **GLUCOSE FASTING (F): PLASMA**<br>*by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)* | **115.89$^{H}$** | **mg/dL** | **NORMAL: < 100.0**<br>**PREDIABETIC: 100.0 - 125.0**<br>**DIABETIC: > 0R = 126.0** |

***INTERPRETATION***

**IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:**

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| | **LIPID PROFILE : BASIC** | | |
| **CHOLESTEROL TOTAL: SERUM**<br>***by CHOLESTEROL OXIDASE PAP*** | **232.11$^{H}$** | **mg/dL** | **OPTIMAL: < 200.0**<br>**BORDERLINE HIGH: 200.0 - 239.0**<br>**HIGH CHOLESTEROL: > OR = 240.0** |
| **TRIGLYCERIDES: SERUM**<br>***by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)*** | **188.16$^{H}$** | **mg/dL** | **OPTIMAL: < 150.0**<br>**BORDERLINE HIGH: 150.0 - 199.0**<br>**HIGH: 200.0 - 499.0**<br>**VERY HIGH: > OR = 500.0** |
| HDL CHOLESTEROL (DIRECT): SERUM<br>*by SELECTIVE INHIBITION* | 68.46 | mg/dL | LOW HDL: < 30.0<br>BORDERLINE HIGH HDL: 30.0 - 60.0<br>HIGH HDL: > OR = 60.0 |
| LDL CHOLESTEROL: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 126.02 | mg/dL | OPTIMAL: < 100.0<br>ABOVE OPTIMAL: 100.0 - 129.0<br>BORDERLINE HIGH: 130.0 - 159.0<br>HIGH: 160.0 - 189.0<br>VERY HIGH: > OR = 190.0 |
| **NON HDL CHOLESTEROL: SERUM**<br>***by CALCULATED, SPECTROPHOTOMETRY*** | **163.65$^{H}$** | **mg/dL** | **OPTIMAL: < 130.0**<br>**ABOVE OPTIMAL: 130.0 - 159.0**<br>**BORDERLINE HIGH: 160.0 - 189.0**<br>**HIGH: 190.0 - 219.0**<br>**VERY HIGH: > OR = 220.0** |
| VLDL CHOLESTEROL: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 37.63 | mg/dL | 0.00 - 45.00 |
| TOTAL LIPIDS: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 652.38 | mg/dL | 350.00 - 700.00 |
| CHOLESTEROL/HDL RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 3.39 | RATIO | LOW RISK: 3.30 - 4.40<br>AVERAGE RISK: 4.50 - 7.0<br>MODERATE RISK: 7.10 - 11.0<br>HIGH RISK: > 11.0 |
| LDL/HDL RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 1.84 | RATIO | LOW RISK: 0.50 - 3.0<br>MODERATE RISK: 3.10 - 6.0<br>HIGH RISK: > 6.0 |

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **TRIGLYCERIDES/HDL RATIO: SERUM**<br>***by CALCULATED, SPECTROPHOTOMETRY*** | **2.75**$^{L}$ | **RATIO** | **3.00 - 5.00** |

***INTERPRETATION:***

1.Measurements in the same patient can show physiological& analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL , VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL &Non HDL.
5. Additional testing for Apolipoprotein B, hsCRP,Lp(a ) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement

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## LIVER FUNCTION TEST (COMPLETE)

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| BILIRUBIN TOTAL: SERUM<br>*by DIAZOTIZATION, SPECTROPHOTOMETRY* | 0.42 | mg/dL | INFANT: 0.20 - 8.00<br>ADULT: 0.00 - 1.20 |
| BILIRUBIN DIRECT (CONJUGATED): SERUM<br>*by DIAZO MODIFIED, SPECTROPHOTOMETRY* | 0.09 | mg/dL | 0.00 - 0.40 |
| BILIRUBIN INDIRECT (UNCONJUGATED): SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 0.33 | mg/dL | 0.10 - 1.00 |
| SGOT/AST: SERUM<br>*by IFCC, WITHOUT PYRIDOXAL PHOSPHATE* | 24.9 | U/L | 7.00 - 45.00 |
| SGPT/ALT: SERUM<br>*by IFCC, WITHOUT PYRIDOXAL PHOSPHATE* | 26.8 | U/L | 0.00 - 49.00 |
| AST/ALT RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 0.93 | RATIO | 0.00 - 46.00 |
| ALKALINE PHOSPHATASE: SERUM<br>*by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL* | 126.25 | U/L | 40.0 - 130.0 |
| **GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM**<br>***by SZASZ, SPECTROPHTOMETRY*** | **68.39$^{H}$** | **U/L** | **0.00 - 55.0** |
| TOTAL PROTEINS: SERUM<br>*by BIURET, SPECTROPHOTOMETRY* | 7.02 | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM<br>*by BROMOCRESOL GREEN* | 4.42 | gm/dL | 3.50 - 5.50 |
| GLOBULIN: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 2.6 | gm/dL | 2.30 - 3.50 |
| A : G RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 1.7 | RATIO | 1.00 - 2.00 |

***INTERPRETATION***

***NOTE***:- **To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.**
**USE**:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

**INCREASED:**

| | |
|---|---|
| DRUG HEPATOTOXICITY | > 2 |
| ALCOHOLIC HEPATITIS | > 2 (Highly Suggestive) |
| CIRRHOSIS | 1.4 - 2.0 |
| INTRAHEPATIC CHOLESTATIS | > 1.5 |
| HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS | > 1.3 (Slightly Increased) |

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

**DECREASED:**

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

**PROGNOSTIC SIGNIFICANCE:**

| | |
|---|---|
| NORMAL | < 0.65 |
| GOOD PROGNOSTIC SIGN | 0.3 - 0.6 |
| POOR PROGNOSTIC SIGN | 1.2 - 1.6 |

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|---|---|---|---|
| **NAME** | **: Mrs. NARESH KUMARI WALIA** | | |
| **AGE/ GENDER** | : 71 YRS/FEMALE | **PATIENT ID** | : 1604959 |
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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **KIDNEY FUNCTION TEST (COMPLETE)** | | | |
| UREA: SERUM<br>*by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)* | 39.71 | mg/dL | 10.00 - 50.00 |
| CREATININE: SERUM<br>*by ENZYMATIC, SPECTROPHOTOMETERY* | 1.09 | mg/dL | 0.40 - 1.20 |
| BLOOD UREA NITROGEN (BUN): SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 18.56 | mg/dL | 7.0 - 25.0 |
| BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 17.03 | RATIO | 10.0 - 20.0 |
| UREA/CREATININE RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 36.43 | RATIO | |
| URIC ACID: SERUM<br>*by URICASE - OXIDASE PEROXIDASE* | 5.03 | mg/dL | 2.50 - 6.80 |
| CALCIUM: SERUM<br>*by ARSENAZO III, SPECTROPHOTOMETRY* | 9.52 | mg/dL | 8.50 - 10.60 |
| PHOSPHOROUS: SERUM<br>*by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY* | 3.37 | mg/dL | 2.30 - 4.70 |
| **ELECTROLYTES** | | | |
| SODIUM: SERUM<br>*by ISE (ION SELECTIVE ELECTRODE)* | 137.5 | mmol/L | 135.0 - 150.0 |
| POTASSIUM: SERUM<br>*by ISE (ION SELECTIVE ELECTRODE)* | 4.18 | mmol/L | 3.50 - 5.00 |
| CHLORIDE: SERUM<br>*by ISE (ION SELECTIVE ELECTRODE)* | 103.13 | mmol/L | 90.0 - 110.0 |
| **ESTIMATED GLOMERULAR FILTERATION RATE** | | | |
| ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM<br>*by CALCULATED* | 54.3 | | |

***INTERPRETATION:***

To differentiate between pre- and post renal azotemia.

**INCREASED RATIO (>20:1) WITH NORMAL CREATININE:**

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion,dehydration, blood loss) due to decreased glomerular filtration rate.
2. Catabolic states with increased tissue breakdown.

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

3. GI haemorrhage.
4. High protein intake.
5. Impaired renal function plus
6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
7. Urine reabsorption (e.g. ureter colostomy)
8. Reduced muscle mass (subnormal creatinine production)
9. Certain drugs (e.g. tetracycline, glucocorticoids)

**INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:**

1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
2. Prerenal azotemia superimposed on renal disease.

**DECREASED RATIO (<10:1) WITH DECREASED BUN :**

1. Acute tubular necrosis.
2. Low protein diet and starvation.
3. Severe liver disease.
4. Other causes of decreased urea synthesis.
5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
6. Inherited hyperammonemias (urea is virtually absent in blood).
7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.
8. Pregnancy.

**DECREASED RATIO (<10:1) WITH INCREASED CREATININE:**

1. Phenacimide therapy (accelerates conversion of creatine to creatinine).
2. Rhabdomyolysis (releases muscle creatinine).
3. Muscular patients who develop renal failure.

**INAPPROPIATE RATIO:**

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies,resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
2. Cephalosporin therapy (interferes with creatinine measurement).

**ESTIMATED GLOMERULAR FILTERATION RATE:**

| CKD STAGE | DESCRIPTION | GFR ( mL/min/1.73m2 ) | ASSOCIATED FINDINGS |
|---|---|---|---|
| G1 | Normal kidney function | >90 | No proteinuria |
| G2 | Kidney damage with normal or high GFR | >90 | Presence of Protein , Albumin or cast in urine |
| G3a | Mild decrease in GFR | 60 -89 | |
| G3b | Moderate decrease in GFR | 30-59 | |
| G4 | Severe decrease in GFR | 15-29 | |
| G5 | Kidney failure | <15 | |

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

COMMENTS:

1. Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
3. In patients, with eGFR creatinine between 45-59 ml/min/1.73 m2 (G3) and without any marker of Kidney damage, It is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fullfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
***7. A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).***

***ADVICE:***

KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **IRON PROFILE** | | | |
| IRON: SERUM<br>*by FERROZINE, SPECTROPHOTOMETRY* | 45.7 | µg/dL | 37.0 - 145.0 |
| UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM<br>*by FERROZINE, SPECTROPHOTOMETERY* | 290.75 | µg/dL | 150.0 - 336.0 |
| TOTAL IRON BINDING CAPACITY (TIBC) :SERUM<br>*by SPECTROPHOTOMETERY* | 336.45 | µg/dL | 230 - 430 |
| **%TRANSFERRIN SATURATION: SERUM**<br>***by CALCULATED, SPECTROPHOTOMETERY (FERENE)*** | **13.58**$^{L}$ | **%** | **15.0 - 50.0** |
| TRANSFERRIN: SERUM<br>*by SPECTROPHOTOMETERY (FERENE)* | 238.88 | mg/dL | 200.0 - 350.0 |

***INTERPRETATION:-***

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

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

**TOTAL IRON BINDING CAPACITY (TIBC):**
1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

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

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## ENDOCRINOLOGY

### THYROID FUNCTION TEST: TOTAL

| | | | |
|---|---|---|---|
| TRIIODOTHYRONINE (T3): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 0.824 | ng/mL | 0.35 - 1.93 |
| THYROXINE (T4): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 8.23 | µgm/dL | 4.87 - 12.60 |
| THYROID STIMULATING HORMONE (TSH): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 1.679 | µIU/mL | 0.35 - 5.50 |

**3rd GENERATION, ULTRASENSITIVE**

***INTERPRETATION:***

*TSH levels are subject to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50%.Hence time of the day has influence on the measured serum TSH concentrations.*TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4)and trilodothyronine (T3).Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction(hyperthyroidism) of T4 and/or T3.

| CLINICAL CONDITION | T3 | T4 | TSH |
|---|---|---|---|
| Primary Hypothyroidism: | Reduced | Reduced | Increased (Significantly) |
| Subclinical Hypothyroidism: | Normal or Low Normal | Normal or Low Normal | High |
| Primary Hyperthyroidism: | Increased | Increased | Reduced (at times undetectable) |
| Subclinical Hyperthyroidism: | Normal or High Normal | Normal or High Normal | Reduced |

**LIMITATIONS:-**

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG),and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (eg: phenytoin , salicylates).
3. Serum T4 levles in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.
4. TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothroidism , pregnancy , phenytoin therapy.

| TRIIODOTHYRONINE (T3) | | THYROXINE (T4) | | THYROID STIMULATING HORMONE (TSH) | |
|---|---|---|---|---|---|
| **Age** | **Refferance Range (ng/mL)** | **Age** | **Refferance Range ( µg/dL)** | **Age** | **Reference Range ( µIU/mL)** |
| **0 - 7 Days** | **0.20 - 2.65** | **0 - 7 Days** | **5.90 – 18.58** | **0 - 7 Days** | **2.43 - 24.3** |
| **7 Days - 3 Months** | **0.36 - 2.59** | **7 Days - 3 Months** | **6.39 - 17.66** | **7 Days - 3 Months** | **0.58 - 11.00** |
| **3 - 6 Months** | **0.51 - 2.52** | **3 - 6 Months** | **6.75 – 17.04** | **3 Days – 6 Months** | **0.70 - 8.40** |

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| Test Name | | | Value | Unit | Biological Reference interval |
|---|---|---|---|---|---|
| 6 - 12 Months | 0.74 - 2.40 | 6 - 12 Months | 7.10 – 16.16 | 6 – 12 Months | 0.70 - 7.00 |
| 1 - 10 Years | 0.92 - 2.28 | 1 - 10 Years | 6.00 - 13.80 | 1 – 10 Years | 0.60 - 5.50 |
| 11- 19 Years | 0.35 - 1.93 | 11 - 19 Years | 4.87- 13.20 | 11 – 19 Years | 0.50 – 5.50 |
| > 20 years (Adults) | 0.35 - 1.93 | > 20 Years (Adults) | 4.87 - 12.60 | > 20 Years (Adults) | 0.35– 5.50 |
| RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY ( µIU/mL) | | | | | |
| 1st Trimester | | | 0.10 – 2.50 | | |
| 2nd Trimester | | | 0.20 – 3.00 | | |
| 3rd Trimester | | | 0.30 – 4.10 | | |

**INCREASED TSH LEVELS:**

1.Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.
2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
3.Hashimotos thyroiditis
4.DRUGS: Amphetamines, idonie containing agents & dopamine antagonist.
5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

**DECREASED TSH LEVELS:**

1.Toxic multi-nodular goitre & Thyroiditis.
2.Over replacement of thyroid harmone in treatment of hypothyroidism.
3.Autonomously functioning Thyroid adenoma
4.Secondary pituatary or hypothalmic hypothyroidism
5.Acute psychiatric illness
6.Severe dehydration.
7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
8.Pregnancy: 1st and 2nd Trimester

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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

| | | | |
|---|---|---|---|
| VITAMIN D (25-HYDROXY VITAMIN D3): SERUM<br>*by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)* | 42.8 | ng/mL | DEFICIENCY: < 20.0<br>INSUFFICIENCY: 20.0 - 30.0<br>SUFFICIENCY: 30.0 - 100.0<br>TOXICITY: > 100.0 |

***INTERPRETATION:***

| | | |
|---|---|---|
| **DEFICIENT:** | < 20 | ng/mL |
| **INSUFFICIENT:** | 21 - 29 | ng/mL |
| **PREFFERED RANGE:** | 30 - 100 | ng/mL |
| **INTOXICATION:** | > 100 | ng/mL |

1.Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
3.Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).
4.Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

**DECREASED:**
1.Lack of sunshine exposure.
2.Inadequate intake, malabsorption (celiac disease)
3.Depressed Hepatic Vitamin D 25- hydroxylase activity
4.Secondary to advanced Liver disease
5.Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

**INCREASED:**
1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

**CAUTION**: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

**NOTE**:-*Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.*

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## VITAMIN B12/COBALAMIN

| | | | |
|---|---|---|---|
| VITAMIN B12/COBALAMIN: SERUM | 609 | pg/mL | 190.0 - 890.0 |

*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*

***INTERPRETATION:-***

| INCREASED VITAMIN B12 | DECREASED VITAMIN B12 |
|---|---|
| 1.Ingestion of Vitamin C | 1.Pregnancy |
| 2.Ingestion of Estrogen | 2.DRUGS:Aspirin, Anti-convulsants, Colchicine |
| 3.Ingestion of Vitamin A | 3.Ethanol Igestion |
| 4.Hepatocellular injury | 4. Contraceptive Harmones |
| 5.Myeloproliferative disorder | 5.Haemodialysis |
| 6.Uremia | 6. Multiple Myeloma |

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.
**NOTE:**A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

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MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

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|---|---|---|---|
| **NAME** | **: Mrs. NARESH KUMARI WALIA** | | |
| **AGE/ GENDER** | : 71 YRS/FEMALE | **PATIENT ID** | : 1604959 |
| **COLLECTED BY** | : SURJESH | **REG. NO./LAB NO.** | **: 012409070017** |
| **REFERRED BY** | : | **REGISTRATION DATE** | : 07/Sep/2024 08:50 AM |
| **BARCODE NO.** | : 01516470 | **COLLECTION DATE** | : 07/Sep/2024 08:52AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 07/Sep/2024 10:00AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

## CLINICAL PATHOLOGY

### URINE ROUTINE & MICROSCOPIC EXAMINATION

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **PHYSICAL EXAMINATION** | | | |
| QUANTITY RECIEVED<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | 10 | ml | |
| COLOUR<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | AMBER YELLOW | | PALE YELLOW |
| TRANSPARANCY<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | CLEAR | | CLEAR |
| SPECIFIC GRAVITY<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | <=1.005 | | 1.002 - 1.030 |
| **CHEMICAL EXAMINATION** | | | |
| REACTION<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | ACIDIC | | |
| PROTEIN<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | Negative | | NEGATIVE (-ve) |
| SUGAR<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | Negative | | NEGATIVE (-ve) |
| pH<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | <=5.0 | | 5.0 - 7.5 |
| BILIRUBIN<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | Negative | | NEGATIVE (-ve) |
| NITRITE<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.* | Negative | | NEGATIVE (-ve) |
| UROBILINOGEN<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | Normal | EU/dL | 0.2 - 1.0 |
| KETONE BODIES<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | Negative | | NEGATIVE (-ve) |
| BLOOD<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | Negative | | NEGATIVE (-ve) |
| ASCORBIC ACID<br>*by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY* | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| **MICROSCOPIC EXAMINATION** | | | |

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

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

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|---|---|---|---|
| RED BLOOD CELLS (RBCs)<br>*by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT* | NEGATIVE (-ve) | /HPF | 0 - 3 |
| PUS CELLS<br>*by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT* | 1-3 | /HPF | 0 - 5 |
| EPITHELIAL CELLS<br>*by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT* | 3-4 | /HPF | ABSENT |
| CRYSTALS<br>*by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT* | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| CASTS<br>*by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT* | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| BACTERIA<br>*by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT* | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| OTHERS<br>*by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT* | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| TRICHOMONAS VAGINALIS (PROTOZOA)<br>*by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT* | ABSENT | | ABSENT |

*** End Of Report ***

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