

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



| Dr. Vinay Ch MD (Pathology & Chairman & Con | | | Pathology) |
|---|------------------|--------------------------|--|
| NAME : Miss. VANSHIKA | | | |
| AGE/ GENDER : 25 YRS/FEMALE | | PATIENT ID | : 1604171 |
| COLLECTED BY : | | REG. NO./LAB NO. | : 012409060058 |
| REFERRED BY : | | REGISTRATION DATE | : 06/Sep/2024 02:00 PM |
| BARCODE NO. : 01516441 | | COLLECTION DATE | :06/Sep/202402:07PM |
| CLIENT CODE. : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 06/Sep/2024 02:26PM |
| CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, | AMBALA CANTT | | |
| Test Name | Value | Unit | Biological Reference interval |
| SM | VASTHYA WE | LLNESS PANEL: 1.0 | |
| | COMPLETE BLO | DOD COUNT (CBC) | |
| RED BLOOD CELLS (RBCS) COUNT AND INDICES | | | |
| HAEMOGLOBIN (HB) by CALORIMETRIC | 15.9 | gm/dL | 12.0 - 16.0 |
| RED BLOOD CELL (RBC) COUNT | 5.5 ^H | Millions/c | mm 3.50 - 5.00 |
| by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ | 48.5 | % | 37.0 - 50.0 |
| MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ | 88.1 | fL | 80.0 - 100.0 |
| MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ | 29 ER | pg | 27.0 - 34.0 |
| MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by calculated by automated hematology analyz | | g/dL | 32.0 - 36.0 |
| RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ | | % | 11.00 - 16.00 |
| RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ | | fL | 35.0 - 56.0 |
| MENTZERS INDEX by CALCULATED | 16.02 | RATIO | BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0 |
| GREEN & KING INDEX by CALCULATED | 22.01 | RATIO | BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0 |
| WHITE BLOOD CELLS (WBCS) | | | |
| TOTAL LEUCOCYTE COUNT (TLC) by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 8580 | /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 ANALYZ DIFFERENTIAL LEUCOCYTE COUNT (DLC) | NIL ER | % | < 10 % |
| DITTERLINITAL LEUGOUTTE GOUNT (DLG) | | | |



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Page 1 of 15

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Dr. Yugam Chopra Dr. Vinay Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Miss. VANSHIKA AGE/ GENDER : 25 YRS/FEMALE **PATIENT ID** :1604171 **COLLECTED BY** :012409060058 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** :06/Sep/2024 02:00 PM **BARCODE NO.** :01516441 **COLLECTION DATE** :06/Sep/2024 02:07PM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :06/Sep/2024 02:26PM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** LYMPHOCYTES 20 - 40 35 % by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 2 % 1-6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES % 2 - 12 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LEUKOCYTES (WBC) COUNT ABSOLUTE NEUTROPHIL COUNT 4891 /cmm 2000 - 7500 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY 800 - 4900 ABSOLUTE LYMPHOCYTE COUNT 3003 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 172 40 - 440 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 515 80 - 880 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 - 110 0 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. 249000 150000 - 450000 PLATELET COUNT (PLT) /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 0.10 - 0.36 PLATELETCRIT (PCT) 0.3 % by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 12 fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) 102000^H 30000 - 90000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) 40.9 % 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) % 15.0 - 17.0 16.6 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, A | MBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | ERYTH | ROCYTE SEDII | MENTATION RATE (ESP | 2) |
| by MODIFIED WESTER INTERPRETATION: 1. ESR is a non-specifi mmune disease, but 4 2. An ESR can be affect as C-reactive protein 3. This test may also b systemic lupus erythe CONDITION WITH LOV A low ESR can be seer (polycythaemia), sign as sickle cells in sickle NOTE: 1. ESR and C - reactive 2. Generally, ESR does 3. CRP is not affected 4. If the ESR is elevated 5. Women tend to hav 6. Drugs such as dexti | does not tell the health practition ted by other conditions besides i matosus V ESR n with conditions that inhibit the ificantly high white blood cell cou e cell anaemia) also lower the ES protein (C-RP) are both markers s not change as rapidly as does CF by as many other factors as is ESR ed, it is typically a result of two ty e a higher ESR, and menstruation | ner exactly wher inflammation. For ty and response normal sedimer unt (leucocytosis R. of inflammation RP, either at the t , making it a bet ypes of proteins, and prognancy | e the inflammation is in the or this reason, the ESR is typ to therapy in both of the al ntation of red blood cells, su s), and some protein abnor start of inflammation or as tter marker of inflammation globulins or fibrinogen. can cause temporary eleva | on associated with infection, cancer and auto- body or what is causing it. bically used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count rmalities. Some changes in red cell shape (such tit resolves. |
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Page 3 of 15





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| Test Name | | Value | Unit | Biological Reference interval |
| | CLIN | IICAL CHEMISTRY | /BIOCHEMISTR | Y |
| | | | | |
| | | GLUCOSE FAS | STING (F) | |

KOS Diagnostic Lab (A Unit of KOS Healthcare)

A fasting plasma glucose level below 100 mg/dl is considered normal.
 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.
 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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Page 4 of 15





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| Test Name | | Value | Unit | Biological Reference interval |
| | | LIPID PROFIL | E : BASIC | |
| CHOLESTEROL TOTA by CHOLESTEROL OX | | 209.02 ^H | mg/dL | OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239. HIGH CHOLESTEROL: > OR = 240 |
| TRIGLYCERIDES: SER by GLYCEROL PHOSP | UM HATE OXIDASE (ENZYMATIC) | 129.37 | mg/dL | OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199. HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0 |
| HDL CHOLESTEROL (by SELECTIVE INHIBITI | | 54.01 | mg/dL | LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0 |
| LDL CHOLESTEROL: S by CALCULATED, SPE | | 129.14 | mg/dL | OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159. HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0 |
| NON HDL CHOLESTE by calculated, spe | | 155.01 ^H | mg/dL | OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189. HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0 |
| VLDL CHOLESTEROL: by CALCULATED, SPE | | 25.87 | mg/dL | 0.00 - 45.00 |
| TOTAL LIPIDS: SERUN by CALCULATED, SPE | N | 547.41 | mg/dL | 350.00 - 700.00 |
| CHOLESTEROL/HDL F by CALCULATED, SPE | RATIO: SERUM | 3.87 | RATIO | LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 |
| LDL/HDL RATIO: SER by CALCULATED, SPE | | 2.39 | RATIO | LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0 |

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Page 5 of 15





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| Test Name | | Value | Unit | Biological Reference interval |
| TRIGLYCERIDES/HD | | 2.4 ^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.

 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.
 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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HEALTHCARE & DIAGNOSTIC Dr. Yugam Chopra

| MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist CEO & Consultant Pathologist | | | | |
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| Test Name | | Value | Unit | Biological Reference interval |
| | | LIVER FUNCTION | TEST (COMPLETE) | |
| BILIRUBIN TOTAL: SI by diazotization, sf | ERUM PECTROPHOTOMETRY | 1.07 | mg/dL | INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20 |
| BILIRUBIN DIRECT (C | CONJUGATED): SERUM | 0.25 | mg/dL | 0.00 - 0.40 |

Dr. Vinay Chopra

| BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY | 0.25 | mg/dL | 0.00 - 0.40 |
|--|---------------------|-------|--------------|
| BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY | 0.82 | mg/dL | 0.10 - 1.00 |
| SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE | 42.8 | U/L | 7.00 - 45.00 |
| SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE | 59.5 ^H | U/L | 0.00 - 49.00 |
| AST/ALT RATIO: SERUM by Calculated, spectrophotometry | 0.72 | RATIO | 0.00 - 46.00 |
| ALKALINE PHOSPHATASE: SERUM by Para NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL | 171.43 ^H | U/L | 40.0 - 130.0 |
| GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by szasz, spectrophtometry | 23.49 | U/L | 0.00 - 55.0 |
| TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY | 7.56 | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM by BROMOCRESOL GREEN | 4.43 | gm/dL | 3.50 - 5.50 |
| GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY | 3.13 | gm/dL | 2.30 - 3.50 |
| A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY | 1.42 | 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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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).

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



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| | EXCELLEN | CE IN HEALTHCARE & DIAGNOSTICS |
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Dr. Vinay Chopra MD (Pathology & Microbio Chairman & Consultant Pat

: Miss. VANSHIKA

| Test Name | Value | Unit | Biological Reference interval |
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| КІ | DNEY FUNCTION TE | ST (COMPLETE) | |
| UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH) | 15.7 | mg/dL | 10.00 - 50.00 |
| CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY | 0.81 | mg/dL | 0.40 - 1.20 |
| BLOOD UREA NITROGEN (BUN): SERUM by calculated, spectrophotometry | 7.34 | mg/dL | 7.0 - 25.0 |
| BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY | 9.06 ^L | RATIO | 10.0 - 20.0 |
| UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY | 19.38 | RATIO | |
| URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE | 5.62 | mg/dL | 2.50 - 6.80 |
| CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY | 10.01 | mg/dL | 8.50 - 10.60 |
| PHOSPHOROUS: SERUM by phosphomolybdate, spectrophotometry ELECTROLYTES | 2.38 | mg/dL | 2.30 - 4.70 |
| SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE) | 140.1 | mmol/L | 135.0 - 150.0 |
| POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE) | 4.11 | mmol/L | 3.50 - 5.00 |
| CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE) ESTIMATED GLOMERULAR FILTERATION RATE | 105.07 | mmol/L | 90.0 - 110.0 |
| ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM by CALCULATED | 103.2 | | |

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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NAME

Page 9 of 15





| AGE / GENDER 25 YRS/FEMALE PATIENT ID 1604171 COLLECTED BY : REG.NO./LAB NO. :012409060058 REFERED BY : REGISTRATION DATE :06/Sep/2024 02:00 PM BARCODE NO. :01516441 COLLECTION DATE :06/Sep/2024 02:00 PM CILENT CODE :KOS DIAGNOSTIC LAB REPORTING DATE :06/Sep/2024 02:05 PM CILENT ADDRESS :6349/1, NICHOLSON ROAD, AMBALA CANTT Biological Reference interval 3. GI haemorrhage. 4 Unit Biological Reference interval 4. High protein intake. 5. Impaired renal function plus 5. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet burns, syngery, cacheska, high fever). 1. Unite reabsorption (e.g. ucter colostomy) 8. Reduced muscle mass (subnormal creatinine production) 9. Certain drugs (e.g. tetracycline, glucocorticoids) WREASED RATIO (2021) WITH ELEVATED CREATININE LEVELS: 1. Posternal azotemia uproposed on renal disease. 9. Cortain drugs (e.g. tetracycline, glucocorticoids) 1. Auste tubular necrosis. 9. Cortain drugs (urea rather than creatinine diffuses out of extracellular fluid). 5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid). 9. Inherited hyperammonemias (urea is v | | | r Chopra ogy & Microbiology) Consultant Pathologist | | gam Chopra MD (Pathology) Itant Pathologist | |
|--|---|--|--|----------------------|---|--------------------------|
| CULLECTED BY :: REG. NO./LAB NO. :: 10.2409060058 REFERRED BY :: REGISTRATION DATE : 06/Sep/2024 02:00 PM BARCODE NO. ::01516411 COLLECTION DATE :: 06/Sep/2024 02:00 PM CLIENT CODE. :: KOS DIAGNOSTIC LAB REPORTING DATE :: 06/Sep/2024 02:058PM CLIENT ADDRESS :: :: :: 06/Sep/2024 02:058PM CLIENT ADDRESS :: <th>NAME</th> <th>: Miss. VANSHIKA</th> <th></th> <th></th> <th></th> <th></th> | NAME | : Miss. VANSHIKA | | | | |
| REFEREND BY :: REGISTRATION DATE :: 06/Sep/2024 02:00 PM BARCODE NO. :::01516441 COLLECTION DATE :::06/Sep/2024 02:07PM CLIENT CODE :::050 DIAGNOSTIC LAB REPORTING DATE ::06/Sep/2024 02:07PM CLIENT ADDRESS ::6349/1, NICHOLSON ROAD, AMBALA CANTT :06/Sep/2024 02:58PM Test Name Value Unit Biological Reference interval 3. GI haemorrhage. : : : : 4. High protein intake. : : : : 5. 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 colostormy) : : : : 8. Gueded muscle mass (subnormal creatinine production) : : : : 9. Cartal drugs (e.g. tetracycline, glucocorticolds) : : : : 19. Obternal azotemia superimposed on renal disease. : : : : : 19. Cartal drugs (e.g. tetracycline, glucocorticolds) : : : : : 19. Obternal a | AGE/ GENDER | : 25 YRS/FEMALE | F | ATIENT ID | : 1604171 | |
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| CLIENT CODE: : KOS DIAGNOSTIC LAB REPORTING DATE : (c/Sep/2024 02:58PM CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT Biological Reference interval 3. GI haemorrhage. 4. High protein intake. 5. Impaired renal function plus 5. Impaired renal function plus 5. Seves 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 (BUM rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUM rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUM rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUM rises disproportionately more than creatinine) (e.g. obstructive uropathy). 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 hyperamonomemias (urea is virtually absent in blood). 7. Skapta (syntherementis) 8. Pregnano; | | : 01516441 | | | | |
| CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit Biological Reference interval 3. GI haemorrhage. 4. High protein intake. 5. Impaired renal function plus 6. Impaired renal function plus 5. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein interval 7. Urine reabsorption (e.g. ureter colostomy) 8. Reduced muscle mass (subnormal creatinine production) 9. Certain drugs (e.g. tetracycline, glucocorticoids) 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 (Suri isse disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (Suri isse disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (Suri isse disproportionately more than creatinine) (e.g. obstructive uropathy). 3. Severe liver disases. 9. Comported RATIO (>10:1) WITH DECREASED BUN : 1. Acute tubular necrosis. 9. Severe liver disease. 4. Other causes of decreased urea synthesis. 9. Comparise 5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid). 9. Inherited hyperamonomemias (urea is invitually absent in blocod). | | | | | • | |
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| 3. Gl haemorrhage. 4. High protein intake. 5. Impaired renal function plus 6. Excess protein intake or production or tissue breakdown (e.g. infection, Gl 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 (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 3. Potereal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 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 | CLIENT ADDRESS | : 6349/1, NICHOLSON RU | AD, AMBALA CAN I I | | | |
| 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. Preneal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Preneal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Preneal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Preneal azotemia superimposed on renal disease. DECREASED RATIO (-10-10) 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. | Test Name | | Value | Unit | Biological | Reference interval |
| 2. Cephalosporin therapy (interferes with creatinine measurement). ESTIMATED GLOMERULAR FILTERATION RATE: CKD STAGE DESCRIPTION G1 Normal kidney function >90 No proteinuria | Low protein diet a Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome e) Pregnancy. DECREASED RATIO (Phenacimide thera Rhabdomyolysis (r Muscular patients INAPPROPIATE RATIO Diabetic ketoacido | nd starvation. e. ccreased urea synthesis. (urea rather than creatinine monemias (urea is virtually of inappropiate antidiuretic f 10:1) WITH INCREASED CREA apy (accelerates conversion of releases muscle creatinine). who develop renal failure. 0: osis (acetoacetate causes fais | absent in blood). harmone) due to tubula TININE: of creatine to creatinine se increase in creatinine | r secretion of urea. | dologies,resulting in norma | ıl ratio when dehydratio |
| CKD STAGEDESCRIPTIONGFR (mL/min/1.73m2)ASSOCIATED FINDINGSG1Normal kidney function>90No proteinuria | 2. Cephalosporin the | rapy (interferes with creatini | | | | |
| G1 Normal kidney function >90 No proteinuria | | | ON GFR (ml | /min/1.73m2) | ASSOCIATED FINDINGS |] |
| | | | | | | 1 |
| G2 Kidney damage with >90 Presence of Protein , normal or high GFR Albumin or cast in urine | G2 | Kidney damag | je with | >90 | Presence of Protein, | 1 |

| G1 | Normal kidney function | >90 | No proteinuria |
|-----|--------------------------|--------|--------------------------|
| G2 | Kidney damage with | >90 | Presence of Protein, |
| | normal or high GFR | | 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 | |
| | | | |



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

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| | Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar | obiology) MI | m Chopra D (Pathology) nt Pathologist |
|--------------------|---|--------------------------|---|
| NAME | : Miss. VANSHIKA | | |
| AGE/ GENDER | : 25 YRS/FEMALE | PATIENT ID | : 1604171 |
| COLLECTED BY | : | REG. NO./LAB NO. | : 012409060058 |
| REFERRED BY | : | REGISTRATION DATE | : 06/Sep/2024 02:00 PM |
| BARCODE NO. | :01516441 | COLLECTION DATE | : 06/Sep/2024 02:07PM |
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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMB/ | ALA CANTT | |
| | | | |
| Test Name | | Value Unit | Biological Reference interval |

COMMENTS:

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.
 eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
 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 of CFD with the commended to measure

KOS Diagnostic Lab (A Unit of KOS Healthcare)

3. In patients, with eGFR cleaning between 45-59 minimit 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 fulfill 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



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

MBBS, MD (PATHOLOGY)

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| Dr. Vinay ChopraDr. YugarMD (Pathology & Microbiology)MDChairman & Consultant PathologistCEO & Consultant | | | (Pathology) | | |
|---|--------------------------------|------------------|---------------------------------|------------------------|---------------|
| NAME | : Miss. VANSHIKA | | | | |
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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, | | | . 00/36p/2024 03.211 W | |
| Test Name | | Value | Unit | Biological Refere | ence interval |
| | BETA H | | RINOLOGY ANTITATIVE): MATERI | ΝΔΙ | |
| SERUM | REGNANCY MATERNAL: | < 1.20 | mIU/mL | < 5.0 | |
| INTERN RETATION. | MEN: | | mIU/mI | < 2.0 | |
| NO | ON PREGNANT PRE-MENOPAUSA | L WOMEN: | mIU/ml | < 5.0 | |
| | MENOPAUSAL WOMEN | : , | mIU/mI | < 7.0 | |
| | BETA HCG EXPECTED VALUES | IN ACCORDANCE TO | O WEEKS OF GESTATIONAL | AGE | |
| | WEEKS OF GESTATION | | Unit | Value | |
| | 4-5 | | mIU/mI | 1500 -23000 | |
| | 5-6 | | mIU/ml | 3400 - 135300 | |
| | 6-7 | | mIU/ml | 10500 - 161000 | |
| | 7-8 | | mIU/mI mIU/mI | 18000 - 209000 | |
| | 9-10 | | miU/mi mIU/mi | 37500 - 219000 | |
| | 10-11 | | miU/mi mIU/mi | 42800 - 218000 | |
| | 10-11 | | mIU/ml | 33700 - 218700 | |
| | 11-12 | | miU/mi mIU/mi | 21800 - 193200 | |
| | | | | 20300 - 166100 | |
| | 13-14 | | mIU/mI | 15400 - 190000 | |
| | 2rd TRIMESTER 3rd TRIMESTER | | mIU/ml | 2800 - 176100 | |
| | 3IU IKIIVIESTER | | mlU/ml | 2800 - 144400 | |





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





| | Dr. Vinay Chopr MD (Pathology & Micr Chairman & Consultar | robiology) M | m Chopra D (Pathology) nt Pathologist |
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| | | | |
| Test Name | | Value Unit | Biological Reference interval |

1.hCG is a Glycoprotein with alpha and beta chains. Beta subunit is specific to hCG.

2.1t is largely secreted by trophoblastic tissue. Small amounts may be secreted by fetal tissues and by the adult ant pituitary.

INCREASED :

1.Pregnancy

2.Gestationalsite & Non gestational trophoblastic neoplasia.

3.In mixed germ cell tumors

SIGNIFICANTLY HIGHER THAN EXPECTED LEVEL:

1.Multiple pregnancies & High risk molar pregnancies are usually associated with levels in excess of one lac mIU/mI. 2.Erythroblastosis fetalis & Downs syndrome.

DECREASED:

1. Ectopic pregnancy

2.Intra-uterine fetal death.

NOTE:

1. The test becomes positive 7-9 days after the midcycle surge that precedes ovulation (time of blastocyst implantation). Blood levels rise rapidly after this and double every 1.4 - 2 days. 2. Peak values are usually seen at 60-80 days of LMP. The levels then begin to taper and ebb out around the 20th week. These low levels are then

maintained throughout pregnancy.

3. Doubling time: In intra-uterine pregnancy, serum hCG levels increase by approximately 66% every 48 hrs. Inappropriately rising serum hCG levels are suggestive of dying or ectopic pregnancy.

CAUTION:

Spuriously high levels (Phantom hCG) may be seen in presence of heterophilic antibodies (found in some normal people). If persistently raised levels are seen in a non-pregnant patient with no evidence of other obvious causes for such an increase a urine hCG assay may help confirm presence of the heterophile antibodies.





DR.VINAY CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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







| Dr. Vinay Che MD (Pathology & Chairman & Cons | | | Dr. Yugam MD CEO & Consultant | (Pathology) |
|---|--|---|-------------------------------------|-------------------------------|
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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, | AMBALA CANTT | | |
| | | | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | | CLINICAL PAT | HOLOGY | |
| | | OUTINE & MICROS | COPIC EXAMINAT | ION |
| PHYSICAL EXAMINA | | | | |
| QUANTITY RECIEVEI | | 10 | ml | |
| | by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY COLOUR by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY | | | |
| | | | | PALE YELLOW |
| TRANSPARANCY | TANCE SPECTROPHOTOMETRT | HAZY | | CLEAR |
| - | TANCE SPECTROPHOTOMETRY | | | |
| SPECIFIC GRAVITY | TANCE SPECTROPHOTOMETRY | >=1.030 | | 1.002 - 1.030 |
| CHEMICAL EXAMINA | | | | |
| REACTION | | ACIDIC | | |
| | TANCE SPECTROPHOTOMETRY | Newsters | | |
| PROTEIN by DIP STICK/REFLEC | TANCE SPECTROPHOTOMETRY | Negative | | NEGATIVE (-ve) |
| SUGAR | | Negative | | NEGATIVE (-ve) |
| | TANCE SPECTROPHOTOMETRY | <=5.0 | | 5.0 - 7.5 |
| pH by DIP STICK/REFLEC | TANCE SPECTROPHOTOMETRY | <=0.0 | | 5.0 - 7.5 |
| BILIRUBIN | | Negative | | NEGATIVE (-ve) |
| by DIP STICK/REFLEC | TANCE SPECTROPHOTOMETRY | Negative | | NEGATIVE (-ve) |
| | TANCE SPECTROPHOTOMETRY. | , i i i i i i i i i i i i i i i i i i i | | |
| | TANCE SPECTROPHOTOMETRY | Normal | EU/dL | 0.2 - 1.0 |
| KETONE BODIES | ANUCE OFEUI KUPHUI UMEI RY | Negative | | NEGATIVE (-ve) |
| by DIP STICK/REFLEC | TANCE SPECTROPHOTOMETRY | | | |
| BLOOD | TANCE SPECTROPHOTOMETRY | Negative | | NEGATIVE (-ve) |
| ASCORBIC ACID | | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| | TANCE SPECTROPHOTOMETRY | | | 、 <i>、</i> |

MICROSCOPIC EXAMINATION



DR.VINAY CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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

Page 14 of 15





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



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

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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AM | /IBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| RED BLOOD CELLS (F | RBCs) CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | /HPF | 0 - 3 |
| PUS CELLS by MICROSCOPY ON C | CENTRIFUGED URINARY SEDIMENT | 1-2 | /HPF | 0 - 5 |
| | | | | |

| by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | | | |
|---|----------------|------|----------------|
| EPITHELIAL CELLS | 3-4 | /HPF | ABSENT |
| by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT CRYSTALS | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | ζ, γ | | |
| by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | ABSENT | | ABSENT |

*** End Of Report ***





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