

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



| | Dr. Vinay Chopr MD (Pathology & Micr Chairman & Consultar | robiology) | | (Pathology) |
|-------------------------------------|------------------------------------------------------------------------|------------|--------------------------|--------------------------------------|
| IAME | : Mr. RAKESH RASTOGI | | | |
| AGE/ GENDER | : 68 YRS/MALE | | PATIENT ID | : 1742161 |
| COLLECTED BY | : | | REG. NO./LAB NO. | : 012502010038 |
| REFERRED BY | : | | REGISTRATION DATE | : 01/Feb/2025 01:08 PM |
| BARCODE NO. | :01524771 | | COLLECTION DATE | :01/Feb/202501:14PM |
| CLIENT CODE. CLIENT ADDRESS | : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMB | ALA CANTT | REPORTING DATE | : 01/Feb/2025 02:02PM |
| Fest Name | | Value | Unit | Biological Reference interval |
| | SWAST | HYA WE | LLNESS PANEL: 1.3 | 2 |
| | COM | PLETE BL | OOD COUNT (CBC) | |
| RED BLOOD CELLS | (RBCS) COUNT AND INDICES | | | |
| HAEMOGLOBIN (H | B) | 14.2 | gm/dL | 12.0 - 17.0 |
| by CALORIMETRIC RED BLOOD CELL (| RBC) COUNT | 4.71 | Millions | /cmm 3.50 - 5.00 |
| by HYDRO DYNAMIC F | OCUSING, ELECTRICAL IMPEDENCE | 42.4 | % | 40.0 - 54.0 |
| by CALCULATED BY A | UTOMATED HEMATOLOGY ANALYZER | | | |
| | AR VOLUME (MCV) UTOMATED HEMATOLOGY ANALYZER | 90 | fL | 80.0 - 100.0 |
| AEAN CORPUSCUL | AR HAEMOGLOBIN (MCH) | 30.2 | pg | 27.0 - 34.0 |
| MEAN CORPUSCUL | UTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC) | 33.5 | g/dL | 32.0 - 36.0 |
| RED CELL DISTRIB | UTOMATED HEMATOLOGY ANALYZER UTION WIDTH (RDW-CV) | 14.3 | % | 11.00 - 16.00 |
| | utomated hematology analyzer UTION WIDTH (RDW-SD) | 48.2 | fL | 35.0 - 56.0 |
| by CALCULATED BY A | UTOMATED HEMATOLOGY ANALYZER | | | |
| MENTZERS INDEX | | 19.11 | RATIO | BETA THALASSEMIA TRAIT: < 13.0 |
| | | | | IRON DEFICIENCY ANEMIA: |
| GREEN & KING IND | DEX | 27.37 | RATIO | >13.0 BETA THALASSEMIA TRAIT:<= |
| by CALCULATED | | | | 65.0 |
| | | | | IRON DEFICIENCY ANEMIA: > 65.0 |
| WHITE BLOOD CE | LLS (WBCS) | | | |
| TOTAL LEUCOCYTE | COUNT (TLC) / by sf cube & microscopy | 6190 | /cmm | 4000 - 11000 |
| • | LOOD CELLS (nRBCS) | NIL | | 0.00 - 20.00 |
| | RT HEMATOLOGY ANALYZER | | % | < 10 % |
| by AUTOMATED 6 PAF | LOOD CELLS (nRBCS) % | NIL | % | < 10 % |





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Dr. Yugam Chopra

MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. RAKESH RASTOGI AGE/ GENDER : 68 YRS/MALE **PATIENT ID** :1742161 **COLLECTED BY** :012502010038 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** :01/Feb/2025 01:08 PM **BARCODE NO.** :01524771 **COLLECTION DATE** :01/Feb/202501:14PM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :01/Feb/202502:02PM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC)** NEUTROPHILS 88^H % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 5^L % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS OL % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 7 % 2 - 12by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE LEUKOCYTES (WBC) COUNT** ABSOLUTE NEUTROPHIL COUNT 5447 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 800 - 4900 310^L /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY 0^L ABSOLUTE EOSINOPHIL COUNT /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 433 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 207000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.24 % 0.10 - 0.36 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) 73000 30000 - 90000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) 35.6 % 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) % 16.215.0 - 17.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

Dr. Vinay Chopra



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| CLIENT ADDRESS | : 6349/1, NICI | HOLSON ROAD, A | MBALA CANTT | | |
| Test Name | | | Value | Unit | Biological Reference interval |
| immune disease', but 2. An ESR can be affe as C-reactive protein | does not tell the ected by other co | e health practition nditions besides | ner exactly where inflammation. Fo | e the inflammation is in the r this reason, the ESR is ty | |
| mmune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus eryth CONDITION WITH LO A low ESR can be see polycythaemia), sign as sickle cells in sick NOTE: 1. ESR and C - reactiv 2. Generally, ESR doe 3. CRP is not affected | does not tell the ected by other co be used to moni- ematosus W ESR en with condition hificantly high wh le cell anaemia) re protein (C-RP) es not change as by as many othe | e health practition nditions besides tor disease activi s that inhibit the hite blood cell co also lower the ES are both markers rapidly as does C er factors as is ESF | ner exactly where inflammation. Fo ty and response to normal sediment unt (leucocytosis R. of inflammation. RP, either at the 2, making it a bet | e the inflammation is in the r this reason, the ESR is ty to therapy in both of the a tation of red blood cells, s) , and some protein abno | e body or what is causing it. pically 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 s it resolves. |





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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | REPO | DRTING DATE | : 01/Feb/2025 03:29PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAI | D, AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | CLINI | ICAL CHEMISTRY | /BIOCHEMISTI | RY |
| | | GLUCOSE FAST | ГING (F) | |
| GLUCOSE FASTIN | G (F): PLASMA se - peroxidase (god-pod) | 130.68 ^H | mg/dL | NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 |

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. 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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Page 4 of 15





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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | R | EPORTING DATE | : 01/Feb/2025 04:03PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON RO | AD, AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | | LIPID PROF | TI F · BASIC | |
| CUALESTEDAL TA | | 134.32 | | OPTIMAL: < 200.0 |
| CHOLESTEROL TOT by CHOLESTEROL OX | | 134.32 | mg/dL | BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0 |
| TRIGLYCERIDES: S. by GLYCEROL PHOSP | ERUM HATE OXIDASE (ENZYMATIC) | 57.32 | mg/dL | OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0 |
| HDL CHOLESTERO | L (DIRECT): SERUM | 35.9 | mg/dL | LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0 |
| LDL CHOLESTEROI by CALCULATED, SPE | | 86.96 | mg/dL | OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0 |
| NON HDL CHOLEST by calculated, spe | | 98.42 | mg/dL | VERT HIGH. > OR = 190.0 OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0 |
| VLDL CHOLESTERC | | 11.46 | mg/dL | 0.00 - 45.00 |
| TOTAL LIPIDS: SER | UM | 325.96 ^L | mg/dL | 350.00 - 700.00 |
| CHOLESTEROL/HD by CALCULATED, SPE | L RATIO: SERUM | 3.74 | RATIO | LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 |



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Page 5 of 15





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| Test Name | | Value | Unit | Biological Reference interval |
| LDL/HDL RATIO: S by CALCULATED, SPE | | 2.42 | RATIO | LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0 |
| TRIGLYCERIDES/H by CALCULATED, SPE INTERPRETATION: | IDL RATIO: SERUM | 1.6 ^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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| Test Name | | Value | Unit | Biological Reference interval |
| | LIVER | FUNCTION 7 | FEST (COMPLETE) | |
| BILIRUBIN TOTAL by DIAZOTIZATION, SI | : SERUM PECTROPHOTOMETRY | 0.66 | mg/dL | INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20 |
| | Г (CONJUGATED): SERUM spectrophotometry | 0.16 | mg/dL | 0.00 - 0.40 |
| BILIRUBIN INDIRE by CALCULATED, SPE | CCT (UNCONJUGATED): SERUM | 0.5 | mg/dL | 0.10 - 1.00 |
| SGOT/AST: SERUM by IFCC, WITHOUT PY | [/RIDOXAL PHOSPHATE | 32.8 | U/L | 7.00 - 45.00 |
| SGPT/ALT: SERUM by IFCC, WITHOUT PY | [/RIDOXAL PHOSPHATE | 32.9 | U/L | 0.00 - 49.00 |
| AST/ALT RATIO: S by CALCULATED, SPE | | 1 | RATIO | 0.00 - 46.00 |
| ALKALINE PHOSPI by PARA NITROPHEN PROPANOL | HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL | 97.03 | U/L | 40.0 - 130.0 |
| GAMMA GLUTAMY by SZASZ, SPECTRO | L TRANSFERASE (GGT): SERUM | 25.63 | U/L | 0.00 - 55.0 |
| TOTAL PROTEINS: by BIURET, SPECTRO | | 6.75 | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM by BROMOCRESOL G | | 4.66 | gm/dL | 3.50 - 5.50 |
| GLOBULIN: SERUN by CALCULATED, SPE | 1 | 2.09 ^L | gm/dL | 2.30 - 3.50 |
| A : G RATIO: SERUI by CALCULATED, SPE | M | 2.23 ^H | 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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| Test Name | | Value | Unit | Biological Reference interva |
| | KIDNE | EY FUNCTION | FEST (COMPLETE) | |
| UREA: SERUM | IATE DEHYDROGENASE (GLDH) | 53.22 ^H | mg/dL | 10.00 - 50.00 |
| CREATININE: SERI | UM | 1.42 ^H | mg/dL | 0.40 - 1.40 |
| - | ROGEN (BUN): SERUM | 24.87 | mg/dL | 7.0 - 25.0 |
| | ROGEN (BUN)/CREATININE | 17.51 | RATIO | 10.0 - 20.0 |
| by CALCULATED, SPE | | | | |
| UREA/CREATININ by CALCULATED, SPE | | 37.48 | RATIO | |
| URIC ACID: SERUM | | 7.14 | mg/dL | 3.60 - 7.70 |
| by URICASE - OXIDAS CALCIUM: SERUM | SE PEROXIDASE | 9.28 | mg/dL | 8.50 - 10.60 |
| by ARSENAZO III, SPE | | 0.00 | | 9 90 4 70 |
| PHOSPHOROUS: SE by PHOSPHOMOLYBL | DATE, SPECTROPHOTOMETRY | 2.98 | mg/dL | 2.30 - 4.70 |
| <u>ELECTROLYTES</u> | | | | |
| SODIUM: SERUM by ISE (ION SELECTIV | | 139.6 | mmol/L | 135.0 - 150.0 |
| POTASSIUM: SERU | M | 4.6 | mmol/L | 3.50 - 5.00 |
| by ISE (ION SELECTIV CHLORIDE: SERUM | 1 | 104.7 | mmol/L | 90.0 - 110.0 |
| by ISE (ION SELECTIV ESTIMATED GLOM | TERULAR FILTERATION RATE | | | |
| | ERULAR FILTERATION RATE | 53.8 | | |

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.

3. GI haemorrhage.



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Page 9 of 15

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





| VAME : Mr. RAKESH RASTOGI XGE/ GENDER : 68 YRS/MALE PATIENT ID : 1742161 COLLECTED BY : REG. NO./LAB NO. : 012502010038 REFERED BY : REGISTRATION DATE : 01/Feb/2025 01:08 PM BARCODE NO. : 01524771 COLLECTION DATE : 01/Feb/2025 01:04 PM BARCODE NO. : 01524771 COLLECTION DATE : 01/Feb/2025 04:49 PM CLIENT CODE : KOS DIAGNOSTIC LAB REPORTING DATE : 01/Feb/2025 04:49 PM CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT Biological Reference intere Test Name Value Unit Biological Reference intere 4. High protein intake. : Numere absorption (e.g. ureter colostomy) : Keess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein did 7. Urine reabsorption (e.g. ureter colostomy) : Reduced muscle mass (subnormal creatinine production) : Portain azotemia superimposed on rend disease. 9. Certain days (e.g. totraycift) : Reduced muscle mass (subnormal creatinine of lesses. : Portain azotemia superimposed on rend disease. 9. Certain days (lesses) : Reduced muscle mass (subnormal creatinine of lesses. : Reduced muscle mass (urea synthesis. </th <th></th> <th></th> <th>Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta</th> <th>robiology)</th> <th></th> <th>am Chopra MD (Pathology) tant Pathologist</th> <th></th> | | | Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta | robiology) | | a m Chopra MD (Pathology) tant Pathologist | |
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| COLLECTED BY :::::::::::::::::::::::::::::::::::: | IAME | : Mr. RAKES | H RASTOGI | | | | |
| Reference by: :::::::::::::::::::::::::::::::::::: | AGE/ GENDER | : 68 YRS/MAJ | LE | PAT | IENT ID | : 1742161 | |
| REFERED BY: E. EXCLAPTION DATE :01/Feb/2025 01:08 PM. SARCODE NO. :01524771 COLLECTION DATE :01/Feb/2025 01:14PM. SLIENT CODE :KOS DIAGNOSTIC LAB REPORTING DATE :01/Feb/2025 04:49PM SLIENT ADDRESS :6349/1, NICHOLSON ROAD, AMBALA CANTT Biological Reference inter 1. Hipp protein intake. | COLLECTED BY | : | | REG | NO./LAB NO. | : 0125020100 | 38 |
| BARCODE NO. : 11524771 COLLECTION DATE : 01/Feb/2025 01:14PM CLIENT CODE : 1000 COLLECTION CALL AND COLLECTION DATE : 01/Feb/2025 04:49PM LIENT ADDREST : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Collection Intake. 1 High protein Intake. 2 Impaired renal function plus 3 Excess protein intake or production or tissue breakdown (e.g. infection, Gi bleeding, thyrotoxicosis, Cushing's syndrome, high protein dia 1. High protein intake or production or tissue breakdown (e.g. infection, Gi bleeding, thyrotoxicosis, Cushing's syndrome, high protein dia 1. Surgery, cachesia, high fever). 3. Reduced muscle mass (subnormal creatinine production) 4. Reduced muscle mass (subnormal creatinine production) 5. Certain drugs (e.g. Lettarcycline, glucocorticoids) NCREASED RATIO (-20:1) WITH LEVATED CREATININE LEVELS: 1. Postrenal azotemia Sugering with the Startage | | | | | | | |
| Element CODE KEND STACLAS REPORTING DATE OIL/Feb/2025 04:49PM SILENT ADDRESS :: 6349/1, NICHOLSON ROAD, AMBALA CANT Test Name Value Unit Biological Reference interent I. High protein intake. : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : : <td></td> <td>· · 01594771</td> <td></td> <td></td> <td></td> <td></td> <td></td> | | · · 01594771 | | | | | |
| CLENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTS Test Name Value Unit Biological Reference intel 4. High protein intake. Impaired renal function plus 5. Excess protein intake or production or tissue breakdown (e.g. infection, Gi bleeding, thyrotoxicosis, Cushing's syndrome, high protein die | | | | | | | |
| Test Name Value Unit Biological Reference inter 4. High protein intake. Impaired renal function plus Stexess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein die Jurns, surgery, cachexia, high fever). Iver reabsorption (e.g. ureter colostomy) 9. Beduced muscle mass (subnormal creatinine production) Octain drugs (e.g. tetracycline, glucocorticoids) NCREASED RATIO (>201) WITH ELEVATED CREATININE LEVELS: 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). 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). 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). Preventive disease. 3. Obstructive disease. Devention distanvation. Severe liver disease. 4. Other causes of decreased urea synthesis. Severe liver disease. Severe liver disease. 9. Other causes of diadysis (urea is virtually absent in blood). Shalf (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea. 3. Pregnancy. <td></td> <td></td> <td></td> <td></td> <td>URTING DATE</td> <td>: 01/FeD/2025 (</td> <td>04:49PM</td> | | | | | URTING DATE | : 01/FeD/2025 (| 04:49PM |
| I High protein intake. 9: Impaired renal function plus 9: Excess protein intake or production or tissue breakdown (e.g. infection, Gl bleeding, thyrotoxicosis, Cushing's syndrome, high protein die jurns, surgery, cachexia, high fever). 7. Urine reabsorption (e.g. ureter colostomy) 8: Reduced muscle mass (subnormal creatinine production). 0: Certain drugs (e.g. tetracycline, glucocorticoids) NCREASED 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 (Subin rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2: Prerenal azotemia (Subin rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2: Prerenal azotemia (Subin rises disproportionately more than creatinine) (e.g. obstructive uropathy). 3: Aret tubular necrosis. 4: Outper disease. 4: Other causes of decreased urea synthesis. 5: Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid). 5: Inherited hyperammonemias (urea is virtually absent in blood). 7: SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea. 8: Pregnancy. 9: Cepanace. 0: A | CLIENT ADDRESS | : 6349/1, NIC | CHOLSON ROAD, AMB | ALA CANTT | | | |
| 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 die 1. Surgery, cachexia, high fever). 7. Urine reabsorption (e.g. ureter colostomy) 8. Reduced muscle mass (subnormal creatinine production) 9. Certain drugs (e.g. lettracycline, glucocorticoids) 1. NOREASED 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. Perenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Perenal azotemia superimposed on renal disease. 2. Dev 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). 5. Inherited hyperammonemias (urea is virtually absent in blood). 7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea. 3. Pregnancy. 2. 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. 1. NAPROPAITE RATIO: 1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehy should produce an increased BUN/creatinine measurement). 5. STIMATE OCMOMENUAR HITTERTATION RATE: 5. CKD STAGE 5. DESCRIPTION 6. Normal kidney function 6. Albumin or cast in urine 6. G.2 Kidney damage with 90 7. Presence of Protein, Albumin or cast in urine 6. G.3 Mild decrease in GFR 6. 0-89 6. G.3b Moderate decrease in GFR 6. 0-89 6. G.3b Moderate decrease in GFR 6. 0-99 | Fest Name | | | Value | Unit | Biolog | gical Reference interva |
| G2Kidney damage with normal or high GFR>90Presence of Protein , Albumin or cast in urineG3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59 | 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia | tetracycline, gl 20:1) WITH ELEV a (BUN rises disp | l creatinine production ucocorticoids) ATED CREATININE LEV proportionately more | ELS: | e.g. obstructive ure | opathy). | |
| G2Kidney damage with normal or high GFR>90Presence of Protein , Albumin or cast in urineG3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59 | 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<' 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (<' 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL | tetracycline, gl tetracycline, gl to:1) WITH ELEV a (BUN rises disp superimposed to:1) WITH DECI osis. and starvation. e. creased urea sy furea rather that monemias (urea finappropiate to:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes | I creatinine production ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. REASED BUN : an creatinine diffuses of a is virtually absent in antidiuretic harmone) REASED CREATININE: conversion of creatin creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measu DN RATE: | ELS: than creatinine) (e but of extracellula blood). due to tubular se e to creatinine). se in creatinine wi urement). | r fluid). cretion of urea. th certain method | dologies,resulting in no | |
| normal or high GFRAlbumin or cast in urineG3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59 | Certain drugs (e.g., NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia CECREASED RATIO (<' Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome of Barden and the second Carl Acute tubular necros Pregnancy. Pregnancy. Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin there STIMATED GLOMERL CKD STAGE | tetracycline, gl tetracycline, gl to:1) WITH ELEV a (BUN rises disp superimposed to:1) WITH DECI osis. and starvation. e. creased urea sy furea rather that monemias (urea finappropiate to:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes JLAR FILTERATIO | I creatinine production ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. REASED BUN : an creatinine diffuses of the ais virtually absent in antidiuretic harmone) REASED CREATININE: conversion of creatin creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measu DN RATE: DESCRIPTION | ELS: than creatinine) (e blood). due to tubular se e to creatinine). se in creatinine wi urement). | r fluid). cretion of urea. th certain method | dologies,resulting in no | |
| G3b Moderate decrease in GFR 30-59 | Certain drugs (e.g., NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia CECREASED RATIO (<' Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. PCEREASED RATIO (Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther ESTIMATED GLOMERL G1 | tetracycline, gl tetracycline, gl to:1) WITH ELEV a (BUN rises disp superimposed to:1) WITH DECI osis. and starvation. e. creased urea sy furea rather that monemias (urea finappropiate to:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes JLAR FILTERATIC | I creatinine production ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. REASED BUN : an creatinine diffuses of a is virtually absent in antidiuretic harmone) REASED CREATININE: conversion of creatin creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measu DN RATE: DESCRIPTION rmal kidney function | ELS: than creatinine) (e blood). due to tubular se e to creatinine). se in creatinine wi urement). GFR (mL/mi >9 | r fluid). cretion of urea. th certain method <u>n/1.73m2)</u> | dologies,resulting in no ASSOCIATED FINDING No proteinuria | S |
| | P. Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia PCREASED RATIO (Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome of Pregnancy. PCREASED RATIO (Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 | tetracycline, gl tetracycline, gl tetracycline, gl superimposed (0:1) WITH DECI osis. Ind starvation. e. creased urea sy furea rather that monemias (urea of inappropiate (urea rather that monemias (urea of inappropiate (urea rather that monemias (urea finappropiate (urea rather that monemias (urea finappropiate (urea rather that monemias (urea finappropiate (urea rather that monemias (urea finappropiate) (urea rather that monemias (urea finappropiate) (urea rather that monemias (urea finappropiate) (urea rather that monemias (urea finappropiate) (urea finappropiate) (urea finappropiate) (urea finappropiate) (urea finappropiate) (urea finappropiate) (urea finappropiate) (urea | I creatinine production ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. REASED BUN : an creatinine diffuses of the ais virtually absent in antidiuretic harmone) REASED CREATININE: to conversion of creatin creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measu DI RATE: DESCRIPTION trmal kidney function idney damage with normal or high GFR | ELS: than creatinine) (e but of extracellula blood). due to tubular se e to creatinine). se in creatinine wi urement). GFR (mL/mi >9 >9 | r fluid). cretion of urea. th certain method n/1.73m2) 0 | dologies,resulting in no ASSOCIATED FINDING No proteinuria Presence of Protein , | S |
| | Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERL G1 G2 | tetracycline, gl tetracycline, gl tetracycline, gl tetracycline, gl superimposed to:1) WITH DECI osis. and starvation. e. creased urea sy furea rather tha monemias (urea of inappropiate to:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes JLAR FILTERATIO No K n No | I creatinine production ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. REASED BUN : an creatinine diffuses of the ais virtually absent in antidiuretic harmone) REASED CREATININE: conversion of creatin creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measu DI RATE: DESCRIPTION rmal kidney function idney damage with normal or high GFR NI decrease in GFR | ELS: than creatinine) (e but of extracellula blood). due to tubular se e to creatinine). se in creatinine wi urement). GFR (mL/mi >9 >9 60 - | r fluid). cretion of urea. th certain method n/1.73m2) 0 0 89 | dologies,resulting in no ASSOCIATED FINDING No proteinuria Presence of Protein , | S |
| G4 Severe decrease in GFR 15-29 G5 Kidney failure <15 | P. Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Perenal azotemia Perenal azotemia DECREASED RATIO (Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome of Pregnancy. DECREASED RATIO (Nabedomyolysis (r Rabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERL G1 G2 G3a G3b | tetracycline, gl tetracycline, gl tetracycline, gl tetracycline, gl superimposed to:1) WITH DECI osis. ad starvation. e. creased urea sy furea rather that monemias (urea of inappropiate to:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes JLAR FILTERATION NO NO NO NO NO NO NO | I creatinine production ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. REASED BUN : an creatinine diffuses of the ais virtually absent in antidiuretic harmone) REASED CREATININE: conversion of creatin creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measu DI RATE: DESCRIPTION rmal kidney function (idney damage with normal or high GFR hild decrease in GFR derate decrease in GFR | ELS: than creatinine) (e blood). due to tubular se e to creatinine). se in creatinine wi urement). GFR (mL/mi >9 >9 60 - 30- | r fluid). cretion of urea. th certain method n/1.73m2) 0 0 89 59 | dologies,resulting in no ASSOCIATED FINDING No proteinuria Presence of Protein , | S |





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









| | Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Patholog | | (Pathology) |
|---------------------|--------------------------------------------------------------------------------------------|--------------------------|--------------------------------------|
| NAME | : Mr. RAKESH RASTOGI | | |
| AGE/ GENDER | : 68 YRS/MALE | PATIENT ID | : 1742161 |
| COLLECTED BY | : | REG. NO./LAB NO. | : 012502010038 |
| REFERRED BY | : | REGISTRATION DATE | : 01/Feb/2025 01:08 PM |
| BARCODE NO. | : 01524771 | COLLECTION DATE | : 01/Feb/2025 01:14PM |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | REPORTING DATE | : 01/Feb/2025 04:49PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMBALA CANT | Т | |
| | | | |
| 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

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)







| | | Chopra gy & Microbiology) Consultant Pathologis | ME | Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist | | |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--|--|
| NAME | : Mr. RAKESH RASTOGI | | | | | |
| AGE/ GENDER | : 68 YRS/MALE | | PATIENT ID | : 1742161 | | |
| COLLECTED BY | : | | REG. NO./LAB NO. | : 012502010038 | | |
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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 01/Feb/2025 03:47PM | | |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROA | AD, AMBALA CANTT | 2 | | | |
| Test Name | | Value | Unit | Biological Reference interval | | |
| | | ENDOC | RINOLOGY | | | |
| | | THYROID FUNC | CTION TEST: TOTAL | | | |
| | | | | | | |
| | | 1.074 | ng/mL | 0.35 - 1.93 | | |
| by CMIA (CHEMILUMIN THYROXINE (T4): S | NESCENT MICROPARTICLE IMMUN SERUM | NOASSAY) 7.51 | ng/mL µgm/dI | | | |
| by CMIA (CHEMILUMIN THYROXINE (T4): S by CMIA (CHEMILUMIN THYROID STIMULA | NESCENT MICROPARTICLE IMMUN SERUM NESCENT MICROPARTICLE IMMUN ATING HORMONE (TSH): SI | NOASSAY) 7.51 NOASSAY) ERUM 1.993 | C C | 4.87 - 12.60 | | |
| by CMIA (CHEMILUMIN THYROXINE (T4): S by CMIA (CHEMILUMIN THYROID STIMULA by CMIA (CHEMILUMIN 3rd GENERATION, ULT | VESCENT MICROPARTICLE IMMUN SERUM VESCENT MICROPARTICLE IMMUN ATING HORMONE (TSH): SI VESCENT MICROPARTICLE IMMUN | NOASSAY) 7.51 NOASSAY) ERUM 1.993 | µgm/dI | 4.87 - 12.60 | | |
| by CMIA (CHEMILUMIN THYROXINE (T4): S by CMIA (CHEMILUMIN THYROID STIMULA by CMIA (CHEMILUMIN 3rd GENERATION, ULT <u>INTERPRETATION</u> : | VESCENT MICROPARTICLE IMMUN SERUM VESCENT MICROPARTICLE IMMUN ATING HORMONE (TSH): SI VESCENT MICROPARTICLE IMMUN RASENSITIVE | NOASSAY) 7.51 NOASSAY) ERUM NOASSAY) | μgm/dI μIU/mI | 4.87 - 12.60 0.35 - 5.50 | | |
| by CMIA (CHEMILUMIN THYROXINE (T4): S by CMIA (CHEMILUMIN THYROID STIMULA by CMIA (CHEMILUMIN 3rd GENERATION, ULT INTERPRETATION: TSH levels are subject to a day has influence on the triiodothyronine (T3).Fai | VESCENT MICROPARTICLE IMMUN SERUM VESCENT MICROPARTICLE IMMUN ATING HORMONE (TSH): SI VESCENT MICROPARTICLE IMMUN RASENSITIVE circadian variation, reaching peak la measured serum TSH concentration ilure at any level of regulation of th | NOASSAY) 7.51 NOASSAY) ERUM 1.993 NOASSAY) evels between 2-4 a.m ai s. TSH stimulates the pr | μgm/dI μIU/mI and at a minimum between 6-10 roduction and secretion of the p | 4.87 - 12.60 | | |
| by CMIA (CHEMILUMIN THYROXINE (T4): S by CMIA (CHEMILUMIN THYROID STIMULA by CMIA (CHEMILUMIN 3rd GENERATION, ULT INTERPRETATION: TSH levels are subject to a day has influence on the triiodothyronine (T3).Fai | VESCENT MICROPARTICLE IMMUN SERUM VESCENT MICROPARTICLE IMMUN ATING HORMONE (TSH): SI VESCENT MICROPARTICLE IMMUN RASENSITIVE circadian variation, reaching peak lo measured serum TSH concentration | NOASSAY) 7.51 NOASSAY) ERUM 1.993 NOASSAY) evels between 2-4 a.m ai s. TSH stimulates the pr | μgm/dI μIU/mI and at a minimum between 6-10 roduction and secretion of the p | 4.87 - 12.60 0.35 - 5.50 <i>orm. The variation is of the order of 50%.Hence time of th</i> netabolically active hormones, thyroxine (T4)and | | |
| THYROXINE (T4): S by CMIA (CHEMILUMIN THYROID STIMULA by CMIA (CHEMILUMIN 3rd GENERATION, ULT INTERPRETATION: TSH levels are subject to day has influence on the trilodothyronine (T3).Fai overproduction(hyperthy | VESCENT MICROPARTICLE IMMUN SERUM VESCENT MICROPARTICLE IMMUN ATING HORMONE (TSH): SI VESCENT MICROPARTICLE IMMUN RASENSITIVE circadian variation, reaching peak le measured serum TSH concentration ilure at any level of regulation of th yroidism) of T4 and/or T3. | VOASSAY) 7.51 VOASSAY) ERUM 1.993 VOASSAY) evels between 2-4 a.m al s. TSH stimulates the pr he hypothalamic-pituita | μgm/dI μIU/mI and at a minimum between 6-10 roduction and secretion of the ry-thyroid axis will result in eith | 4.87 - 12.60 0.35 - 5.50 om. The variation is of the order of 50%.Hence time of th netabolically active hormones, thyroxine (T4)and her underproduction (hypothyroidism) or | | |

| 111 | <i>ι</i> ιτΔ | TIO | NS:- |
|-----|--------------|-----|------|

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

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

Increased

Normal or High Normal

Reduced (at times undetectable)

Reduced

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 (e.g.: phenytoin , salicylates).

3. Serum T4 levels 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 hypothyroidism, 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 |
| 6 - 12 Months | 0.74 - 2.40 | 6 - 12 Months | 7.10 - 16.16 | 6 – 12 Months | 0.70 - 7.00 |

Increased

Normal or High Normal





CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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







| | | Dr. Vinay Ch MD (Pathology & Chairman & Con | | | ugam Chopi MD (Patholog ultant Patholog | gy) | |
|--------------------|-------------|---------------------------------------------------|--------------|-------------------------------------------------|-----------------------------------------------|--------------------------------------|--|
| NAME | : Mr. RAKE | SH RASTOGI | | | | | |
| AGE/ GENDER | : 68 YRS/M | : 68 YRS/MALE | | PATIENT ID | : 1742 | 161 | |
| COLLECTED BY | : | : | | REG. NO./LAB NO. : 01 | | 012502010038 | |
| REFERRED BY | : | : | | REGISTRATION DATE : 01/Feb/2025 01:08 PM | | eb/2025 01:08 PM | |
| BARCODE NO. | :01524771 | : 01524771 | | COLLECTION DATE : 01/Feb/2025 01:14Pl | | eb/2025 01:14PM | |
| CLIENT CODE. | : KOS DIAG | : KOS DIAGNOSTIC LAB | | REPORTING DATE | :01/F | eb/2025 03:47PM | |
| CLIENT ADDRESS | : 6349/1, N | ICHOLSON ROAD, | AMBALA CANTT | | | | |
| | | | | | | | |
| Test Name | | | Value | Unit | t | Biological Reference interval | |
| 1 - 10 Years | 0.92 - 2.28 | 1 - 10 Years | 6.00 - 13.80 | 1 – 10 Years | 0.60 - 5.50 | | |

| 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 | |
| | RECOM | MENDATIONS OF TSH LE | VELS DURING PREGN | IANCY (µIU/mL) | | |
| 1st Trimester | | | | 0.10 - 2.50 | | |
| | 2nd Trimester | | | 0.20 - 3.00 | | 1 |
| 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, iodine 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 goiter & Thyroiditis.

2. Over replacement of thyroid hormone in treatment of hypothyroidism.

3. Autonomously functioning Thyroid adenoma

4. Secondary pituitary or hypothalamic 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





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







| | Dr. Vinay Chopra MD (Pathology & Microbiole Chairman & Consultant Path | | Dr. Yugam MD O & Consultant | (Pathology) |
|------------------------------------------------------------|------------------------------------------------------------------------------|----------------|-----------------------------------|--------------------------------------|
| NAME : Mr. RAKESH | H RASTOGI | | | |
| AGE/ GENDER : 68 YRS/MAL | .E | PATIENT | ID | : 1742161 |
| COLLECTED BY : | | REG. NO./ | LAB NO. | : 012502010038 |
| REFERRED BY : | | REGISTRA | TION DATE | : 01/Feb/2025 01:08 PM |
| BARCODE NO. : 01524771 | | COLLECTI | ON DATE | :01/Feb/202501:14PM |
| CLIENT CODE. : KOS DIAGNO | | REPORTI | NG DATE | : 01/Feb/2025 04:03PM |
| CLIENT ADDRESS : 6349/1, NIC | HOLSON ROAD, AMBALA C | ANTT | | |
| Test Name | Valu | le | Unit | Biological Reference interval |
| | CLINI | CAL PATHO | LOGY | |
| | URINE ROUTINE & | | | ATION |
| PHYSICAL EXAMINATION | | | | |
| QUANTITY RECIEVED | 10 | | ml | |
| by DIP STICK/REFLECTANCE SPECTRON | | LE YELLOW | | PALE YELLOW |
| by DIP STICK/REFLECTANCE SPECTRO | PHOTOMETRY | | | |
| TRANSPARANCY by DIP STICK/REFLECTANCE SPECTRON | | EAR | | CLEAR |
| SPECIFIC GRAVITY | 1.0 | 2 | | 1.002 - 1.030 |
| by DIP STICK/REFLECTANCE SPECTRO | PHOTOMETRY | | | |
| <u>CHEMICAL EXAMINATION</u> REACTION | ACI | DIC | | |
| by DIP STICK/REFLECTANCE SPECTRON | | DIC | | |
| PROTEIN by DIP STICK/REFLECTANCE SPECTRO | | | | NEGATIVE (-ve) |
| SUGAR | Neg | gative | | NEGATIVE (-ve) |
| by DIP STICK/REFLECTANCE SPECTRON | PHOTOMETRY 5.5 | | | 5.0 - 7.5 |
| by DIP STICK/REFLECTANCE SPECTRO | | | | 5.0 - 7.5 |
| BILIRUBIN by DIP STICK/REFLECTANCE SPECTRON | | gative | | NEGATIVE (-ve) |
| NITRITE | Neg | gative | | NEGATIVE (-ve) |
| by DIP STICK/REFLECTANCE SPECTRON UROBILINOGEN | | rmal | EU/dL | 0.2 - 1.0 |
| by DIP STICK/REFLECTANCE SPECTRO | | mai | EU/ dL | 0.2 - 1.0 |
| KETONE BODIES by DIP STICK/REFLECTANCE SPECTRO | | gative | | NEGATIVE (-ve) |
| BLOOD | | gative | | NEGATIVE (-ve) |
| by DIP STICK/REFLECTANCE SPECTRO | PHOTOMETRY | | | |
| ASCORBIC ACID by DIP STICK/REFLECTANCE SPECTRON | | GATIVE (-ve) | | NEGATIVE (-ve) |
| MICROSCOPIC EXAMINATION | | | | |
| RED BLOOD CELLS (RBCs) by MICROSCOPY ON CENTRIFUGED URI | | GATIVE (-ve) | /HPF | 0 - 3 |
| by WIGNUSCOF I ON CENTRIFUGED URI | | | | |





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

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: Ilnd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana 0171-2643898, +91 99910 43898 | care@koshealthcare.com | www.koshealthcare.com







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

| NAME | : Mr. RAKESH RASTOGI | | | | |
|--------------------|----------------------------------------|-------|--------------------------|------------------------------------------------|--|
| AGE/ GENDER | : 68 YRS/MALE | | PATIENT ID | : 1742161 | |
| COLLECTED BY | : | | REG. NO./LAB NO. | : 012502010038 | |
| REFERRED BY | : | | REGISTRATION DATE | : 01/Feb/2025 01:08 PM | |
| BARCODE NO. | : 01524771 : KOS DIAGNOSTIC LAB | | COLLECTION DATE | : 01/Feb/2025 01:14PM : 01/Feb/2025 04:03PM | |
| CLIENT CODE. | | | REPORTING DATE | | |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | | | |
| Test Name | | Value | Unit | Biological Reference interval | |
| PUS CELLS | CENTRIFUGED URINARY SEDIMENT | 3-4 | /HPF | 0 - 5 | |
| | | | | | |

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

| EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | 2-3 | /HPF | ABSENT |
|-----------------------------------------------------------------------------------|----------------|------|----------------|
| CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| BACTERIA 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 ***



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

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

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