

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



| | Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar | obiology) | | (Pathology) |
|--------------------------------------|---|-------------------|-------------------|---|
| NAME | : Mrs. SUDESH KUMARI | | | |
| AGE/ GENDER | : 75 YRS/FEMALE | | PATIENT ID | : 1793984 |
| COLLECTED BY | : SURJESH | | REG. NO./LAB NO. | : 012503170022 |
| REFERRED BY | : | | REGISTRATION DATE | : 17/Mar/2025 09:14 AM |
| BARCODE NO. | : 01527245 | | COLLECTION DATE | : 17/Mar/2025 09:22AM |
| CLIENT CODE. CLIENT ADDRESS | : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMB/ | | REPORTING DATE | : 17/Mar/2025 09:43AM |
| Test Name | | Value | Unit | Biological Reference interval |
| | SWAST | THYA WE | ELLNESS PANEL: G | |
| | COMP | PLETE BLO | DOD COUNT (CBC) | |
| RED BLOOD CELLS | (RBCS) COUNT AND INDICES | | | |
| HAEMOGLOBIN (HI | 3) | 10.2 ^L | gm/dL | 12.0 - 16.0 |
| RED BLOOD CELL (I | | 4.9 | Millions/ | cmm 3.50 - 5.00 |
| by HYDRO DYNAMIC FO | DCUSING, ELECTRICAL IMPEDENCE | 33.5 ^L | % | 37.0 - 50.0 |
| by CALCULATED BY A | JTOMATED HEMATOLOGY ANALYZER | | fL | 80.0 - 100.0 |
| | JTOMATED HEMATOLOGY ANALYZER | 68.4 ^L | IL | |
| | AR HAEMOGLOBIN (MCH) JTOMATED HEMATOLOGY ANALYZER | 20.9 ^L | pg | 27.0 - 34.0 |
| MEAN CORPUSCUL | AR HEMOGLOBIN CONC. (MCHC) JTOMATED HEMATOLOGY ANALYZER | 30.5 ^L | g/dL | 32.0 - 36.0 |
| | JTION WIDTH (RDW-CV) JTOMATED HEMATOLOGY ANALYZER | 17.7 ^H | % | 11.00 - 16.00 |
| RED CELL DISTRIBU | JTION WIDTH (RDW-SD) JTOMATED HEMATOLOGY ANALYZER | 45.4 | fL | 35.0 - 56.0 |
| MENTZERS INDEX by CALCULATED | | 13.96 | RATIO | BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0 |
| GREEN & KING IND by CALCULATED | | 24.81 | RATIO | BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: > 65.0 |
| WHITE BLOOD CEI | | 7.102 | | |
| TOTAL LEUCOCYTE by FLOW CYTOMETRY | COUNT (TLC) By SF CUBE & MICROSCOPY | 7430 | /cmm | 4000 - 11000 |
| NUCLEATED RED B | LOOD CELLS (nRBCS) t hematology analyzer | NIL | | 0.00 - 20.00 |
| | | | | |

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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

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 0171-2643898, +91 99910 43898
 care@koshealthcare.com

 www.koshealthcare.com
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Dr Vinay Ch



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| Dr. Vinay Cho MD (Pathology & N | | | Dr. Yugam | m Chopra D (Pathology) | |
|--|---|--------------------|------------------|-------------------------------|--|
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| Test Name | | Value | Unit | Biological Reference interval | |
| | UCOCYTE COUNT (DLC) | | | | |
| NEUTROPHILS | | 57 | % | 50 - 70 | |
| by FLOW CYTOMETRY LYMPHOCYTES | Y BY SF CUBE & MICROSCOPY | 34 | % | 20 - 40 | |
| | Y BY SF CUBE & MICROSCOPY | 34 | % | 20 - 40 | |
| EOSINOPHILS | Y BY SF CUBE & MICROSCOPY | 2 | % | 1 - 6 | |
| MONOCYTES | Y BY SF CUBE & MICROSCOPY | 7 | % | 2 - 12 | |
| BASOPHILS | Y BY SF CUBE & MICROSCOPY | 0 | % | 0 - 1 | |
| • | CYTES (WBC) COUNT | | | | |
| ABSOLUTE NEUTR by FLOW CYTOMETR | OPHIL COUNT y by sf cube & microscopy | 4235 | /cmm | 2000 - 7500 | |
| ABSOLUTE LYMPH by FLOW CYTOMETRY | OCYTE COUNT (by sf cube & microscopy | 2526 | /cmm | 800 - 4900 | |
| ABSOLUTE EOSINC | | 149 | /cmm | 40 - 440 | |
| ABSOLUTE MONOC | YTE COUNT | 520 | /cmm | 80 - 880 | |
| | Y BY SF CUBE & MICROSCOPY D THER PLATELET PREDICTIV | E MARKERS | | | |
| PLATELET COUNT | | 277000 | /cmm | 150000 - 450000 | |
| | OCUSING, ELECTRICAL IMPEDENCE | 0.0 | 0/ | 0.10, 0.00 | |
| PLATELETCRIT (PC by HYDRO DYNAMIC F | OCUSING, ELECTRICAL IMPEDENCE | 0.3 | % | 0.10 - 0.36 | |
| MEAN PLATELET V | OLUME (MPV) OCUSING, ELECTRICAL IMPEDENCE | 11 | fL | 6.50 - 12.0 | |
| PLATELET LARGE | CELL COUNT (P-LCC) CCUSING, ELECTRICAL IMPEDENCE | 97000 ^H | /cmm | 30000 - 90000 | |
| PLATELET LARGE | CELL RATIO (P-LCR) | 35 | % | 11.0 - 45.0 | |
| PLATELET DISTRIE | BUTION WIDTH (PDW) | 15.7 | % | 15.0 - 17.0 | |
| - | CTED ON EDTA WHOLE BLOOD | | | | |





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| | , | | | |
| Test Name | | Value | Unit | Biological Reference interva |
| WHOLE BLOOD by HPLC (HIGH PERFOR | EMOGLOBIN (HbA1c): | 7.8 ^H | % | 4.0 - 6.4 |
| | GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) | 177.16 ^H | mg/dL | 60.00 - 140.00 |
| INTERPRETATION: | , | | | |
| | AS PER AMERICAN E | DIABETES ASSOCIATION | I (ADA): | |
| | REFERENCE GROUP | | YLATED HEMOGLOGIB | (HBAIC) in % |
| Non dia | abetic Adults >= 18 years | / | <5.7 | |
| | t Risk (Prediabetes) | | 5.7 – 6.4 | |
| D | iagnosing Diabetes | | >= 6.5 | |
| | | | Age > 19 Years | |
| These l | in and a few shares in a sector i | Goals of Th | | < 7.0 |
| Inerapeut | ic goals for glycemic control | Actions Sugg | | >8.0 |
| 1 | | Goal of the | Age < 19 Years | |
| | | | | <7.5 |

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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 HbAlc. 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 appropriate.

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 |
| | ERYTHROC | CYTE SEDIME | NTATION RATE (| ESR) |
| INTERPRETATION: 1. ESR is a non-specifimmune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythe CONDITION WITH LOV A low ESR can be see (polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactive 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dext | does not tell the health practitioner cted by other conditions besides infl be used to monitor disease activity a ematosus W ESR n with conditions that inhibit the no ificantly high white blood cell count e cell anaemia) also lower the ESR. e protein (C-RP) are both markers of s not change as rapidly as does CRP, by as many other factors as is ESR , n ed, it is typically a result of two type ye a higher ESR. and menstruation al | exactly where the lammation. For the and response to the prmal sedimentation (leucocytosis), a finflammation. , either at the stan making it a better se of proteins, glo nd pregnancy car | e inflammation is in the his reason, the ESR is typ herapy in both of the a on of red blood cells, su and some protein abno rt of inflammation or as marker of inflammatior bulins or fibrinogen. i cause temporary eleva | 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 irmalities. Some changes in red cell shape (such s it resolves. n . |
| | | | | |





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

MBBS, MD (PATHOLOGY)



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| | | & Microbiology) nsultant Pathologist | Dr. Yugan MD CEO & Consultant | (Pathology) |
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| Test Name | | Value | Unit | Biological Reference interval |
| | CLINI | CAL CHEMISTRY GLUCOSE FAS | | TRY |
| GLUCOSE FASTING by glucose oxidas | (F): PLASMA E - PEROXIDASE (GOD-POD) | 136.1 ^H | mg/dL | NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0 |

IN ACCRDANCE 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 TO | | 154.48 | mg/dL | OPTIMAL: < 200.0 |
| by CHOLESTEROL OX | | 134.40 | ing/ uL | BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0 |
| TRIGLYCERIDES: S. by GLYCEROL PHOSP | ERUM HATE OXIDASE (ENZYMATIC) | 132.4 | mg/dL | OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 |
| | | | | VERY HIGH: $> OR = 500.0$ |
| HDL CHOLESTEROI by SELECTIVE INHIBIT. | L (DIRECT): SERUM Ion | 53.14 | mg/dL | LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0 |
| LDL CHOLESTEROI by CALCULATED, SPE | | 74.86 | 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 | | 101.34 | mg/dL | OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 |
| VLDL CHOLESTER | | 26.48 | mg/dL | VERY HIGH: > OR = 220.0 0.00 - 45.00 |
| by CALCULATED, SPE TOTAL LIPIDS: SER by CALCULATED, SPE | CUM | 441.36 | mg/dL | 350.00 - 700.00 |
| by CALCULATED, SPE CHOLESTEROL/HD by CALCULATED, SPE | DL RATIO: SERUM | 2.91 | 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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| Test Name | | Value | Unit | Biological Reference interval |
| LDL/HDL RATIO: S by CALCULATED, SPE | | 1.41 | RATIO | LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0 |
| TRIGLYCERIDES/H by CALCULATED, SPE | IDL RATIO: SERUM | 2.49 ^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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| | LIVER | FUNCTION | TEST (COMPLETE) | |
| BILIRUBIN TOTAL by DIAZOTIZATION, SI | : SERUM PECTROPHOTOMETRY | 0.62 | mg/dL | INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20 |
| | Г (CONJUGATED): SERUM spectrophotometry | 0.15 | mg/dL | 0.00 - 0.40 |
| BILIRUBIN INDIRE by CALCULATED, SPE | CCT (UNCONJUGATED): SERUM | 0.47 | mg/dL | 0.10 - 1.00 |
| SGOT/AST: SERUM by IFCC, WITHOUT PY | [/RIDOXAL PHOSPHATE | 22.3 | U/L | 7.00 - 45.00 |
| SGPT/ALT: SERUM by IFCC, WITHOUT PY | [/RIDOXAL PHOSPHATE | 15.8 | U/L | 0.00 - 49.00 |
| AST/ALT RATIO: S by CALCULATED, SPE | | 1.41 | RATIO | 0.00 - 46.00 |
| ALKALINE PHOSPI by PARA NITROPHEN PROPANOL | HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL | 82.36 | U/L | 40.0 - 130.0 |
| GAMMA GLUTAMY by SZASZ, SPECTROF | L TRANSFERASE (GGT): SERUM | 18.41 | U/L | 0.00 - 55.0 |
| TOTAL PROTEINS: by BIURET, SPECTRO | | 6.72 | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM by BROMOCRESOL G | | 3.87 | gm/dL | 3.50 - 5.50 |
| GLOBULIN: SERUM | 1 | 2.85 | gm/dL | 2.30 - 3.50 |
| by CALCULATED, SPE A : G RATIO: SERUI | | 1.36 | RATIO | 1.00 - 2.00 |

by CALCULATED, SPECTROPHOTOMETRY

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).

PROGNOSTIC SIGNIFICANCE:

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



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 0171-2643898, +91 99910 43898
 care@koshealthcare.com
 www.koshealthcare.com







| | Dr. Vinay Cho MD (Pathology & N Chairman & Consu | 1icrobiology) | Dr. Yugam MD CEO & Consultant | (Pathology) |
|---|--|-------------------|-------------------------------------|------------------------------|
| NAME | : Mrs. SUDESH KUMARI | | | |
| AGE/ GENDER | : 75 YRS/FEMALE |] | PATIENT ID | : 1793984 |
| COLLECTED BY | : SURJESH |] | REG. NO./LAB NO. | : 012503170022 |
| REFERRED BY | : |] | REGISTRATION DATE | : 17/Mar/2025 09:14 AM |
| BARCODE NO. | : 01527245 | (| COLLECTION DATE | : 17/Mar/2025 09:22AM |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | 1 | REPORTING DATE | : 17/Mar/2025 12:20PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AN | MBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interva |
| | KIDNE | EY FUNCTION | N TEST (COMPLETE) | |
| UREA: SERUM by UREASE - GLUTAN | IATE DEHYDROGENASE (GLDH) | 43.01 | mg/dL | 10.00 - 50.00 |
| CREATININE: SERI | | 0.99 | mg/dL | 0.40 - 1.20 |
| - | ROGEN (BUN): SERUM | 20.1 | mg/dL | 7.0 - 25.0 |
| | ROGEN (BUN)/CREATININE | 20.3 ^H | RATIO | 10.0 - 20.0 |
| UREA/CREATININ by CALCULATED, SPE | E RATIO: SERUM | 43.44 | RATIO | |
| URIC ACID: SERUM | 1 | 4.05 | mg/dL | 2.50 - 6.80 |
| CALCIUM: SERUM by ARSENAZO III, SPE | | 9.58 | mg/dL | 8.50 - 10.60 |
| PHOSPHOROUS: SE by PHOSPHOMOLYBE | | 3.46 | mg/dL | 2.30 - 4.70 |
| <u>ELECTROLYTES</u> | | | | |
| SODIUM: SERUM by ISE (ION SELECTIV | (F ELECTRODE) | 140.9 | mmol/L | 135.0 - 150.0 |
| POTASSIUM: SERU by ISE (ION SELECTIV | M | 5.06 ^H | mmol/L | 3.50 - 5.00 |
| CHLORIDE: SERUN by ISE (ION SELECTIV | 1 | 105.68 | mmol/L | 90.0 - 110.0 |
| | ERULAR FILTERATION RATE | 59.5 | | |

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.



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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





| | Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist | | | | | | |
|---|---|--|--|---|----------------------------------|-----------------|------------|
| IAME | : Mrs. SUDESH KUMARI | | | | | | |
| GE/ GENDER | : 75 YRS/FEMALE | 1 | PATIENT ID | : 1793 | 984 | | |
| OLLECTED BY | : SURJESH | I | REG. NO./LAB NO | . : 012; | 503170022 | | |
| REFERRED BY | | | REGISTRATION D | | lar/2025 09:1 | 1 <i>4</i> AM | |
| ARCODE NO. | : 01527245 | | COLLECTION DAT | | lar/2025 09:2 | | |
| LIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DAT | | lar/2025 12:2 | | |
| LIENT CODE. | : 6349/1, NICHOLSON ROAI | | LF OK I ING DA I | E . 1771VI | idi / 2023 12.2 | | |
| | | | | | | | |
| Fest Name | | Value | Ur | nit | Biologica | l Reference i | nterval |
| 9. Certain drugs (e.g. NCREASED RATIO (>2 . Postrenal azotemia | hass (subnormal creatinine pro tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately supprimposed on ronal disease | NE LEVELS: / more than creatinin | e) (e.g. obstructiv | e uropathy). | | | |
| Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in SETIMATED GLOMERI CKD STAGE | tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false creased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION | NE LEVELS: / more than creatinin e. ffuses out of extrace bsent in blood). rmone) due to tubula VINE: creatine to creatinine increase in creatinine measurement). J | Ilular fluid). r secretion of urea e). e with certain me ./min/1.73m2) | a. thodologies,resu | FINDINGS | al ratio when c | dehydratio |
| Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin thei STIMATED GLOMERI CKD STAGE G1 | tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun | NE LEVELS: y more than creatinin e. ffuses out of extrace bsent in blood). rmone) due to tubula JINE: creatine to creatinine increase in creatinine measurement). J GFR (mil nction | Ilular fluid). r secretion of urea e). e with certain me <u>/min/1.73m2)</u> >90 | a. thodologies,resu ASSOCIATED No prote | FINDINGS | al ratio when c | dehydratio |
| Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin thei STADE GLOMERI STAGE | tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage normal or high | NE LEVELS: more than creatinin e. ffuses out of extrace bsent in blood). rmone) due to tubula MINE: creatine to creatinine increase in creatinine measurement). M GFR (mil GFR | Ilular fluid). r secretion of urea e). e with certain me ./min/1.73m2) | a. thodologies,resu | FINDINGS einuria Protein , | al ratio when c | dehydratio |
| Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Perenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin the STIMATED GLOMERI CKD STAGE | tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. treased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage normal or high Mild decrease in | NE LEVELS: (more than creatinin e. ffuses out of extrace bsent in blood). rmone) due to tubula JINE: creatine to creatinine increase in creatinine measurement). J GFR (minimum GFR | Ilular fluid). r secretion of urea e). e with certain me <u>/min/1.73m2)</u> >90 | a. thodologies,resu ASSOCIATED No prote Presence of | FINDINGS einuria Protein , | al ratio when c | dehydratio |
| Certain drugs (e.g. VCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Loid produce an in Cephalosporin thee STIMATED GLOMERNI G1 G2 G3a G3b | tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage normal or high Moderate decrease | NE LEVELS: y more than creatining e. offuses out of extrace osent in blood). rmone) due to tubula JINE: creatine to creatining increase in creatining measurement). M GFR GFR in GFR | Ilular fluid). r secretion of urea e). e with certain me <u>/min/1.73m2)</u> >90 >90 60 -89 30-59 | a. thodologies,resu ASSOCIATED No prote Presence of | FINDINGS einuria Protein , | al ratio when c | dehydratio |
| Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin thei STIMATED GLOMERI G1 G2 | tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. treased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage normal or high Mild decrease in | NE LEVELS: / more than creatining e. offuses out of extrace osent in blood). rmone) due to tubula JINE: creatine to creatining increase in creatining measurement). M GFR in GFR in GFR in GFR | Ilular fluid). r secretion of urea e). e with certain me <u>/min/1.73m2)</u> >90 >90 60 -89 | a. thodologies,resu ASSOCIATED No prote Presence of | FINDINGS einuria Protein , | al ratio when c | dehydratio |





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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMBAL | | | | |
| Test Name | v | alue 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

End Of Report ***





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

MBBS, MD (PATHOLOGY)

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