



| | Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar | obiology) | | (Pathology) |
|-----------------------------------|---|-------------------|------------------------------------|--|
| NAME | : Mrs. SONIA DABAS | | | |
| AGE/ GENDER | : 48 YRS/FEMALE | | PATIENT ID | : 1751247 |
| COLLECTED BY | : SURJESH | | REG. NO./LAB NO. | : 012502100007 |
| REFERRED BY | : | | REGISTRATION DATE | : 10/Feb/2025 08:23 AM |
| BARCODE NO. | : 01525249 | | COLLECTION DATE | : 10/Feb/2025 08:29AM |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 10/Feb/2025 09:38AM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMB/ | ALA CANTI | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | СОМР | | LLNESS PANEL: G OOD COUNT (CBC) | |
| | (RBCS) COUNT AND INDICES | | | |
| HAEMOGLOBIN (HI | 3) | 8.3 ^L | gm/dL | 12.0 - 16.0 |
| RED BLOOD CELL (I | RBC) COUNT | 4.35 | Millions | /cmm 3.50 - 5.00 |
| PACKED CELL VOLU | | 27.3 ^L | % | 37.0 - 50.0 |
| MEAN CORPUSCULA | | 62.9 ^L | fL | 80.0 - 100.0 |
| by CALCULATED BY A | AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER | 19 ^L | pg | 27.0 - 34.0 |
| | AR HEMOGLOBIN CONC. (MCHC) | 30.2 ^L | g/dL | 32.0 - 36.0 |
| | JTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER | 18.6 ^H | % | 11.00 - 16.00 |
| RED CELL DISTRIBU | JTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER | 43.7 | fL | 35.0 - 56.0 |
| MENTZERS INDEX | | 14.46 | RATIO | BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0 |
| GREEN & KING IND by CALCULATED | | 26.78 | RATIO | BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0 |
| TOTAL LEUCOCYTE | | 10010 | /cmm | 4000 - 11000 |
| NUCLEATED RED B | LOOD CELLS (nRBCS) | NIL | | 0.00 - 20.00 |
| | LOOD CELLS (nRBCS) % | NIL | % | < 10 % |





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

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| Test Name | | Value | Unit | Biological Reference interval |
| DIFFERENTIAL LE | EUCOCYTE COUNT (DLC) | | | |
| NEUTROPHILS | | 68 | % | 50 - 70 |
| • | Y BY SF CUBE & MICROSCOPY | 94 | 0/ | 20 40 |
| LYMPHOCYTES by FLOW CYTOMETR | Y BY SF CUBE & MICROSCOPY | 24 | % | 20 - 40 |
| EOSINOPHILS | | 4 | % | 1 - 6 |
| | Y BY SF CUBE & MICROSCOPY | 4 | 0/ | 0 10 |
| MONOCYTES by FLOW CYTOMETR | Y BY SF CUBE & MICROSCOPY | 4 | % | 2 - 12 |
| BASOPHILS | | 0 | % | 0 - 1 |
| | Y BY SF CUBE & MICROSCOPY | | | |
| | OCYTES (WBC) COUNT | 0007 | | 0000 7500 |
| ABSOLUTE NEUTR | Y BY SF CUBE & MICROSCOPY | 6807 | /cmm | 2000 - 7500 |
| ABSOLUTE LYMPH | | 2402 | /cmm | 800 - 4900 |
| | Y BY SF CUBE & MICROSCOPY | 100 | | |
| ABSOLUTE EOSINO | OPHIL COUNT Y by sf cube & microscopy | 400 | /cmm | 40 - 440 |
| ABSOLUTE MONOC | | 400 | /cmm | 80 - 880 |
| | Y BY SF CUBE & MICROSCOPY | | | |
| | OTHER PLATELET PREDICTIVE | | | |
| PLATELET COUNT | (PLT) FOCUSING, ELECTRICAL IMPEDENCE | 254000 | /cmm | 150000 - 450000 |
| PLATELETCRIT (P | | 0.28 | % | 0.10 - 0.36 |
| by HYDRO DYNAMIC | FOCUSING, ELECTRICAL IMPEDENCE | | | |
| MEAN PLATELET V | /OLUME (MPV) FOCUSING, ELECTRICAL IMPEDENCE | 11 | fL | 6.50 - 12.0 |
| | CELL COUNT (P-LCC) | 94000 ^H | /cmm | 30000 - 90000 |
| • | FOCUSING, ELECTRICAL IMPEDENCE | | | |
| | CELL RATIO (P-LCR) FOCUSING, ELECTRICAL IMPEDENCE | 37.2 | % | 11.0 - 45.0 |
| | BUTION WIDTH (PDW) | 16.2 | % | 15.0 - 17.0 |
| by HYDRO DYNAMIC I | FOCUSING, ELECTRICAL IMPEDENCE | | | |
| NOTE: TEST CONDU | JCTED ON EDTA WHOLE BLOOD | | | |





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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, | AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | GLY | COSYLATED HAEMOGI | LOBIN (HBA1C) | |
| GLYCOSYLATED HAE WHOLE BLOOD | MOGLOBIN (HbA1c): | 6.8 ^H | % | 4.0 - 6.4 |
| | | | | |
| by HPLC (HIGH PERFORM | E PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) | 148.46 ^H | mg/dL | 60.00 - 140.00 |
| by HPLC (HIGH PERFORM | IANCE LIQUID CHROMATOGRAPHY) | | mg/dL | 60.00 - 140.00 |
| by HPLC (HIGH PERFORM INTERPRETATION: | IANCE LIQUID CHROMATOGRAPHY) | BETES ASSOCIATION (ADA): | mg/dL MOGLOGIB (HBAIC) ii | |
| by HPLC (HIGH PERFORM INTERPRETATION: RE Non diab | MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAI FERENCE GROUP Metic Adults >= 18 years | BETES ASSOCIATION (ADA): GLYCOSYLATED HEI | Moglogib (HBAIC) in <5.7 | |
| by HPLC (HIGH PERFORM <u>NTERPRETATION:</u> RE Non diab At F | AS PER AMERICAN DIA FERENCE GROUP Metic Adults >= 18 years Risk (Prediabetes) | BETES ASSOCIATION (ADA): GLYCOSYLATED HEI 5. | MOGLOGIB (HBAIC) ii <5.7 7 – 6.4 | |
| by HPLC (HIGH PERFORM INTERPRETATION: Non diab At F | MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAI FERENCE GROUP Metic Adults >= 18 years | BETES ASSOCIATION (ADA): GLYCOSYLATED HEI 5. | MOGLOGIB (HBAIC) in <5.7 7 – 6.4 >= 6.5 | |
| by HPLC (HIGH PERFORM NTERPRETATION: RE Non diab At F | AS PER AMERICAN DIA FERENCE GROUP Metic Adults >= 18 years Risk (Prediabetes) | BETES ASSOCIATION (ADA): GLYCOSYLATED HEI 5. Age 2 | MOGLOGIB (HBAIC) ii <5.7 7 – 6.4 >= 6.5 > 19 Years | n % |
| by HPLC (HIGH PERFORM INTERPRETATION: | AANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAI FERENCE GROUP wetic Adults >= 18 years Risk (Prediabetes) gnosing Diabetes | BETES ASSOCIATION (ADA): GLYCOSYLATED HEI 5. 2. 2. 3. 4. 4. 5. 2. 5. 2. 5. 2. 5. 2. 5. 2. 5. 2. 5. 2. 5. 2. 5. 2. 5. 2. 5. 2. 5. 5. 5. 5. 5. 5. 5. 5. 5. 5. 5. 5. 5. | MOGLOGIB (HBAIC) in <5.7 7 – 6.4 >= 6.5 > 19 Years < 7.0 | n % |
| INTERPRETATION: RE Non diab At F Diag | AS PER AMERICAN DIA FERENCE GROUP Metic Adults >= 18 years Risk (Prediabetes) | BETES ASSOCIATION (ADA): GLYCOSYLATED HEI 5. 5. Age 2 Goals of Therapy: Actions Suggested: | MOGLOGIB (HBAIC) ii <5.7 7 – 6.4 >= 6.5 > 19 Years | n % |

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 PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, A | MBALA CANTT | |
| Test Name | | Value Unit | Biological Reference interval |

| Name : Age : Gender : | Case : Department : | Patient Type Sample Type | : : Whole Blood EDTA | Test Date:10/02/2025 17:38:3 Sample Id:01525249 Total Area:4987 |
|-----------------------------|------------------------|-----------------------------|-------------------------|---|
| Peak Name | Retention Time(s) | Absorbance | Area | Result (Area %) |
| НЬА0 | 69 | 1405 | 4352 | 82.0 |
| HbA1c | 37 | 37 | 363 | 6.8 |
| La1c | 26 | 22 | 146 | 2.8 |
| HbF | 19 | 10 | 9 | 0.2 |
| Hba1b | 14 | 16 | 62 | 1.1 |
| Hba1a | 11 | 13 | 55 | 1.0 |
| 0.03 | | | 1 | Choromotography Hba1c |
| 0.025 | | 11 | | Pibalic |
| 0.02- | | 11 | | |
| ¥ 0.015 − | | Add | | |
| 0.01 — | | J~~ (| | |
| 0.005 — | | ſ | | |
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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | REF | ORTING DATE | : 10/Feb/2025 09:56AM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, A | MBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interva |
| <i>by RED CELL AGGREG</i> ITERPRETATION: ESR is a non-specif nmune disease, but | does not tell the health practition | often indicates the phere exactly where the | inflammation is in the | ion associated with infection, cancer and aut |
| s C-reactive protein . This test may also ystemic lupus erythe | be used to monitor disease activit ematosus N ESR | ry and response to th | erapy in both of the a | bove diseases as well as some others, such a |
| low ESR can be see polycythaemia), sigr s sickle cells in sickl | n with conditions that inhibit the ificantly high white blood cell cou e cell anaemia) also lower the ES | unt (leucocytosis), a | n of red blood cells, s nd some protein abno | uch as a high red blood cell count rmalities. Some changes in red cell shape (su |
| A low ESR can be see polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactiv 2. Generally, ESR doe 8. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 5. Drugs such as dext | ificantly high white blood cell cou e cell anaemia) also lower the ES e 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 ve a higher ESR, and menstruation | unt (leucocytosis) , a R. RP, either at the star e , making it a better n pes of proteins, glob and pregnancy can | nd some protein abno t of inflammation or a: harker of inflammatior ulins or fibrinogen. cause temporary eleva | ormalities. Šome changes in red cell shape (si s it resolves. n . |
| (polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactiv 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 5. Drugs such as dext | ificantly high white blood cell cou e cell anaemia) also lower the ES e 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 ve a higher ESR, and menstruatior ran, methyldopa, oral contracept | unt (leucocytosis) , a R. RP, either at the star e , making it a better n pes of proteins, glob and pregnancy can | nd some protein abno t of inflammation or a: harker of inflammatior ulins or fibrinogen. cause temporary eleva | ormalities. Šome changes in red cell shape (su s it resolves. n. ations. |





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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD | D, AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | CLINI | ICAL CHEMISTRY GLUCOSE FAS | | 'nY |
| | | | | |

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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





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| Fest Name | | Value | Unit | Biological Reference interval |
| | | LIPID PRO | OFILE : BASIC | |
| CHOLESTEROL TO | TAL: SERUM | 157.25 | mg/dL | OPTIMAL: < 200.0 |
| by CHOLESTEROL OX | | 107.20 | ing, ui | BORDERLINE HIGH: 200.0 - |
| | | | | 239.0 |
| | | | | HIGH CHOLESTEROL: > OR = 240.0 |
| RIGLYCERIDES: S | | 133.25 | mg/dL | OPTIMAL: < 150.0 |
| by GLYCEROL PHOSP | PHATE OXIDASE (ENZYMATIC) | | | BORDERLINE HIGH: 150.0 - |
| | | | | 199.0 HIGH: 200.0 - 499.0 |
| | | | | VERY HIGH: > OR = 500.0 |
| IDL CHOLESTERO | L (DIRECT): SERUM | 36.13 | mg/dL | LOW HDL: < 30.0 |
| by SELECTIVE INHIBIT | ION | | | BORDERLINE HIGH HDL: 30.0 60.0 |
| | | | | HIGH HDL: $> OR = 60.0$ |
| DL CHOLESTEROI | | 94.47 | mg/dL | OPTIMAL: < 100.0 |
| by CALCOLATED, SFL | CIROPHOTOMETRY | | | ABOVE OPTIMAL: 100.0 - 129. BORDERLINE HIGH: 130.0 - |
| | | | | 159.0 |
| | | | | HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0 |
| ION HDL CHOLEST | FEROL: SERUM | 121.12 | mg/dL | OPTIMAL: < 130.0 |
| by CALCULATED, SPE | | | 8 | ABOVE OPTIMAL: 130.0 - 159. |
| | | | | BORDERLINE HIGH: 160.0 - 189.0 |
| | | | | HIGH: 190.0 - 219.0 |
| | | | | VERY HIGH: $> OR = 220.0$ |
| LDL CHOLESTER(by CALCULATED, SPE | | 26.65 | mg/dL | 0.00 - 45.00 |
| TOTAL LIPIDS: SER | CUM | 447.75 | mg/dL | 350.00 - 700.00 |
| by CALCULATED, SPE CHOLESTEROL/HD | | 4.35 | RATIO | LOW RISK: 3.30 - 4.40 |
| by CALCULATED, SPE | | 4.55 | KAIIO | 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 | | 2.61 | 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 | 3.69 | 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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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 10/Feb/2025 10:25AM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AM | BALA CANTT | | |
| | | | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | I IVED | FUNCTION | TEST (COMPLETE) | |
| | | | | |
| BILIRUBIN TOTAL by DIAZOTIZATION, SI | : SERUM PECTROPHOTOMETRY | 0.33 | mg/dL | INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20 |
| | C (CONJUGATED): SERUM | 0.15 | mg/dL | 0.00 - 0.40 |
| BILIRUBIN INDIRE | CT (UNCONJUGATED): SERUM | 0.18 | mg/dL | 0.10 - 1.00 |
| SGOT/AST: SERUM by IFCC, WITHOUT PY | [/RIDOXAL PHOSPHATE | 19.16 | U/L | 7.00 - 45.00 |
| SGPT/ALT: SERUM | [/RIDOXAL PHOSPHATE | 23.01 | U/L | 0.00 - 49.00 |
| AST/ALT RATIO: S | ERUM ECTROPHOTOMETRY | 0.83 | RATIO | 0.00 - 46.00 |
| ALKALINE PHOSPI by PARA NITROPHEN PROPANOL | HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL | 121.2 | U/L | 40.0 - 150.0 |
| GAMMA GLUTAMY by SZASZ, SPECTRO | L TRANSFERASE (GGT): SERUM PHTOMETRY | 40.4 | U/L | 0.00 - 55.0 |
| TOTAL PROTEINS: by BIURET, SPECTRO | | 7.26 | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM | | 4.11 | gm/dL | 3.50 - 5.50 |
| GLOBULIN: SERUN by CALCULATED, SPE | 1 | 3.15 | gm/dL | 2.30 - 3.50 |
| A : G RATIO: SERU | | 1.3 | 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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| | Dr. Vinay Chopra MD (Pathology & Microbi Chairman & Consultant P | | (Pathology) |
|--------------------|---|--------------------------|-------------------------------|
| NAME | : Mrs. SONIA DABAS | | |
| AGE/ GENDER | : 48 YRS/FEMALE | PATIENT ID | : 1751247 |
| COLLECTED BY | : SURJESH | REG. NO./LAB NO. | : 012502100007 |
| REFERRED BY | : | REGISTRATION DATE | : 10/Feb/2025 08:23 AM |
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| Test Name | Va | alue Unit | Biological Reference interval |

DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)

2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

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



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| | MD (Pathology & M | Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist | | Chopra (Pathology) Pathologist | |
|--|------------------------------------|---|-------------------|---|--|
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| CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, A | | | | | |
| Test Name | | Value | Unit | Biological Reference interval | |
| | KIDNE | EY FUNCTION | N TEST (COMPLETE) | | |
| UREA: SERUM | | 15.6 | mg/dL | 10.00 - 50.00 | |
| | MATE DEHYDROGENASE (GLDH) | | 0 | | |
| CREATININE: SERUM | | 0.8 | mg/dL | 0.40 - 1.20 | |
| by ENZYMATIC, SPECTROPHOTOMETERY BLOOD UREA NITROGEN (BUN): SERUM | | 7.29 | mg/dL | 7.0 - 25.0 | |
| by CALCULATED, SPECTROPHOTOMETRY | | | | | |
| BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM | | 9.11 ^L | RATIO | 10.0 - 20.0 | |
| | ECTROPHOTOMETRY | | | | |
| UREA/CREATININ | | 19.5 | RATIO | | |
| by CALCULATED, SPECTROPHOTOMETRY | | 5.02 | ma/dI | 2.50 - 6.80 | |
| URIC ACID: SERUN by URICASE - OXIDAS | | 5.02 | mg/dL | 2.30 - 0.80 | |
| CALCIUM: SERUM | | 9.47 | mg/dL | 8.50 - 10.60 | |
| by ARSENAZO III, SPECTROPHOTOMETRY | | 3.08 | mg/dI | 2.30 - 4.70 | |
| PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY | | 3.08 | mg/dL | 2.30 - 4.70 | |
| <u>ELECTROLYTES</u> | | | | | |
| SODIUM: SERUM | | 140.6 | mmol/L | 135.0 - 150.0 | |
| by ISE (ION SELECTIN | | 4.15 | mm ol /I | | |
| POTASSIUM: SERU by ISE (ION SELECTIV | | 4.15 | mmol/L | 3.50 - 5.00 | |
| CHLORIDE: SERUM | Л | 105.45 | mmol/L | 90.0 - 110.0 | |
| by ISE (ION SELECTIN | | | | | |
| | MERULAR FILTERATION RATE | | | | |
| ESTIMATED GLOM (eGFR): SERUM | IERULAR FILTERATION RATE | 90.8 | | | |
| by CALCULATED | | | | | |
| INTERPRETATION: | and and and served served a | | | | |
| io airrerentiate betw | veen 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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| | Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist | | | Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist | | | | |
|--|---|---|--|--|--|---------------------------------|------------------|-----------|
| NAME | : Mrs. SONIA D | ABAS | | | | | | |
| AGE/ GENDER | : 48 YRS/FEMA | LE | PATI | ENT ID | : 17512 | 247 | | |
| COLLECTED BY | : SURJESH | | REG. | NO./LAB NO. | :0125 | 02100007 | | |
| REFERRED BY | | | REGI | STRATION DA | TE : 10/Fe | b/2025 08:2 | 23 AM | |
| BARCODE NO. | :01525249 | | | ECTION DATE | | b/202508:2 | | |
| CLIENT CODE. | : KOS DIAGNOS | TIC I AB | | ORTING DATE | | b/2025 10:2 | | |
| CLIENT CODE. | | OLSON ROAD, AMBA | | JAING DAIL | . 10/14 | D/ 2023 10.2 | JAM | |
| LLIEN I ADDRESS | . 0349/ 1, MUII | OLSON KOAD, AMBA | LA CANTI | | | | | |
| Test Name | | | Value | Unit | | Biologica | l Reference in | ıterval |
| | a (built lises displi | oportionately more th | .S: nan creatinine) (e | .g. obstructive ι | uropathy). | | | |
| 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 <u>ESTIMATED GLOMERI</u> <u>CKD STAGE</u> <u>G1</u> <u>G2</u> <u>G3a</u> | superimposed or 10:1) WITH DECRE Tosis. Ind starvation. e. ecreased urea syn (urea rather than monemias (urea of inappropiate ar 10:1) WITH INCRE/ apy (accelerates con- releases muscle cr who develop ren bis (acetoacetate increased BUN/cre- rapy (interferes w JLAR FILTERATION Norm Kid no Millo | ASED BUN : ASED BUN : creatinine diffuses ou is virtually absent in to tidiuretic harmone) of ASED CREATININE: onversion of creatine reatinine). al failure. causes false increase atinine ratio). ith creatinine measur RATE: DESCRIPTION nal kidney function ney damage with rmal or high GFR d decrease in GFR | an creatinine) (e ut of extracellular blood). lue to tubular sec to creatinine). in creatinine wit ement). GFR (mL/mir >9(>9(60 -6 | r fluid). cretion of urea. ch certain metho h/1.73m2) | | FINDINGS inuria Protein , | al ratio when d | ehydratio |
| DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 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 thei ESTIMATED GLOMERI G1 G2 G3a G3a G3b | superimposed or 10:1) WITH DECRE rosis. Ind starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate ar 10:1) WITH INCRE/ apy (accelerates co releases muscle cr who develop ren creased BUN/cre- rapy (interferes w JLAR FILTERATION Norm Kid no Mile Mode | thesis. creatinine diffuses ou is virtually absent in the tidiuretic harmone) of ASED CREATININE: onversion of creatine reatinine). al failure. causes false increase atinine ratio). ith creatinine measur IRATE: DESCRIPTION nal kidney function ney damage with rmal or high GFR d decrease in GFR rate decrease in GFR | an creatinine) (e ut of extracellular blood). lue to tubular sec to creatinine). in creatinine wit ement). GFR (mL/min >9(>9(60 -4 30-5 | r fluid). cretion of urea. ch certain metho h/1.73m2) | odologies,resul <u>ASSOCIATED</u> <u>No prote</u> Presence of | FINDINGS inuria Protein , | al ratio when de | ehydratic |
| DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 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 there ESTIMATED GLOMERI CKD STAGE G1 G2 G3a | superimposed or 10:1) WITH DECRE rosis. Ind starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate ar 10:1) WITH INCRE/ py (accelerates created releases muscle cr who develop ren creased BUN/cre rapy (interferes w <u>JLAR FILTERATION</u> <u>Sevential</u> Norm Kid no Mode | ASED BUN : ASED BUN : creatinine diffuses ou is virtually absent in to tidiuretic harmone) of ASED CREATININE: onversion of creatine reatinine). al failure. causes false increase atinine ratio). ith creatinine measur RATE: DESCRIPTION nal kidney function ney damage with rmal or high GFR d decrease in GFR | an creatinine) (e ut of extracellular blood). lue to tubular sec to creatinine). in creatinine wit ement). GFR (mL/mir >9(>9(60 -6 | h certain methor | odologies,resul <u>ASSOCIATED</u> <u>No prote</u> Presence of | FINDINGS inuria Protein , | al ratio when de | ehydratic |





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









| | Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant | biology) MI | m Chopra D (Pathology) nt Pathologist |
|--------------------|--|--------------------------|---|
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| 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





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



KOS Diagnostic Lab (A Unit of KOS Healthcare)

| Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Patholog | | licrobiology) | | |
|---|--|--|---|---|
| NAME | : Mrs. SONIA DABAS | | | |
| AGE/ GENDER | : 48 YRS/FEMALE | | PATIENT ID | : 1751247 |
| COLLECTED BY | : SURJESH | | REG. NO./LAB NO. | : 012502100007 |
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| BARCODE NO. | : 01525249 | | COLLECTION DATE | : 10/Feb/2025 08:29AM |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 10/Feb/2025 01:57PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AM | MBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | ІММЦ | J NOPATHO | DLOGY/SEROLOGY | X |
| | RHEUMATOID | FACTOR (R | A): QUANTITATIVE | - SERUM |
| RHEUMATOID (RA) SERUM by NEPHLOMETRY | FACTOR QUANTITATIVE: | 4.23 | IU/mL | NEGATIVE: < 18.0 BORDERLINE: 18.0 - 25.0 POSITIVE: > 25.0 |
| RHEUMATOID ARTHIR 1. Rheumatoid Arthin membrane lining (syr 2. The disease spreda 3. The diagnosis of R/ measurement of RA fa CAUTION (FALSE POST 1. RA factor is not special 2. Non rheumatoid an RA patients have a no 3. Patients with variou lupus erythematosus, 4. Anti-CCP have been specific (98%) than RA 5. Upto 30 % of patier | itis is a systemic autoimmune dise hovium) joints which ledas to prog s from small to large joints, with g A is primarily based on clinical, rac actor. TIVE): cific for Rheumatoid arthiritis, as it i d rheumatoid arthritis (RA) populati nreactive titer and 8% of nonrheum is nonrheumatoid diseases, characte polymyositis, tuberculosis, syphilis, discovered in joints of patients with factor. the with Seronegative Rheumatoid a ive value of Anti-CCP antibodies for | ase that is mult ressive joint de reatest damage liological & imm s often present in tons are not clea atoid patients ha rized by chronic viral hepatitis, in RA, but not in o rthiritis also sho Rheumatoid Arti | i-functional in origin and i struction and in most case in early phase. nunological features. The m n healthy individuals with o rly separate with regard to ave a positive titer). inflammation may have pos fectious mononucleosis, an ther form of joint disease. A w Anti-CCP antibodies. hiritis is far greater than Rh | nti-CCP2 is HIGHLY SENSITIVE (71%) & more |
| | * * | * End Of Re | eport *** | |
| | there | | hopra | |

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