

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



KOS Diagnostic Lab (A Unit of KOS Healthcare)

| | Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar | obiology) | | (Pathology) | |
|--|---|-------------------|-------------------------------------|--------------|----------------------------|
| AME : | : Mrs. KIRAN KAPOOR | | | | |
| GE/ GENDER : | : 68 YRS/FEMALE |] | PATIENT ID | : 1767301 | |
| OLLECTED BY : | : |] | REG. NO./LAB NO. | :01250223 | 0009 |
| EFERRED BY : | : | | REGISTRATION DATE | :23/Feb/202 | |
| | : 01526001 | | COLLECTION DATE | : 23/Feb/202 | |
| | : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMB/ | - | REPORTING DATE | : 23/Feb/202 | 25 09:25AM |
| 'est Name | | Value | Unit | Bio | logical Reference interval |
| | | | LNESS PANEL: 1.5 OOD COUNT (CBC) | | |
| ED BLOOD CELLS () | RBCS) COUNT AND INDICES | | | | |
| AEMOGLOBIN (HB) | | 11.3 ^L | gm/dL | 12. | 0 - 16.0 |
| ED BLOOD CELL (RE | BC) COUNT CUSING, ELECTRICAL IMPEDENCE | 3.89 | Millions/ | cmm 3.5 | 0 - 5.00 |
| ACKED CELL VOLUM | | 34.8 ^L | % | 37. | 0 - 50.0 |
| EAN CORPUSCULAR | R VOLUME (MCV) Tomated hematology analyzer | 89.4 | fL | 80. | 0 - 100.0 |
| | R HAEMOGLOBIN (MCH) | 29 | pg | 27. | 0 - 34.0 |
| by CALCULATED BY AUT | R HEMOGLOBIN CONC. (MCHC) | 32.5 | g/dL | 32. | 0 - 36.0 |
| by CALCULATED BY AUT | TION WIDTH (RDW-CV) FOMATED HEMATOLOGY ANALYZER | 14 | % | 11. | 00 - 16.00 |
| | TION WIDTH (RDW-SD) fomated hematology analyzer | 46.6 | fL | 35. | 0 - 56.0 |
| IENTZERS INDEX | | 22.98 | RATIO | 13. | ON DEFICIENCY ANEMIA: |
| REEN & KING INDE. by calculated | X | 32.12 | RATIO | 65. | ON DEFICIENCY ANEMIA: > |
| HITE BLOOD CELL | <u>S (WBCS)</u> | | | | |
| | OUNT (TLC) y sf cube & microscopy | 7640 | /cmm | | 00 - 11000 |
| • | | NIT | | 0.0 | 0 - 20.00 |
| by FLOW CYTOMETRY B UCLEATED RED BLO by AUTOMATED 6 PART | OOD CELLS (nRBCS) hematology analyzer OOD CELLS (nRBCS) % | NIL NIL | % | | 0 % |





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

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



Page 1 of 19





Dr. Yugam Chopra

MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mrs. KIRAN KAPOOR AGE/ GENDER : 68 YRS/FEMALE **PATIENT ID** :1767301 **COLLECTED BY** :012502230009 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 23/Feb/2025 08:26 AM **BARCODE NO.** :01526001 **COLLECTION DATE** : 23/Feb/2025 08:29AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 23/Feb/2025 09:25AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC) NEUTROPHILS** 58 % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 26% 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 10^H % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 6 % 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 4431 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 1986 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 764^H /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 458 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 228000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) % 0.10 - 0.36 0.31by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 14^H MEAN PLATELET VOLUME (MPV) fL. 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) 30000 - 90000 118000^H /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 51.8^H PLATELET LARGE CELL RATIO (P-LCR) % 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





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

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 www.koshealthcare.com







| | Dr. Vinay Cho MD (Pathology & Chairman & Cons | Microbiology) | Dr. Yugam MD CEO & Consultant | (Pathology) |
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| NAME | : Mrs. KIRAN KAPOOR | | | |
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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | REPO | RTING DATE | : 23/Feb/2025 03:36PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, A | AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interva |
| | | | | |
| GLYCOSYLATED HAE WHOLE BLOOD | GLYCC EMOGLOBIN (HbA1c): | DSYLATED HAEMO 5.6 | GLOBIN (HBA1C % |) 4.0 - 6.4 |
| WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM | | | | |
| WHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI | EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) | 5.6 114.02 DIABETES ASSOCIATION (| % mg/dL (ADA): | 4.0 - 6.4 60.00 - 140.00 |
| WHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI INTERPRETATION: | EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN EFERENCE GROUP | 5.6 114.02 DIABETES ASSOCIATION (| % mg/dL (ADA): LATED HEMOGLOGIB | 4.0 - 6.4 60.00 - 140.00 |
| WHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI INTERPRETATION: RI | EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN EFERENCE GROUP betic Adults >= 18 years | 5.6 114.02 DIABETES ASSOCIATION (| % mg/dL (ADA): LATED HEMOGLOGIB <5.7 | 4.0 - 6.4 60.00 - 140.00 |
| WHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI INTERPRETATION: RI Non diat At | EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN EFERENCE GROUP betic Adults >= 18 years Risk (Prediabetes) | 5.6 114.02 DIABETES ASSOCIATION (| % mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4 | 4.0 - 6.4 60.00 - 140.00 |
| WHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI INTERPRETATION: RI Non diat At | EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN EFERENCE GROUP betic Adults >= 18 years | 5.6 114.02 DIABETES ASSOCIATION (| % mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 | 4.0 - 6.4 60.00 - 140.00 |
| WHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI INTERPRETATION: RI Non diat At Dia | EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN EFERENCE GROUP betic Adults >= 18 years Risk (Prediabetes) | 5.6 114.02 DIABETES ASSOCIATION (| % mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years rapy: | 4.0 - 6.4 60.00 - 140.00 |
| WHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI INTERPRETATION: RI Non diat At Dia | EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN EFERENCE GROUP betic Adults >= 18 years Risk (Prediabetes) agnosing Diabetes | 5.6 114.02 DIABETES ASSOCIATION (GLYCOSY GOals of The | % mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years rapy: | 4.0 - 6.4 60.00 - 140.00 (HBAIC) in % |

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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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| LIENT CODE. | : KOS DIAGNOSTIC LAB | REI | PORTING DATE | : 23/Feb/2025 10:36AM | |
| LIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AM | IBALA CANTT | | | |
| Cest Name | | Value | Unit | Biological Reference | interval |
| | ERYTHRO | CYTE SEDIME | NTATION RATE (| ESR) | |
| An ESR can be affe c-reactive protein This test may also stemic lupus eryth DNDITION WITH LO low ESR can be see olycythaemia), sig sickle cells in sick OTE: ESR and C - reactiv Generally, ESR doo CRP is not affected If the ESR is elevat Women tend to ha Drugs such as dex | be used to monitor disease activity ematosus W ESR in with conditions that inhibit the non- ificantly high white blood cell coun le cell anaemia) also lower the ESR. e protein (C-RP) are both markers of es not change as rapidly as does CRP I by as many other factors as is ESR, r ed, it is typically a result of two type we a higher ESR, and menstruation a | Tammation. For this and response to the prmal sedimentation t (leucocytosis), a f inflammation. c, either at the star making it a better r es of proteins, glob and pregnancy can | s reason, the ESR is ty herapy in both of the a on of red blood cells, s nd some protein abno t of inflammation or a narker of inflammatior ulins or fibrinogen. cause temporary eleva | picallý used in conjunction with othe bove diseases as well as some other uch as a high red blood cell count ormalities. Some changes in red cell s s it resolves. n. | rs, such as shape (such |
| | | | | | |





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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | RE | PORTING DATE | : 23/Feb/2025 11:29AM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAI | D, AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | CLIN | ICAL CHEMISTR | Y/BIOCHEMIST | TRY |
| | | GLUCOSE FA | STING (F) | |
| GLUCOSE FASTING by glucose oxidas | G (F): PLASMA E - PEROXIDASE (GOD-POD) | 91.58 | 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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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROA | D, AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | | LIPID PROF | ILE · BASIC | |
| CHOLESTEROL TO | TAL · SERUM | 174.96 | mg/dL | OPTIMAL: < 200.0 |
| by CHOLESTEROL O | | 174.90 | ing/uL | BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0 |
| TRIGLYCERIDES: S by GLYCEROL PHOSE | ERUM HATE OXIDASE (ENZYMATIC) | 134.36 | mg/dL | OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0 |
| HDL CHOLESTERO by SELECTIVE INHIBIT | L (DIRECT): SERUM | 61.52 | mg/dL | LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0 |
| LDL CHOLESTERO | | 86.57 | 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 CHOLES' by calculated, spe | | 113.44 | mg/dL | 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 CHOLESTER | | 26.87 | mg/dL | 0.00 - 45.00 |
| TOTAL LIPIDS: SEF | RUM | 484.28 | mg/dL | 350.00 - 700.00 |
| CHOLESTEROL/HI by CALCULATED, SPE | DL RATIO: SERUM | 2.84 | 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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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 23/Feb/2025 12:56PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD | , AMBALA CANT | Т | |
| 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 | 2.18 ^L | RATIO | 3.00 - 5.00 |

<u>INTERPRETATION:</u> 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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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMI | BALA CANTT | | |
| | | | | |
| Test Name | | Value | Unit | Biological Reference interval |
| BILIRUBIN TOTAL | | FUNCTION 1.42 ^H | N TEST (COMPLETE) mg/dL | INFANT: 0.20 - 8.00 |
| | PECTROPHOTOMETRY | 1.42** | iiig/ uL | ADULT: 0.00 - 1.20 |
| | C (CONJUGATED): SERUM | 0.21 | mg/dL | 0.00 - 0.40 |
| | CCT (UNCONJUGATED): SERUM | 1.21 ^H | mg/dL | 0.10 - 1.00 |
| SGOT/AST: SERUM by IFCC, WITHOUT PY | [/RIDOXAL PHOSPHATE | 14.8 | U/L | 7.00 - 45.00 |
| SGPT/ALT: SERUM by IFCC, WITHOUT PY | [/RIDOXAL PHOSPHATE | 12.3 | U/L | 0.00 - 49.00 |
| AST/ALT RATIO: S by CALCULATED, SPE | ERUM ECTROPHOTOMETRY | 1.2 | RATIO | 0.00 - 46.00 |
| ALKALINE PHOSPI by PARA NITROPHEN PROPANOL | HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL | 128.7 | U/L | 40.0 - 130.0 |
| GAMMA GLUTAMY by SZASZ, SPECTRO | L TRANSFERASE (GGT): SERUM | 17.29 | U/L | 0.00 - 55.0 |
| TOTAL PROTEINS: by BIURET, SPECTRO | SERUM | 6.52 | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM by BROMOCRESOL G | | 4.14 | gm/dL | 3.50 - 5.50 |
| GLOBULIN: SERUN | | 2.38 | gm/dL | 2.30 - 3.50 |
| A : G RATIO: SERUI | | 1.74 | 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:

| > 2 |
|----------------------------|
| > 2 (Highly Suggestive) |
| 1.4 - 2.0 |
| > 1.5 |
| > 1.3 (Slightly Increased) |
| |



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| | Dr. Vinay Chop MD (Pathology & Mic Chairman & Consulta | crobiology) MI | m Chopra D (Pathology) nt Pathologist |
|--------------------|--|--------------------------|---|
| NAME | : Mrs. KIRAN KAPOOR | | |
| AGE/ GENDER | : 68 YRS/FEMALE | PATIENT ID | : 1767301 |
| COLLECTED BY | : | REG. NO./LAB NO. | : 012502230009 |
| REFERRED BY | : | REGISTRATION DATE | : 23/Feb/2025 08:26 AM |
| BARCODE NO. | : 01526001 | COLLECTION DATE | : 23/Feb/2025 08:29AM |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | REPORTING DATE | : 23/Feb/2025 01:05PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AM | BALA CANTT | |
| Test Name | | Value Unit | Biological Reference interva |

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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| 0 3001 . 2000 0211 | | | | | |
|--|-----------------------------------|-------------|--------------------------|------------------------------|--|
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| Test Name | | Value | Unit | Biological Reference interva | |
| | KIDN | EY FUNCTIO | N TEST (COMPLETE) |) | |
| UREA: SERUM | | 23.34 | mg/dL | 10.00 - 50.00 | |
| by UREASE - GLUTAN CREATININE: SER | MATE DEHYDROGENASE (GLDH) | 0.69 | mg/dI | 0.40 - 1.20 | |
| | UNI CTROPHOTOMETERY | 0.69 | mg/dL | 0.40 - 1.20 | |
| | ROGEN (BUN): SERUM | 10.91 | mg/dL | 7.0 - 25.0 | |
| by CALCULATED, SPECTROPHOTOMETRY BLOOD UREA NITROGEN (BUN)/CREATININE | | 15.81 | RATIO | 10.0 - 20.0 | |
| RATIO: SERUM | | | | | |
| by CALCULATED, SPI | ECTROPHOTOMETRY | 33.83 | RATIO | | |
| by CALCULATED, SPI | ECTROPHOTOMETRY | | | | |
| URIC ACID: SERUN by URICASE - OXIDAS | | 3.63 | mg/dL | 2.50 - 6.80 | |
| CALCIUM: SERUM | | 8.66 | mg/dL | 8.50 - 10.60 | |
| by ARSENAZO III, SPE PHOSPHOROUS: SI | ECTROPHOTOMETRY | 3.71 | mg/dL | 2.30 - 4.70 | |
| | DATE, SPECTROPHOTOMETRY | 5.71 | liig/ uL | 2.30 - 4.70 | |
| <u>ELECTROLYTES</u> | | | | | |
| SODIUM: SERUM by ISE (ION SELECTIN | | 143.6 | mmol/L | 135.0 - 150.0 | |
| POTASSIUM: SERU | | 4.21 | mmol/L | 3.50 - 5.00 | |
| by ISE (ION SELECTIVE ELECTRODE) | | | | | |
| CHLORIDE: SERUN by ISE (ION SELECTIV | | 107.7 | mmol/L | 90.0 - 110.0 | |
| | MERULAR FILTERATION RATE | 2 | | | |
| | IERULAR FILTERATION RATE | 94.5 | | | |
| (eGFR): SERUM | | | | | |
| INTERPRETATION: | | | | | |
| To differentiate betw | icon pro, and pact ronal azotomia | | | | |

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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| MD (Pa | | | | i gam Chopra MD (Pathology) ultant Pathologist | ID (Pathology) | |
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| Test Name | | Value | Unit | Bio | ological Refer | ence interva |
| 8. Reduced muscle m 9. Certain drugs (e.g. I NCREASED RATIO (>2 1. Postrenal azotemia | (e.g. ureter colostomy) ass (subnormal creatinine pro tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal diseas | NE LEVELS: more than creatinine |) (e.g. obstructive (| uropathy). | | |
| Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (<1 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 c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther <u>EXTIMATED GLOMERL CKD STAGE G1 </u> | ass (subnormal creatinine pro tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal diseas 0:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 0:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine ULAR FILTERATION RATE: DESCRIPTION Normal kidney fun | NE LEVELS: more than creatinine e. ffuses out of extracell sent in blood). mone) due to tubular lincrease in creatinine) increase in creatinine measurement). | ular fluid). secretion of urea. with certain metho <u>min/1.73m2)</u> >90 | odologies,resulting i ASSOCIATED FINDI No proteinuria | INGS a | when dehydra |
| Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Perenal azotemia CECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome of SIADH (syndrome of Repancy. Pregnancy. Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STAGE | ass (subnormal creatinine pro tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal diseas 0:1) WITH DECREASED BUN : osis. ad starvation. 2. creased urea synthesis. urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 0:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine ILAR FILTERATION RATE: DESCRIPTION Normal kidney fur Kidney damage | NE LEVELS: more than creatinine e. ffuses out of extracell sent in blood). mone) due to tubular INNE: creatine to creatinine) increase in creatinine measurement). Image: Comparison of the system increase in creatinine measurement). Image: Comparison of the system inction with | ular fluid). secretion of urea. with certain methe min/1.73m2) | odologies,resulting i <u>ASSOCIATED FINDI</u> <u>No proteinuria</u> Presence of Prote | INGS a ein , | when dehydra |
| B. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Nuscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERL CKD STAGE G1 G2 | ass (subnormal creatinine pro tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal diseas 0:1) WITH DECREASED BUN : osis. ad starvation. 2. creased urea synthesis. urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 0:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine ULAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage normal or high | NE LEVELS: more than creatinine e. ffuses out of extracell sent in blood). mone) due to tubular INE: creatine to creatinine) increase in creatinine measurement). Image: ortion with GFR | ular fluid). secretion of urea. with certain metho <u>min/1.73m2)</u> >90 >90 | odologies,resulting i ASSOCIATED FINDI No proteinuria | INGS a ein , | when dehydra |
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| A. Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (<1 Nhenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin ther ESTIMATED GLOMERL G1 G2 G3a | ass (subnormal creatinine pro tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal diseas 0:1) WITH DECREASED BUN : osis. ad starvation. 2. creased urea synthesis. urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 0:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine ILAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage normal or high | NE LEVELS: more than creatinine e. ffuses out of extracell sent in blood). mone) due to tubular INNE: creatine to creatinine measurement). Increase in creatinine with GFR 6 in GFR 3 | ular fluid). secretion of urea. with certain metho <u>min/1.73m2)</u> >90 >90 0 -89 | odologies,resulting i <u>ASSOCIATED FINDI</u> <u>No proteinuria</u> Presence of Prote | INGS a ein , | when dehydra |





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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 Name | | | Value | Unit | Biological Reference inter | val |
| | | | IRON | PROFILE | | |
| IRON: SERUM | TROPHOTOMETRY | , | 35.78 ^L | μg/dL | 37.0 - 145.0 | |
| UNSATURATED IRC SERUM | | | 236.94 | μg/dL | 150.0 - 336.0 | |
| by FERROZINE, SPEC TOTAL IRON BIND SERUM by SPECTROPHOTOM | ING CAPACITY | | 272.72 | µg/dL | 230 - 430 | |
| %TRANSFERRIN SA by CALCULATED, SPE | ATURATION: S | | 13.12 ^L | % | 15.0 - 50.0 | |
| TRANSFERRIN: SE | | | 193.63 ^L | mg/dL | 200.0 - 350.0 | |
| INTERPRETATION:- | ыгс | | | | THALASSEANA - /2 TRAIT | |
| VARIAB SERUM IF | | ANEMIA OF CHROI Normal to Re | | IRON DEFICIENCY ANEMIA Reduced | THALASSEMIA α/β TRAIT Normal | |
| SEROWIN | | Normal to Re | 44004 | neudeeu | Norman | |

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

IRON:

1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency

anemia, anemia of chronic disease and thalassemia syndromes.
 It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.
 TOTAL IRON BINDING CAPACITY (TIBC): It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:

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



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| Test Name | | Value | Unit | Biological Refer | ence interval |
| | | ENDOCRIN | DLOGY | | |
| | 1 | HYROID FUNCTION | I TEST: TOTAL | | |
| TRIIODOTHYRONI | NE (T3): SERUM IESCENT MICROPARTICLE IMMUNO | 1.021 DASSAY) | ng/mL | 0.35 - 1.93 | |
| THYROXINE (T4): S by CMIA (CHEMILUMIN | SERUM IESCENT MICROPARTICLE IMMUN | 8.15 DASSAY) | µgm/dL | 4.87 - 12.60 | |
| by CMIA (CHEMILUMIN | TING HORMONE (TSH): SE | | µIU/mL | 0.35 - 5.50 | |
| 3rd GENERATION, ULT INTERPRETATION: | RASENSITIVE | | | | |
| TSH levels are subject to o day has influence on the triiodothyronine (T3).Fai | measured serum TSH concentrations | TSH stimulates the productio | n and secretion of the n | om. The variation is of the order of 50 netabolically active hormones, thyro er underproduction (hypothyroidisr | oxine (T4)and |
| CLINICAL CONDITION | T3 | T, | | TSH | |
| Primary Hypothyroidis | m: Reduced | I Rec | uced | ncreased (Significantly) | |

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

LIMITATIONS:-

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG), and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.

2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (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.

| TRIIODOTH | TRIIODOTHYRONINE (T3) | | THYROXINE (T4) | | LATING 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 |





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

| l est Name | | | value | Unit | | Biological Reference Interval |
|---------------------|---------------|----------------------|------------------|---------------------|-------------|-------------------------------|
| 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 | EVELS DURING PRE | GNANCY (µIU/mL) | | |
| | 1st Trimester | | | 0.10 - 2.50 | | |
| | 2nd Trimester | | | 0.20 - 3.00 | | |
| | 3rd Trimester | | | 0.30 - 4.10 | | |
| • | | | | | | |

INCREASED TSH LEVELS:

1. Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.

2. Hypothyroid patients receiving insufficient thyroid replacement therapy.

3. Hashimotos thyroiditis

4.DRUGS: Amphetamines, 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)





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



| | | Chopra / & Microbiology) onsultant Pathologist | | (Pathology) | |
|--|--|---|--|--|----------------------------|
| NAME | : Mrs. KIRAN KAPOOR | | | | |
| AGE/ GENDER | : 68 YRS/FEMALE | | PATIENT ID | : 1767301 | |
| OLLECTED BY | : | | REG. NO./LAB NO. | : 012502230009 | |
| EFERRED BY | : | | REGISTRATION DATE | : 23/Feb/2025 08:26 AM | |
| ARCODE NO. | :01526001 | | COLLECTION DATE | : 23/Feb/2025 08:29AM | |
| LIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 23/Feb/2025 11:47AM | |
| LIENT ADDRESS | : 6349/1, NICHOLSON ROA | | | | |
| 'est Name | | Value | Unit | Biological Reference | e interval |
| | | VIT | AMINS | | |
| | VI | | DROXY VITAMIN D | 2 | |
| | DROXY VITAMIN D3): SERU ESCENCE IMMUNOASSAY) | IM 44.2 | ng/mL | DEFICIENCY: < 20.0 INSUFFICIENCY: 20 SUFFICIENCY: 30.0 TOXICITY: > 100.0 |).0 - 30.0 |
| DEFI | CIENT: | < 20 | n | g/mL | |
| | FICIENT: | 21 - 29 | | g/mL | |
| | ED RANGE: | 30 - 100 > 100 | | g/mL g/mL | |
| onversion of 7- dihy .25-OHVitamin D r ssue and tightly bou .Vitamin D plays a p hosphate reabsorpt .Severe deficiency r ECREASED: | rdrocholecalciferol to Vitamin epresents the main body rese- und by a transport protein wh primary role in the maintenand cion, skeletal calcium deposition may lead to failure to mineralize | D3 in the skin upon voir and transport fo ile in circulation. ce of calcium homeo on, calcium mobilizat | Ultraviolet exposure. Irm of Vitamin D and trans Istatis. It promotes calciun tion, mainly regulated by p | ecalciferol (from animals, Vitamin port form of Vitamin D, being stor n absorption, renal calcium absor parathyroid harmone (PTH). ickets in children and osteomalac | ed in adipose ption and |
| Depressed Hepatic Secondary to advar Osteoporosis and S Enzyme Inducing di ICREASED: | malabsorption (celiac disease Vitamin D 25- hydroxylase act need Liver disease Secondary Hyperparathroidism rugs: anti-epileptic drugs like p | ivity (Mild to Moderate henytoin, phenobar | bital and carbamazepine, | that increases Vitamin D metaboli | |
| evere hypercalcemia AUTION : Replaceme ypervitaminosis D | a and hyperphophatemia. ent therapy in deficient individ | uals must be monito | red by periodic assessmen | of Vitamin D. When it occurs, it ca t of Vitamin D levels in order to p | revent |
| OTE:-Dark coloured | individuals as compare to white in D absorption. | es, is at higher risk of | developing Vitamin D defic | iency due to excess of melanin pign | nent which |

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)

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







| Dr. Vinay Ch MD (Pathology & Chairman & Cor | | licrobiology) | Dr. Yugan MD CEO & Consultant | (Pathology) | | |
|---|--|--|--|--|--|--|
| NAME | : Mrs. KIRAN KAPOOR | | | | | |
| AGE/ GENDER | : 68 YRS/FEMALE | PAT | TENT ID | : 1767301 | | |
| COLLECTED BY | : | DEC | . NO./LAB NO. | : 012502230009 | | |
| | • | | | | | |
| REFERRED BY | : | REC | ISTRATION DATE | : 23/Feb/2025 08:26 AM | | |
| BARCODE NO. | : 01526001 | COI | LECTION DATE | : 23/Feb/2025 08:29AM | | |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | REF | ORTING DATE | : 23/Feb/2025 12:41PM | | |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AN | MBALA CANTT | | | | |
| Test Name | | Value | Unit | Biological Reference interval | | |
| | | VITAMIN B12/(229 AY) | C OBALAMIN pg/mL | 190.0 - 890.0 | | |
| INTERPRETATION:- | | | | 1240 | | |
| | SED VITAMIN B12 | 1. Drognonou | DECREASED VITAMIN | N B12 | | |
| 1.Ingestion of Vitam 2.Ingestion of Estro | | 1.Pregnancy 2.DRUGS:Aspirin, Anti-convulsants, Colchicine | | | | |
| 3.Ingestion of Vitam | | 3.Ethanol Igestion | | | | |
| 4.Hepatocellular in | | | tive Harmones | | | |
| 5.Myeloproliferativ | e disorder | 5.Haemodialysis | | | | |
| 6.Uremia | | 6. Multiple N | , , | | | |
| 2.In humans, it is obt 3.The body uses its v excreted. 4.Vitamin B12 deficie ileal resection, small 5.Vitamin B12 deficie proprioception, poor the neurologic defect 6.Serum methylmalo 7.Follow-up testing fo | ency may be due to lack of IF secret intestinal diseases). ency frequently causes macrocytic coordination, and affective behav as without macrocytic anemia. nic acid and homocysteine levels a or antibodies to intrinsic factor (IF) n concentration of vitamin B12 doe | nd requires intrinsionally, reabsorbing vitar tion by gastric mucco anemia, glossitis, po- ioral changes. These re also elevated in v b is recommended to es not rule out tissue | c factor (IF) for absorp nin B12 from the ileun sa (eg, gastrectomy, g eripheral neuropathy, e manifestations may vitamin B12 deficiency o identify this potentia e deficiency of vitamin | n and returning it to the liver; very little is astric atrophy) or intestinal malabsorption (eg weakness, hyperreflexia, ataxia, loss of occur in any combination; many patients have | | |





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







| | Dr. Vinay Cho MD (Pathology & Chairman & Cons | | Dr. Yugam MD CEO & Consultant | (Pathology) |
|--|--|-----------------|-------------------------------------|--------------------------------------|
| NAME : M | rs. KIRAN KAPOOR | | | |
| AGE/ GENDER : 68 | 3 YRS/FEMALE | PATIE | NT ID | : 1767301 |
| COLLECTED BY : | | REG. N | O./LAB NO. | : 012502230009 |
| REFERRED BY : | | REGIS | FRATION DATE | : 23/Feb/2025 08:26 AM |
| | 526001 | COLLE | CTION DATE | : 23/Feb/2025 08:29AM |
| | OS DIAGNOSTIC LAB | | RTING DATE | : 23/Feb/2025 10:08AM |
| CLIENT ADDRESS : 63 | 349/1, NICHOLSON ROAD, A | AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | | CLINICAL PATI | 101.0GY | |
| | URINE RO | UTINE & MICROSC | | ATION |
| PHYSICAL EXAMINATI | <u>ON</u> | | | |
| QUANTITY RECIEVED by DIP STICK/REFLECTANCI | | 10 | ml | |
| COLOUR | | PALE YELLOW | | PALE YELLOW |
| by DIP STICK/REFLECTANCI TRANSPARANCY by DIP STICK/REFLECTANCI | | HAZY | | CLEAR |
| SPECIFIC GRAVITY | | 1.01 | | 1.002 - 1.030 |
| CHEMICAL EXAMINAT | | | | |
| REACTION | | ACIDIC | | |
| by DIP STICK/REFLECTANCI PROTEIN | | Negative | | NEGATIVE (-ve) |
| by DIP STICK/REFLECTANCI | | Negative | | NEGATIVE (-ve) |
| by DIP STICK/REFLECTANCE pH by DIP STICK/REFLECTANCE | | 6.5 | | 5.0 - 7.5 |
| BILIRUBIN | | Negative | | NEGATIVE (-ve) |
| by DIP STICK/REFLECTANCE | | Negative | | NEGATIVE (-ve) |
| by DIP STICK/REFLECTANCE UROBILINOGEN by DIP STICK/REFLECTANCE | | Normal | EU/dL | 0.2 - 1.0 |
| KETONE BODIES by DIP STICK/REFLECTANCI | | Negative | | NEGATIVE (-ve) |
| BLOOD by DIP STICK/REFLECTANCI | | Negative | | NEGATIVE (-ve) |
| ASCORBIC ACID by DIP STICK/REFLECTANCE MICROSCOPIC EXAMIN | E SPECTROPHOTOMETRY | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| RED BLOOD CELLS (RBC | | NEGATIVE (-ve) | /HPF | 0 - 3 |



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Page 18 of 19

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





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

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

| NAME | : Mrs. KIRAN KAPOOR | | | | |
|---------------------------------|--|-------|--------------------------|-------------------------------|--|
| AGE/ GENDER | : 68 YRS/FEMALE | | PATIENT ID | : 1767301 | |
| COLLECTED BY | : | | REG. NO./LAB NO. | : 012502230009 | |
| REFERRED BY | : | | REGISTRATION DATE | : 23/Feb/2025 08:26 AM | |
| BARCODE NO. | :01526001 | | COLLECTION DATE | : 23/Feb/2025 08:29AM | |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 23/Feb/2025 10:08AM | |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | , | | |
| Test Name | | Value | Unit | Biological Reference interval | |
| by MICROSCOPY ON C | CENTRIFUGED URINARY SEDIMENT | | | | |
| PUS CELLS by MICROSCOPY ON (| CENTRIFUGED URINARY SEDIMENT | 3-4 | /HPF | 0 - 5 | |
| | | 16 | /LIDE | ADCENIT | |

| EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | 4-6 | /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)

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