

(A Unit of KOS Healthcare)



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

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

NAME : Mr. RAJEEV GUPTA

AGE/ GENDER : 56 YRS/MALE **PATIENT ID** : 1551632

COLLECTED BY : REG. NO./LAB NO. : 012407170001

 REFERRED BY
 : 17/Jul/2024 06:22 AM

 BARCODE NO.
 : 01513277
 COLLECTION DATE
 : 17/Jul/2024 07:39AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 17/Jul/2024 08:52AM

CLIENT ADDRESS: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

SWASTHYA WELLNESS PANEL: 1.5 COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOCLORINI (HR)

| HAEMOGLOBIN (HB) by CALORIMETRIC | 14.4 | gm/aL | 12.0 - 17.0 |
|---|-------------------|--------------|--|
| RED BLOOD CELL (RBC) COUNT by Hydro dynamic focusing, electrical impedence | 5.51 ^H | Millions/cmm | 3.50 - 5.00 |
| PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 44.2 | % | 40.0 - 54.0 |
| MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 80.2 | fL | 80.0 - 100.0 |
| MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 26.1 ^L | pg | 27.0 - 34.0 |
| MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 32.5 ^L | g/dL | 32.0 - 36.0 |
| RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 15 | % | 11.00 - 16.00 |
| RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 45 | fL | 35.0 - 56.0 |
| MENTZERS INDEX by CALCULATED | 14.56 | RATIO | BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0 |
| GREEN & KING INDEX by CALCULATED | 21.8 | RATIO | BETA THALASSEMIA TRAIT: < = 65.0 IRON DEFICIENCY ANEMIA: > 65.0 |
| | | | |

WHITE BLOOD CELLS (WBCS)

| TOTAL LEUCOCYTE COUNT (TLC) | 5160 | /cmm | 400 | 0 - 11000 |
|--|------|------|------|-----------|
| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | | |
| NUCLEATED RED BLOOD CELLS (nRBCS) | NIL | | 0.00 | - 20.00 |
| by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & | | | | |
| MICROSCOPY | | | | |
| NUCLEATED RED BLOOD CELLS (nRBCS) % | NIL | % | < 10 | % |
| NUCALCULATED BY ALITOMATED HEMATOLOGY ANALYZED & | | | | |

DIFFERENTIAL LEUCOCYTE COUNT (DLC)



MICROSCOPY

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





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|---|---------------------|---------------|-------------------------------|
| NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 48 ^L | % | 50 - 70 |
| LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 42 ^H | % | 20 - 40 |
| EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 2 | % | 1 - 6 |
| MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 8 | % | 2 - 12 |
| BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 0 | % | 0 - 1 |
| ABSOLUTE LEUKOCYTES (WBC) COUNT | | | |
| ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 2477 | /cmm | 2000 - 7500 |
| ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 2167 | /cmm | 800 - 4900 |
| ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 103 | /cmm | 40 - 440 |
| ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 413 | /cmm | 80 - 880 |
| ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 0 | /cmm | 0 - 110 |
| PLATELETS AND OTHER PLATELET PREDICTIVE MARKE | RS. | | |
| PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 120000 ^L | /cmm | 150000 - 450000 |
| PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 0.13 | % | 0.10 - 0.36 |
| MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 13 ^H | fL | 6.50 - 12.0 |
| PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 49000 | /cmm | 30000 - 90000 |
| PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 50.3 ^H | % | 11.0 - 45.0 |
| PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 15.9 | % | 15.0 - 17.0 |
| ADVICE | KINDLY CORRELA | TE CLINICALLY | |



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NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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Test Name Value Unit **Biological Reference interval**

RECHECKED

CLIENT CODE.



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GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): 5.7 % 4.0 - 6.4

WHOLE BLOOD

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE 116.89 mg/dL 60.00 - 140.00

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

INTERPRETATION:

| AS PER AMERICAN DIABETES ASSOCIATION (ADA): | | | | |
|---|---------------------|-------------------|--|--|
| REFERENCE GROUP | GLYCOSYLATED HEMOGL | OGIB (HBAIC) in % | | |
| Non diabetic Adults >= 18 years | <5.7 | | | |
| At Risk (Prediabetes) | 5.7 – 6. | 4 | | |
| Diagnosing Diabetes | >= 6.5 | | | |
| | Age > 19 Years | | | |
| | Goals of Therapy: | < 7.0 | | |
| Therapeutic goals for glycemic control | Actions Suggested: | >8.0 | | |
| | Age < 19 Years | | | |
| | Goal of therapy: | <7.5 | | |

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-spienctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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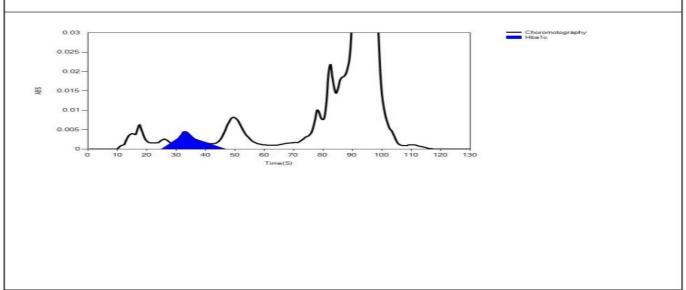
Test Name Value Unit **Biological Reference interval**

REPORTING DATE

LIFOTRONIC Graph Report

| Name : | Case: | Patient Type : | Test Date: 17/07/2024 14:02:48 |
|---------|-------------|-------------------------------|--------------------------------|
| Age: | Department: | Sample Type: Whole Blood EDTA | Sample ld: 01513277 |
| Gender: | | | Total Area: 15190 |

| Peak Name | Retention Time(s) | Absorbance | Area | Result (Area %) |
|-----------|-------------------|------------|-------|-----------------|
| HbA0 | 68 | 4661 | 13749 | 88.6 |
| HbA1c | 36 | 82 | 881 | 5.7 |
| La1c | 28 | 19 | 222 | 1.4 |
| HbF | 19 | 26 | 40 | 0.2 |
| Hba1b | 12 | 63 | 166 | 1.1 |
| Hba1a | 11 | 40 | 132 | 0.8 |





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CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit **Biological Reference interval**

ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)

0 - 20

by MODIFIED WESTERGREN AUTOMATED METHOD

INTERPRETATION:

- 1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
- 2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
- 3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus CONDITION WITH LOW ESR

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis), and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

- ESR and C reactive protein (C-RP) are both markers of inflammation.
 Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
 CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.
 If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
- 5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
- 6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it



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Test Name Value Unit **Biological Reference interval**

CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE FASTING (F)

97.23 GLUCOSE FASTING (F): PLASMA mg/dL NORMAL: < 100.0

by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

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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| Test Name | Value | Unit | Biological Reference interval |
|--|---------------------|---------|--|
| | LIPID PROFILE | : BASIC | |
| CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP | 192.23 | mg/dL | OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0 |
| TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC) | 150.11 ^H | mg/dL | OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0 |
| HDL CHOLESTEROL (DIRECT): SERUM by SELECTIVE INHIBITION | 61.96 | mg/dL | LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0 |
| LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY | 100.25 | 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 CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY | 130.27 ^H | 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 CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY | 30.02 | mg/dL | 0.00 - 45.00 |
| TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY | 534.57 | mg/dL | 350.00 - 700.00 |
| CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY | 3.1 | RATIO | LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 |
| LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY | 1.62 | RATIO | LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0 |



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TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY 2.42^L

RATIO

3.00 - 5.00

INTERPRETATION:

1. Measurements in the same patient can show physiological analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.

2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.

3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available

to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.

4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL &Non

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: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY | 1.33 ^H | mg/dL | INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20 |
|---|-------------------|-------|---|
| BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY | 0.45 ^H | mg/dL | 0.00 - 0.40 |
| BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY | 0.88 | mg/dL | 0.10 - 1.00 |
| SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE | 29.72 | U/L | 7.00 - 45.00 |
| SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE | 27.56 | U/L | 0.00 - 49.00 |
| AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY | 1.08 | RATIO | 0.00 - 46.00 |
| ALKALINE PHOSPHATASE: SERUM by Para nitrophenyl phosphatase by amino methyl propanol | 72 | U/L | 40.0 - 150.0 |
| GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY | 22 | U/L | 0.00 - 55.0 |
| TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY | 7.22 | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM by BROMOCRESOL GREEN | 4.51 | gm/dL | 3.50 - 5.50 |
| GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY | 2.71 | gm/dL | 2.30 - 3.50 |
| A: G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY | 1.66 | 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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 : 17/Jul/2024 07:39AM

CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 17/Jul/2024 11:23AM

CLIENT ADDRESS: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value 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).

PROGNOSTIC SIGNIFICANCE:

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



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



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NAME : Mr. RAJEEV GUPTA

AGE/ GENDER : 56 YRS/MALE **PATIENT ID** : 1551632

COLLECTED BY : REG. NO./LAB NO. : 012407170001

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 : 17/Jul/2024 12:20PM

CLIENT ADDRESS: 6349/1, NICHOLSON ROAD, AMBALA CANTT

| Test Name | Value | Unit | Biological Reference interval |
|--|--------------------|---------------|-------------------------------|
| | KIDNEY FUNCTION TE | ST (COMPLETE) | |
| UREA: SERUM by urease - glutamate dehydrogenase (gldh) | 23.77 | mg/dL | 10.00 - 50.00 |
| CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY | 1.05 | mg/dL | 0.40 - 1.40 |
| BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY | 11.11 | mg/dL | 7.0 - 25.0 |
| BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM | 10.58 | RATIO | 10.0 - 20.0 |
| by CALCULATED, SPECTROPHOTOMETRY UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY | 22.64 | RATIO | |
| URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE | 6.5 | mg/dL | 3.60 - 7.70 |
| CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY | 9.3 | mg/dL | 8.50 - 10.60 |
| PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY | 3.26 | mg/dL | 2.30 - 4.70 |
| ELECTROLYTES | | | |
| SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE) | 140.1 | mmol/L | 135.0 - 150.0 |
| POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE) | 4.22 | mmol/L | 3.50 - 5.00 |
| CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE) | 105.07 | mmol/L | 90.0 - 110.0 |
| ESTIMATED GLOMERULAR FILTERATION RATE | | | |
| ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM | 83.3 | | |

by CALCULATED INTERPRETATION:

To differentiate between pre- and post renal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

- 1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased glomerular filtration rate.
- 2. Catabolic states with increased tissue breakdown.



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NAME : Mr. RAJEEV GUPTA

AGE/ GENDER : 56 YRS/MALE **PATIENT ID** : 1551632

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Test Name Value Unit **Biological Reference interval**

- 3. GI haemorrhage.
- 4. High protein intake.
- 5. Impaired renal function plus
- 6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
- 7. Urine reabsorption (e.g. ureter colostomy)
- 8. Reduced muscle mass (subnormal creatinine production)
- 9. Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

- 1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
- 2. Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN:

- 1. Acute tubular necrosis.
- 2. Low protein diet and starvation.
- 3. Severe liver disease.
- 4. Other causes of decreased urea synthesis.
- 5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
- 6. Inherited hyperammonemias (urea is virtually absent in blood).
- 7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.
- 8. Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

- 1. Phenacimide therapy (accelerates conversion of creatine to creatinine).
- 2. Rhabdomyolysis (releases muscle creatinine).
- 3. Muscular patients who develop renal failure.

INAPPROPIATE RATIO:

- 1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
- 2. Cephalosporin therapy (interferes with creatinine measurement). ESTIMATED GLOMERULAR FILTERATION RATE:

| ESTIMINATED GEOMEROES IN THE TENTITION NAME: | | | |
|--|--------------------------|-----------------------|--------------------------|
| CKD STAGE | DESCRIPTION | GFR (mL/min/1.73m2) | ASSOCIATED FINDINGS |
| G1 | Normal kidney function | >90 | No proteinuria |
| G2 | Kidney damage with | >90 | Presence of Protein, |
| | normal or high GFR | | Albumin or cast in urine |
| G3a | Mild decrease in GFR | 60 -89 | |
| G3b | Moderate decrease in GFR | 30-59 | |
| G4 | Severe decrease in GFR | 15-29 | |
| G5 | Kidney failure | <15 | |



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Test Name Value Unit **Biological Reference interval**

COMMENTS:

CLIENT CODE.

1. 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.

2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012

3. 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

REPORTING DATE

4. eGFR category G1 OR G2 does not fullfill 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).

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 interval |
|--|--------------|-------|-------------------------------|
| | IRON PROFILE | | |
| IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY | 87.1 | μg/dL | 65.0 - 175.0 |
| UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY | 247.9 | μg/dL | 150.0 - 336.0 |
| TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETERY | 335 | μg/dL | 230 - 430 |
| %TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE) | 26 | % | 15.0 - 50.0 |
| TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE) | 237.85 | mg/dL | 200.0 - 350.0 |

INTERPRETATION:-

| 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 |

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.
- 2. 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):

 1. It is essential to isolate iron deficiency anemia, is severely contra-indicated in Thalassemia.

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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 Reference interval

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM 0.943 ng/mL 0.35 - 1.93

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROXINE (T4): SERUM 7.81 $\mu gm/dL$ 4.87 - 12.60

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROID STIMULATING HORMONE (TSH): SERUM 1.083 μIU/mL 0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

TSH levels are subject to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50%. Hence time of the day has influence on the measured serum TSH concentrations. TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4) and trilodothyronine (T3). Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction (hyperthyroidism) of T4 and/or T3.

| CLINICAL CONDITION | T3 | T4 | TSH |
|------------------------------|-----------------------|-----------------------|---------------------------------|
| Primary Hypothyroidism: | Reduced | Reduced | Increased (Significantly) |
| Subclinical Hypothyroidism: | Normal or Low Normal | Normal or Low Normal | High |
| Primary Hyperthyroidism: | Increased | 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, 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 (eq. phenytoin , salicylates).
- 3. Serum T4 levles 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 hypothroidism, pregnancy, phenytoin therapy.

| TRIIODOTHYRONINE (T3) | | THYROXINE (T4) | | THYROID STIMULATING HORMONE (TSH) | |
|-----------------------|-----------------------------|-------------------|-----------------------------|-----------------------------------|------------------------------|
| Age | Refferance Range (ng/mL) | Age | Refferance Range (μg/dL) | Age | Reference Range (μΙυ/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 |



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| Test Name | | | Value | Unit | | Biolog | ical Reference interval |
|---------------------|---------------|-----------------------|------------------|---------------------|-------------|--------|-------------------------|
| 6 - 12 Months | 0.74 - 2.40 | 6 - 12 Months | 7.10 – 16.16 | 6 – 12 Months | 0.70 - 7.00 | | |
| 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 | | |
| | RECON | IMENDATIONS OF TSH LE | VELS DURING PREG | SNANCY (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, idonie 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 goitre & Thyroiditis.
- $2. Over \ replacement \ of \ thyroid \ harmone \ in \ treatment \ of \ hypothyroid ism.$
- 3. Autonomously functioning Thyroid adenoma
- 4. Secondary pituatary or hypothalmic 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



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Test Name Value Unit **Biological Reference interval**

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM 39.5 ng/mL DEFICIENCY: < 20.0

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) INSUFFICIENCY: 20.0 - 30.0

SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0

INTERPRETATION:

| DEFICIENT: | < 20 | ng/mL |
|------------------|----------|-------|
| INSUFFICIENT: | 21 - 29 | ng/mL |
| PREFFERED RANGE: | 30 - 100 | ng/mL |
| INTOXICATION: | > 100 | ng/mL |

1. Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.

2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.

3. Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).

4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

- 1.Lack of sunshine exposure.
- 2.Inadequate intake, malabsorption (celiac disease)
 3.Depressed Hepatic Vitamin D 25- hydroxylase activity
- 4. Secondary to advanced Liver disease
- 5. Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- 6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.
- 1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.



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Test Name Value Unit Biological Reference interval

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM 191 pg/mL 190.0 - 890.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:-

| INCREASED VITAMIN B12 | DECREASED VITAMIN B12 |
|-------------------------------|---|
| 1.Ingestion of Vitamin C | 1.Pregnancy |
| 2.Ingestion of Estrogen | 2.DRUGS:Aspirin, Anti-convulsants, Colchicine |
| 3.Ingestion of Vitamin A | 3.Ethanol Igestion |
| 4.Hepatocellular injury | 4. Contraceptive Harmones |
| 5.Myeloproliferative disorder | 5.Haemodialysis |
| 6.Uremia | 6. Multiple Myeloma |

- 1. Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
- 2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
- 3. The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
- 4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg. gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
- 5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
- 6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
- 7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.

 NOTE:A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.



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Test Name Value Unit Biological Reference interval

CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

PHYSICAL EXAMINATION

QUANTITY RECIEVED 10 ml by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

COLOUR AMBER YELLOW PALE YELLOW

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

TRANSPARANCY CLEAR CLEAR

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SPECIFIC GRAVITY

1.002 - 1.030

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CHEMICAL EXAMINATION

REACTION ACIDIC

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PROTEIN Negative NEGATIVE (-ve)

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SUGAR Negative NEGATIVE (-ve)
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pH <=5.0 5.0 - 7.5

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BILIRUBIN Negative NEGATIVE (-ve)

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NITRITE Negative NEGATIVE (-ve)

UROBILINOGEN Normal EU/dL 0.2 - 1.0

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KETONE BODIES

Negative

NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

BLOOD Negative NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

ASCORBIC ACID NEGATIVE (-ve) NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

MICROSCOPIC EXAMINATION



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





CLIENT CODE.

KOS Diagnostic Lab

(A Unit of KOS Healthcare)



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

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

: 17/Jul/2024 09:51AM

NAME : Mr. RAJEEV GUPTA

AGE/ GENDER PATIENT ID : 56 YRS/MALE : 1551632

COLLECTED BY REG. NO./LAB NO. : 012407170001

REFERRED BY **REGISTRATION DATE** : 17/Jul/2024 06:22 AM **COLLECTION DATE** BARCODE NO. :01513277 : 17/Jul/2024 07:39AM

: KOS DIAGNOSTIC LAB **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

| Test Name | Value | Unit | Biological Reference interval |
|--|----------------|------|--------------------------------------|
| RED BLOOD CELLS (RBCs) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | /HPF | 0 - 3 |
| PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | 2-4 | /HPF | 0 - 5 |
| EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | 0-3 | /HPF | ABSENT |
| CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | NEGATIVE (-ve) | | NEGATIVE (-ve) |
| TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT | ABSENT | | ABSENT |

REPORTING DATE

End Of Report



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

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



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