



| | Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar | obiology) | | (Pathology) | |
|---|---|--------------------|--------------------------|--|-------|
| NAME | : Mr. DAVINDER ARORA | | | | |
| AGE/ GENDER | : 47 YRS/MALE | | PATIENT ID | : 71310 | |
| COLLECTED BY | : | | REG. NO./LAB NO. | : 012408020005 | |
| REFERRED BY | : CENTRAL PHOENIX CLUB (AMBAI | LA CANTT) | REGISTRATION DATE | : 02/Aug/2024 07:54 AM | |
| BARCODE NO. | :01514278 | | COLLECTION DATE | : 02/Aug/2024 10:10AM | |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 02/Aug/2024 08:42AM | |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMB/ | ALA CANTT | | | |
| Test Name | | Value | Unit | Biological Reference inte | erval |
| | SWAST | THYA WE | LLNESS PANEL: GT | | |
| | COM | IPLETE BL | OOD COUNT (CBC) | | |
| RED BLOOD CELLS (R | BCS) COUNT AND INDICES | | | | |
| HAEMOGLOBIN (HB) | | 13.6 | gm/dL | 12.0 - 17.0 | |
| RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i> PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i> MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i> | | 6.8 ^H | Millions/ | /cmm 3.50 - 5.00 | |
| | | 43.7 | % | 40.0 - 54.0 | |
| | | 64.3 ^L | fL | 80.0 - 100.0 | |
| MEAN CORPUSCULA | R HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER | 19.9 ^L | Pg | 27.0 - 34.0 | |
| | R HEMOGLOBIN CONC. (MCHC) | 31 ^L | g/dL | 32.0 - 36.0 | |
| by CALCULATED BY A | ION WIDTH (RDW-CV) automated hematology analyzer | 16.5 ^H | % | 11.00 - 16.00 | |
| | ION WIDTH (RDW-SD) utomated hematology analyzer | 39.2 | fL | 35.0 - 56.0 | |
| MENTZERS INDEX by CALCULATED | | 9.46 | RATIO | BETA THALASSEMIA TRA IRON DEFICIENCY ANEM | |
| GREEN & KING INDE | X | 15.52 | RATIO | BETA THALASSEMIA TRA 65.0 IRON DEFICIENCY ANEM | |
| WHITE BLOOD CELLS | <u>s (WBCS)</u> | | | | |
| TOTAL LEUCOCYTE C | OUNT (TLC) / by sf cube & microscopy | 11630 ^H | /cmm | 4000 - 11000 | |
| NUCLEATED RED BLC | | NIL | | 0.00 - 20.00 | |
| MICROSCOPY | UTOMATED HEMATOLOGY ANALYZER & | NIL | % | < 10 % | |
| DIFFERENTIAL LEUCO | <u>DCYTE COUNT (DLC)</u> | | | | |



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

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Dr. Yugam Chopra

MD (Pathology)

Dr. Vinay Chopra

MD (Pathology & Microbiology)

| | Chairman & Consul | tant Pathologis | st CEO & Consultant | Pathologist |
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| | | | | |
| Test Name | | Value | Unit | Biological Reference interval |
| NEUTROPHILS | | 59 | % | 50 - 70 |
| LYMPHOCYTES | Y BY SF CUBE & MICROSCOPY | 32 | % | 20 - 40 |
| EOSINOPHILS | BY SF CUBE & MICROSCOPY | 4 | % | 1 - 6 |
| MONOCYTES | BY SF CUBE & MICROSCOPY | 5 | % | 2 - 12 |
| BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | 0 | % | 0 - 1 |
| ABSOLUTE LEUKOCY | | | | |
| ABSOLUTE NEUTROP | PHIL COUNT Y BY SF CUBE & MICROSCOPY | 6862 | /cmm | 2000 - 7500 |
| ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | 3722 | /cmm | 800 - 4900 |
| ABSOLUTE EOSINOP | HIL COUNT Y by sf cube & microscopy | 465 ^H | /cmm | 40 - 440 |
| ABSOLUTE MONOCY | TE COUNT / by sf cube & microscopy | 582 | /cmm | 80 - 880 |
| ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | 0 | /cmm | 0 - 110 |
| | IER PLATELET PREDICTIVE MARKE | RS. | | |
| PLATELET COUNT (PL | .T) | 291000 | /cmm | 150000 - 450000 |
| PLATELETCRIT (PCT) | OCUSING, ELECTRICAL IMPEDENCE | 0.31 | % | 0.10 - 0.36 |

by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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|---|--------|---|
| | yhopra | , |
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97000^H

33.5

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

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fL

%

%

/cmm

6.50 - 12.0

11.0 - 45.0

15.0 - 17.0

30000 - 90000

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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, A | AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | GL | YCOSYLATED H | AEMOGLOBIN (HBA1C) | |
| GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY) | | 9.1 ^H | % | 4.0 - 6.4 |
| ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY) INTERPRETATION: | | 214.47 ^H | mg/dL | 60.00 - 140.00 |
| | AS PER AMERICAN DIAB | | | |
| REFERENCE GROUP | | GLYCOSYLATED HEMOGLOGIB (HBAIC) in % | | in % |
| | etic Adults >= 18 years | <5.7 | | |
| | Risk (Prediabetes) gnosing Diabetes | 5.7 - 6.4 | | |
| Dia | | | Age > 19 Years | |
| | | Goals of The | | .0 |
| Therapeutic | goals for glycemic control | Actions Sugg | | |
| | | | Age < 19 Years | |
| | | Goal of the | | |

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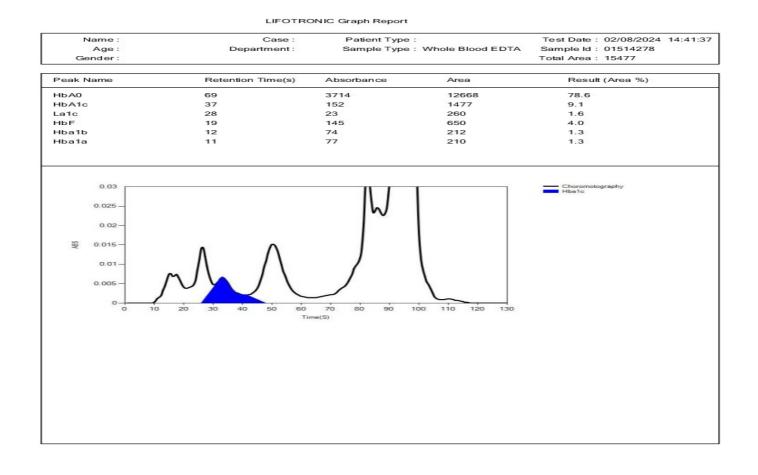
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| Test Name | Value | Unit | Biological Reference interval |







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| Test Name | | Value | Unit | Biological Reference interval |
| | ERYTH | ROCYTE SEDI | MENTATION RATE (ESF | 8) |
| by MODIFIED WESTER INTERPRETATION: 1. ESR is a non-specifi mmune disease, but 2. An ESR can be affect as C-reactive protein 3. This test may also I systemic lupus erythe CONDITION WITH LOV A low ESR can be seen (polycythaemia), sign as sickle cells in sickle NOTE: 1. ESR and C - reactive 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevate 5. Women tend to har 6. Drugs such as dext | does not tell the health practitior cted by other conditions besides i be used to monitor disease activi- matosus V ESR n with conditions that inhibit the ificantly high white blood cell col e cell anaemia) also lower the ES e protein (C-RP) are both markers s not change as rapidly as does C by as many other factors as is ESF ed, it is typically a result of two ty ve a higher ESR, and menstruation | ner exactly wher inflammation. For ty and response normal sedimer unt (leucocytosi RR, of inflammation RP, either at the R , making it a be ypes of proteins, n and pregnancy | re the inflammation is in the or this reason, the ESR is typ to therapy in both of the ak ntation of red blood cells, su s) , and some protein abnor h. e start of inflammation or as tter marker of inflammation globulins or fibrinogen. can cause temporary eleval | vicallý used in conjunction with other test such pove diseases as well as some others, such as the as a high red blood cell count malities. Some changes in red cell shape (such it resolves. |



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| CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE FASTING (F) GLUCOSE FASTING (F): PLASMA 161.24 ^H mg/dL NORMAL: < 100.0 by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) PREDIABETIC: 100.0 - 125.0 | CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, A | MBALA CANTT | | |
| GLUCOSE FASTING (F): PLASMA NORMAL: < 100.0 | Test Name | | | | - |
| GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)161.24 ^H mg/dLNORMAL: < 100.0PREDIABETIC: 100.0 - 125.0 | | CLINI | | | Ŷ |
| by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) PREDIABETIC: 100.0 - 125.0 | | | | | |
| DIABETIC > OR = 126.0 | | | 161.24 ⁿ | mg/dL | |

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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| SO 9001 : 2008 CERTIFIED LAB | | | EXCELLENCE IN HEALTHCARE & DIAGNOSTICS | | | |
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| Test Name | | Value | Unit | Biological Reference interval | | |
| | | מם חומן ו | OFILE : BASIC | | | |
| CHOLESTEROL TOTAL by CHOLESTEROL OXI | | 140.01 | mg/dL | OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0 | | |
| TRIGLYCERIDES: SER | UM HATE OXIDASE (ENZYMATIC) | 168.01 ^H | mg/dL | OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0 | | |
| HDL CHOLESTEROL (E | | 31.38 | mg/dL | LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0 | | |
| LDL CHOLESTEROL: SI by CALCULATED, SPEC | | 75.03 | 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 CHOLESTER by CALCULATED, SPEC | | 108.63 | 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: by calculated, spec | | 33.6 | mg/dL | 0.00 - 45.00 | | |
| TOTAL LIPIDS: SERUN | 1 | 448.03 | mg/dL | 350.00 - 700.00 | | |
| CHOLESTEROL/HDL R by CALCULATED, SPEC | ATIO: SERUM | 4.46 ^H | 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: SERI | | 2.39 | RATIO | LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0 | | |
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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AMBALA CAN | TT | |
| Test Name | Value | Unit | Biological Reference interval |
| TRIGLYCERIDES/HD | 5.55 | 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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Dr. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. DAVINDER ARORA AGE/ GENDER : 47 YRS/MALE **PATIENT ID** :71310 **COLLECTED BY** :012408020005 REG. NO./LAB NO. **REFERRED BY** : CENTRAL PHOENIX CLUB (AMBALA CANTT) **REGISTRATION DATE** :02/Aug/2024 07:54 AM **BARCODE NO.** :01514278 **COLLECTION DATE** :02/Aug/2024 10:10AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :02/Aug/2024 10:58AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Value Unit **Biological Reference interval** Test Name LIVER FUNCTION TEST (COMPLETE) **BILIRUBIN TOTAL: SERUM** 1.2 mg/dL INFANT: 0.20 - 8.00 by DIAZOTIZATION, SPECTROPHOTOMETRY ADULT: 0.00 - 1.20 **BILIRUBIN DIRECT (CONJUGATED): SERUM** 0.00 - 0.40 0.48^H mg/dL by DIAZO MODIFIED, SPECTROPHOTOMETRY BILIRUBIN INDIRECT (UNCONJUGATED): SERUM 0.72 0.10 - 1.00 mg/dL by CALCULATED, SPECTROPHOTOMETRY SGOT/AST: SERUM 71.4^H U/L 7.00 - 45.00 by IFCC, WITHOUT PYRIDOXAL PHOSPHATE U/L 0.00 - 49.00 SGPT/ALT: SERUM 128^H by IFCC, WITHOUT PYRIDOXAL PHOSPHATE AST/ALT RATIO: SERUM 0.56 RATIO 0.00 - 46.00 by CALCULATED, SPECTROPHOTOMETRY ALKALINE PHOSPHATASE: SERUM 105.54 U/L 40.0 - 130.0 by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM 234.97^H U/L 0.00 - 55.0 by SZASZ, SPECTROPHTOMETRY TOTAL PROTEINS: SERUM 6.79 gm/dL 6.20 - 8.00 by BIURET, SPECTROPHOTOMETRY 3.9 ALBUMIN: SERUM gm/dL 3.50 - 5.50 by BROMOCRESOL GREEN **GLOBULIN: SERUM** 2.89 gm/dL 2.30 - 3.50 by CALCULATED, SPECTROPHOTOMETRY RATIO 1.35 1.00 - 2.00

A : G RATIO: SERUM 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 & Mi Chairman & Consult | crobiology) | Dr. Yugan MD CEO & Consultant | (Pathology) |
|--------------------|---|----------------------|-------------------------------------|-------------------------------|
| NAME | : Mr. DAVINDER ARORA | | | |
| AGE/ GENDER | : 47 YRS/MALE | PA | ATIENT ID | : 71310 |
| COLLECTED BY | : | RI | EG. NO./LAB NO. | : 012408020005 |
| REFERRED BY | : CENTRAL PHOENIX CLUB (AMB | ALA CANTT) RI | EGISTRATION DATE | : 02/Aug/2024 07:54 AM |
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| 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).

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



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| Test Name | | Value | Unit | Biological Reference interval |
| | KI | DNEY FUNCTION TES | T (COMPLETE) | |
| UREA: SERUM | | 14.37 | mg/dL | 10.00 - 50.00 |
| - | ATE DEHYDROGENASE (GLDH) | 0.00 | no o (all | 0.40 1.40 |
| CREATININE: SERUN by ENZYMATIC, SPEC | | 0.88 | mg/dL | 0.40 - 1.40 |
| BLOOD UREA NITRO | GEN (BUN): SERUM | 6.71 ^L | mg/dL | 7.0 - 25.0 |
| by CALCULATED, SPI | <i>ectrophotometry</i>)GEN (BUN)/CREATININE | = col | RATIO | 10.0 - 20.0 |
| RATIO: SERUM | GEN (DUN)/CREATININE | 7.63 ^L | KATIO | 10.0 - 20.0 |
| by CALCULATED, SPI | | | | |
| UREA/CREATININE F by CALCULATED, SPE | | 16.33 | RATIO | |
| URIC ACID: SERUM | | 3.63 | mg/dL | 3.60 - 7.70 |
| by URICASE - OXIDAS | SE PEROXIDASE | | | |
| CALCIUM: SERUM | | 9.33 | mg/dL | 8.50 - 10.60 |
| by ARSENAZO III, SPE PHOSPHOROUS: SER | | 3.03 | mg/dL | 2.30 - 4.70 |
| | DATE, SPECTROPHOTOMETRY | | | 2.00 |
| ELECTROLYTES | | | | |
| SODIUM: SERUM | | 141.3 | mmol/L | 135.0 - 150.0 |
| by ISE (ION SELECTIV POTASSIUM: SERUM | | 4.4 | mmol/L | 3.50 - 5.00 |
| by ISE (ION SELECTIV | | 4.4 | THITIOI/L | 3.30 - 3.00 |
| CHLORIDE: SERUM | | 105.98 | mmol/L | 90.0 - 110.0 |
| by ISE (ION SELECTIV | 'E ELECTRODE) RULAR FILTERATION RATE | | | |
| | | 10/ 7 | | |
| eGFR): SERUM | RULAR FILTERATION RATE | 106.7 | | |
| 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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| | | IA CLUD (AMDALA C | | | : 02/Aug/2024 07:5 | |
| BARCODE NO. | :01514278 | | COLLECTIO | | : 02/Aug/2024 10:10 | |
| CLIENT CODE. | : KOS DIAGNOSTIC | | REPORTING | DATE | : 02/Aug/2024 10:5 | 8AM |
| CLIENT ADDRESS | : 6349/1, NICHOLS | SON ROAD, AMBALA | CANTT | | | |
| Test Name | | Va | lue | Unit | Biological | Reference interval |
| Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia | superimposed on rer | inine production) rticoids) CREATININE LEVELS: rtionately more than nal disease. | ocreatinine) (e.g. obst | ructive uropa | thy). | |
| Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia PCREASED RATIO (Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. PCREASED RATIO (| (e.g. ureter colostom ass (subnormal creat tetracycline, glucoco io:1) WITH ELEVATED a (BUN rises dispropol superimposed on rer i0:1) WITH DECREASE osis. ad starvation. e. creased urea synthes urea rather than creat monemias (urea is vi of inappropiate antidi i0:1) WITH INCREASEE py (accelerates conve | inine production) rticoids) CREATININE LEVELS: rtionately more than hal disease. D BUN : sis. atinine diffuses out of rtually absent in bloo uretic harmone) due D CREATININE: ersion of creatine to | of extracellular fluid). od). e to tubular secretion o | | thy). | |
| Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia CECREASED RATIO (Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. Pregnancy. Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido | (e.g. ureter colostom ass (subnormal creat tetracycline, glucoco io:1) WITH ELEVATED a (BUN rises dispropol superimposed on rer i0:1) WITH DECREASE osis. and starvation. e. creased urea synthes urea rather than creat monemias (urea is vi of inappropiate antidi i0:1) WITH INCREASEE py (accelerates conve eleases muscle creat who develop renal fa : sis (acetoacetate cau | inine production) rticoids) CREATININE LEVELS: rtionately more than hal disease. D BUN : sis. atinine diffuses out of rtually absent in blod uretic harmone) due D CREATININE: ersion of creatine to inine). hilure. | of extracellular fluid). od). to tubular secretion o creatinine). | of urea. | | al ratio when dehydratio |
| Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia DECREASED RATIO (<2 Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome of Pregnancy. DECREASED RATIO (<2 Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther | (e.g. ureter colostom ass (subnormal creat tetracycline, glucoco io:1) WITH ELEVATED (BUN rises dispropol superimposed on rer i0:1) WITH DECREASE osis. Ind starvation. E. creased urea synthes urea rather than creat monemias (urea is vi of inappropiate antidi i0:1) WITH INCREASEE py (accelerates conve eleases muscle creat who develop renal fa : sis (acetoacetate cau creased BUN/creatin rapy (interferes with c | inine production) rticoids) CREATININE LEVELS: rtionately more than hal disease. D BUN : sis. atinine diffuses out of rtually absent in bloc uretic harmone) due D CREATININE: ersion of creatine to inine). hilure. uses false increase in ine ratio). creatinine measurem | of extracellular fluid). od). to tubular secretion of creatinine). creatinine with certa | of urea. | | al ratio when dehydratio |
| Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia DECREASED RATIO (<2 Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome of Pregnancy. DECREASED RATIO (<2 Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther | (e.g. ureter colostom ass (subnormal creat tetracycline, glucoco 0:1) WITH ELEVATED a (BUN rises dispropol superimposed on rer 10:1) WITH DECREASE osis. ad starvation. e. creased urea synthes urea rather than creat monemias (urea is vi of inappropiate antidi 10:1) WITH INCREASEL py (accelerates conve eleases muscle creat who develop renal fa : sis (acetoacetate cau creased BUN/creatin rapy (interferes with o JLAR FILTERATION RA | inine production) rticoids) CREATININE LEVELS: rtionately more than hal disease. D BUN : sis. atinine diffuses out of rtually absent in bloc uretic harmone) due D CREATININE: ersion of creatine to inine). hilure. uses false increase in ine ratio). creatinine measurem | of extracellular fluid). od). to tubular secretion of creatinine). creatinine with certa | of urea. in methodolo | | al ratio when dehydratio |
| Reduced muscle mu | (e.g. ureter colostom ass (subnormal creat tetracycline, glucoco io:1) WITH ELEVATED a (BUN rises dispropol superimposed on rer i0:1) WITH DECREASE osis. and starvation. e. creased urea synthes urea rather than creat monemias (urea is vi of inappropiate antidi i0:1) WITH INCREASEE py (accelerates conve eleases muscle creat who develop renal fa : sis (acetoacetate cau creased BUN/creatin rapy (interferes with o <u>JLAR FILTERATION RA</u> <u>DES</u> Normal k | inine production) rticoids) CREATININE LEVELS: rtionately more than hal disease. D BUN : sis. atinine diffuses out of rtually absent in blod uretic harmone) due D CREATININE: ersion of creatine to inine). hilure. uses false increase in ine ratio). creatinine measurem TE: SCRIPTION kidney function | of extracellular fluid). od). to tubular secretion of creatinine). creatinine with certa nent). | of urea. in methodolo | gies,resulting in norma SOCIATED FINDINGS No proteinuria | al ratio when dehydratio |
| Reduced muscle mu | (e.g. ureter colostom ass (subnormal creat tetracycline, glucoco io:1) WITH ELEVATED a (BUN rises dispropol superimposed on rer i0:1) WITH DECREASE osis. and starvation. e. creased urea synthes urea rather than creat monemias (urea is vi of inappropiate antidi i0:1) WITH INCREASEE py (accelerates conve eleases muscle creat who develop renal fa : sis (acetoacetate cau creased BUN/creatin rapy (interferes with o <u>JLAR FILTERATION RATE</u> Normal k | inine production) rticoids) CREATININE LEVELS: rtionately more than hal disease. D BUN : sis. atinine diffuses out of rtually absent in blod uretic harmone) due D CREATININE: ersion of creatine to inine). hilure. uses false increase in ine ratio). creatinine measurem TE: SCRIPTION | of extracellular fluid). od). to tubular secretion of creatinine). creatinine with certa nent). | of urea. in methodolo | gies,resulting in norma SOCIATED FINDINGS | al ratio when dehydratio |

Kidney failure



G3b

G4

G5

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Moderate decrease in GFR

Severe decrease in GFR

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30-59

15-29

<15









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

COMMENTS: 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 eGFR with Creatine CFP.

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 interval |
| | | ENDOC | RINOLOGY | |
| | TH | YROID FUN | CTION TEST: TOTAL | |
| TRIIODOTHYRONINE | E (T3): SERUM IESCENT MICROPARTICLE IMMUNOASSA | 0.952 (4 <i>Y</i>) | ng/mL | 0.35 - 1.93 |
| THYROXINE (T4): SE by CMIA (CHEMILUMI IMMUNOASSAY) | RUM NESCENT MICROPARTICLE | 4.43 ^L | µgm/dL | 4.87 - 12.60 |
| THYROID STIMULAT | ING HORMONE (TSH): SERUM IESCENT MICROPARTICLE IMMUNOASSA RASENSITIVE | 5.144 (4) | μlU/mL | 0.35 - 5.50 |

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, 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 (eg: 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.

| TRIIODOTH | (RONINE (T3) | THYROXI | NE (T4) | THYROID STIMUL | ATING 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 |





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

| Test Name | | | Value | Unit | t | Biological Reference interva |
|---------------------|---------------|-----------------------|------------------|---------------------|-------------|------------------------------|
| 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 | |
| | RECO | VIMENDATIONS OF TSH L | 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, 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 hypothyroidism.

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





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

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| | Dr. Vinay Ch MD (Pathology & Chairman & Cons | Microbiology) | | (Pathology) |
|-----------------------|--|---------------|-----------------------|------------------------------|
| NAME | : Mr. DAVINDER ARORA | | | |
| AGE/ GENDER | : 47 YRS/MALE | | PATIENT ID | : 71310 |
| COLLECTED BY | | | REG. NO./LAB NO. | : 012408020005 |
| | · | | | |
| REFERRED BY | : CENTRAL PHOENIX CLUB (Al | MBALA CANTT) | | : 02/Aug/2024 08:02 AM |
| BARCODE NO. | : 01514278 | | COLLECTION DATE | : 02/Aug/2024 10:10AM |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | | REPORTING DATE | : 02/Aug/2024 04:17PM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, A | AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interva |
| | | CLINICAL | PATHOLOGY | |
| | | OUTINE & MI | CROSCOPIC EXAMINA | TION |
| PHYSICAL EXAMINATI | | | | |
| | | 10 | ml | |
| | ANCE SPECTROPHOTOMETRY | 10 | ml | |
| COLOUR | | PALE YELL | OW | PALE YELLOW |
| | ANCE SPECTROPHOTOMETRY | | | |
| TRANSPARANCY | | HAZY | | CLEAR |
| by DIP STICK/REFLECT/ | ANCE SPECTROPHOTOMETRY | | | |
| SPECIFIC GRAVITY | | 1.02 | | 1.002 - 1.030 |
| - | ANCE SPECTROPHOTOMETRY | | | |
| CHEMICAL EXAMINAT | <u>FION</u> | | | |
| REACTION | | ACIDIC | | |
| | ANCE SPECTROPHOTOMETRY | | | |
| PROTEIN | | Negative | | NEGATIVE (-ve) |
| by DIP STICK/REFLECT | ANCE SPECTROPHOTOMETRY | | | |
| SUGAR | | 2+ | | NEGATIVE (-ve) |
| | ANCE SPECTROPHOTOMETRY | | | |
| OH | ANCE SPECTROPHOTOMETRY | 5.5 | | 5.0 - 7.5 |
| BILIRUBIN | ANCE SPECIROPHOTOMETRY | Negative | | NEGATIVE (-ve) |
| | ANCE SPECTROPHOTOMETRY | Negative | | NEGATIVE (-VE) |
| NITRITE | | Negative | | NEGATIVE (-ve) |
| | ANCE SPECTROPHOTOMETRY. | Nogativo | | |
| UROBILINOGEN | | Normal | EU/dL | 0.2 - 1.0 |
| by DIP STICK/REFLECT/ | ANCE SPECTROPHOTOMETRY | | | |
| KETONE BODIES | | Negative | | NEGATIVE (-ve) |
| | ANCE SPECTROPHOTOMETRY | | | |
| BLOOD | | Negative | | NEGATIVE (-ve) |
| | ANCE SPECTROPHOTOMETRY | NEO ATU | | |
| | | NEGATIVE | : (-ve) | NEGATIVE (-ve) |
| MICROSCOPIC EXAMI | ANCE SPECTROPHOTOMETRY | | | |



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

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

| NAME | : Mr. DAVINDER ARORA | | | | |
|--------------------|---------------------------------------|-------------|-----------|-----------|-------------------------------|
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| | | | | | / |
| Test Name | | Value | | Unit | Biological Reference interval |
| RED BLOOD CELLS (F | RBCs) Centrifuged urinary sediment | NEGATIVE | E (-ve) | /HPF | 0 - 3 |
| PUS CELLS | CENTRIFUGED URINARY SEDIMENT | 2-4 | | /HPF | 0 - 5 |
| EPITHELIAL CELLS | CENTRIFUGED URINARY SEDIMENT | 1-2 | | /HPF | ABSENT |
| CRYSTALS | | NEGATIVE | E (-ve) | | NEGATIVE (-ve) |

Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT CASTS NEGATIVE (-ve) NEGATIVE (-ve) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT BACTERIA NEGATIVE (-ve) NEGATIVE (-ve) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT NEGATIVE (-ve) NEGATIVE (-ve) OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT TRICHOMONAS VAGINALIS (PROTOZOA) ABSENT ABSENT

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

*** End Of Report ***





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

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