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NAME	**: Mr. RAM KARAN**		
AGE/ GENDER	: 44 YRS/MALE	**PATIENT ID**	: 1763807
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012502200022**
REFERRED BY	:	**REGISTRATION DATE**	: 20/Feb/2025 10:23 AM
BARCODE NO.	: 01525823	**COLLECTION DATE**	: 20/Feb/2025 10:50AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 20/Feb/2025 11:12AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

HAEMATOLOGY

COMPLETE BLOOD COUNT (CBC)

Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) *by CALORIMETRIC*	**5.2^{L}**	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	**1.9^{L}**	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**16.4^{L}**	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	86.5	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	27.4	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**31.7^{L}**	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**18.9^{H}**	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**60.9^{H}**	fL	35.0 - 56.0
MENTZERS INDEX *by CALCULATED*	45.53	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX *by CALCULATED*	86.14	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	4580	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) *by AUTOMATED 6 PART HEMATOLOGY ANALYZER*	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	NIL	%	< 10 %

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	69	%	50 - 70
LYMPHOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	**18^{L}**	%	20 - 40
EOSINOPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	4	%	1 - 6
MONOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	9	%	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*	3160	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	824	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	183	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	412	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.			
PLATELET COUNT (PLT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	229000	/cmm	150000 - 450000
PLATELETCRIT (PCT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	0.23	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	10	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	59000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	25.9	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	15.7	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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Test Name	Value	Unit	Biological Reference interval

GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	4.2	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	73.84	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):		
REFERENCE GROUP	**GLYCOSYLATED HEMOGLOGIB (HBAIC) in %**	
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.4	
Diagnosing Diabetes	>= 6.5	
Therapeutic goals for glycemic control	**Age > 19 Years**	
	Goals of Therapy:	< 7.0
	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 HbAIc. 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 appropiate.

4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications

5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution , given the pathological processes including anemia,increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.

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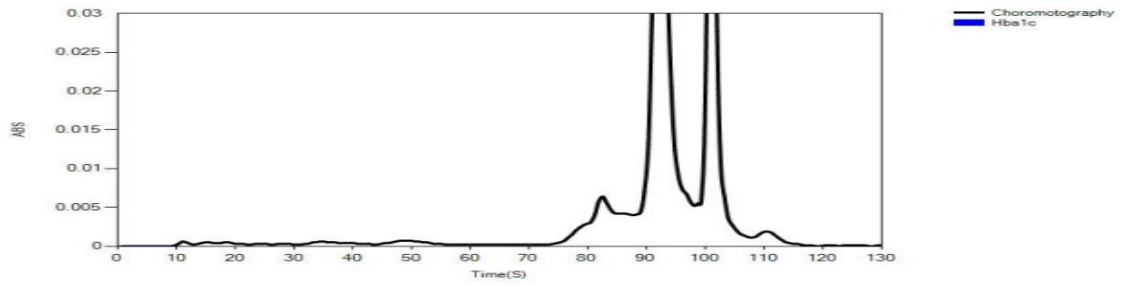
Test Name	Value	Unit	Biological Reference interval

LIFOTRONIC Graph Report

Name :	Case :	Patient Type :		Test Date : 20/02/2025 18:46:10
Age :	Department :	Sample Type : Whole Blood EDTA		Sample Id : 01525823
Gender :				Total Area : 0

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	00	0	0	00.0
HbA1c	00	0	0	0.0
La1c	00	0	0	0.0
HbF	00	0	0	0.0
Hba1b	00	0	0	0.0
Hba1a	00	0	0	0.0



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PERIPHERAL BLOOD SMEAR

TEST NAME:

PERIPHERAL BLOOD FILM/SMEAR (PBF)

RED BLOOD CELLS (RBC'S):

RBCs mostly appear normocytic & normochromic.Occ. polychromatic cells seen.No normoblastic activity noted.

WHITE BLOOD CELLS (WBC'S):

No immature leucocytes seen.

PLATELETS:

Platelets are adequate.

HEMOPARASITES:

NOT SEEN.

IMPRESSION:

Normocytic normochromic picture.

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Test Name	Value	Unit	Biological Reference interval
RETICULOCYTE COUNT			
RETICULOCYTE COUNT	2.4	%	0.5 - 2.5

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CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 20/Feb/2025 01:41PM
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Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F) AND POST PRANDIAL (PP)

GLUCOSE FASTING (F): PLASMA *by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)*	99.33	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0
GLUCOSE POST PRANDIAL (PP): PLASMA *by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)*	**168.02^{H}**	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > 0R = 200.0

INTERPRETATION:

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose below 100 mg/dL and post-prandial plasma glucose level below 140 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl and post-prandial plasma glucose level between 140 – 200 mg/dL is considered as glucose intolerant or pre diabetic. 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 and post-prandial plasma glucose level above 200 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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LIPID PROFILE : BASIC

Test Name	Value	Unit	Biological Reference interval
CHOLESTEROL TOTAL: SERUM *by CHOLESTEROL OXIDASE PAP*	128.25	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM *by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)*	97.21	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*	37.06	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	71.75	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*	91.19	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*	19.44	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	353.71	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	3.46	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0

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Test Name	Value	Unit	Biological Reference interval
LDL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.94	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**2.62^{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 HDL.
5. Additional testing for Apolipoprotein B, hsCRP,Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement

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LIVER FUNCTION TEST (COMPLETE)

Test Name	Value	Unit	Biological Reference interval
BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	0.44	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.14	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.3	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	10.2	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	13.8	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.74	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	**161^{H}**	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM *by SZASZ, SPECTROPHTOMETRY*	13.54	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	6.29	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	4.17	gm/dL	3.50 - 5.50
GLOBULIN: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**2.12^{L}**	gm/dL	2.30 - 3.50
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.97	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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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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Test Name	Value	Unit	Biological Reference interval
KIDNEY FUNCTION TEST (COMPLETE)			
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	**206.04**^{H}	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	**9.34**^{H}	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**96.28**^{H}	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	10.31	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	22.06	RATIO	
URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	**8.27**^{H}	mg/dL	3.60 - 7.70
CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	**8.45**^{L}	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM *by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY*	**4.93**^{H}	mg/dL	2.30 - 4.70
ELECTROLYTES			
SODIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	139.2	mmol/L	135.0 - 150.0
POTASSIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	**6.02**^{H}	mmol/L	3.50 - 5.00
CHLORIDE: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	104.4	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RATE			
ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM *by CALCULATED*	6.5		
NOTE 2	RESULT RECHECKED TWICE		
ADVICE	**KINDLY CORRELATE CLINICALLY**		

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

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Test Name	Value	Unit	Biological Reference interval

glomerular filtration rate.
2. Catabolic states with increased tissue breakdown.
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:

CKD STAGE	DESCRIPTION	GFR (mL/min/1.73m2)	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with normal or high GFR	>90	Presence of Protein , 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:

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 Cystatin C for confirmation of CKD
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).

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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IRON PROFILE

Test Name	Value	Unit	Biological Reference interval
IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	**190.32**^{H}	µg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM *by FERROZINE, SPECTROPHOTOMETERY*	**8.18**^{L}	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM *by SPECTROPHOTOMETERY*	**198.5**^{L}	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM *by CALCULATED, SPECTROPHOTOMETERY (FERENE)*	**95.88**^{H}	%	15.0 - 50.0
TRANSFERRIN: SERUM *by SPECTROPHOTOMETERY (FERENE)*	**140.94**^{L}	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 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 Reference interval

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.863	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	7.26	µgm/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	1.639	µIU/mL	0.35 - 5.50

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 triiodothyronine (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 (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.

TRIIODOTHYRONINE (T3)		THYROXINE (T4)		THYROID STIMULATING 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
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
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (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

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Test Name	Value	Unit	Biological Reference interval

INTACT PARATHYROID HORMONE (PTH)

INTACT PARATHROID HORMONE (PTH): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**613.7^{H}**	pg/mL	9.5 - 75.0

Intrepretation:-

Parathyroid hormone (PTH) is produced and secreted by the parathyroid glands, which are located along the posterior aspect of the thyroid gland. The serum calcium level regulates PTH secretion via negative feedback through the parathyroid calcium sensing receptor (CASR). Decreased calcium levels stimulate PTH release. Secreted PTH interacts with its specific type II G-protein receptor, causing rapid increases in renal tubular reabsorption of calcium and decreased phosphorus reabsorption. It also participates in long-term calciostatic functions by enhancing mobilization of calcium from bone and increasing renal synthesis of 1,25-dihydroxy vitamin D, which, in turn, increases intestinal calcium absorption.

The assay is useful for:

- Differential diagnosis of hypercalcemia
- Diagnosis of primary, secondary, and tertiary hyperparathyroidism
- Diagnosis of hypoparathyroidism
- Monitoring end-stage renal failure patients for possible renal osteodystrophy

Interpretation of results:

- An (appropriately) low PTH level and high phosphorus level in a hypercalcemic patient suggests that the hypercalcemia is not caused by PTH or PTH-like substances.
- An (appropriately) low PTH level with a low phosphorus level in a hypercalcemic patient suggests the diagnosis of paraneoplastic hypercalcemia.
- A low or normal PTH in a patient with hypocalcemia suggests hypoparathyroidism.

Low serum calcium and high PTH levels in a patient with normal renal function suggest resistance to PTH action (pseudohypoparathyroidism type 1a, 1b, 1c, or 2) or, very rarely, bio-ineffective PTH.

Elevated PTH value with a normal serum calcium in many cases in India is due to secondary hyperparathyroidism, primary cause being Vitamin D deficiency.

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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

HEPATITIS C VIRUS (HCV) ANTIBODY: TOTAL

HEPATITIS C ANTIBODY (HCV) TOTAL: SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.06	S/CO	NEGATIVE: < 1.00 POSITIVE: > 1.00
HEPATITIS C ANTIBODY (HCV) TOTAL RESULT *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	NON - REACTIVE		

INTERPRETATION:-

RESULT (INDEX)	REMARKS
< 1.00	NON - REACTIVE/NOT - DETECTED
> =1.00	REACTIVE/ASYMPTOMATIC/INFECTIVE STATE/CARRIER STATE.

Hepatitis C (HCV) is an RNA virus of Favivirus group transmitted via blood transfusions, transplantation, injection drug abusers, accidental needle punctures in healthcare workers, dialysis patients and rarely from mother to infant. 10 % of new cases show sexual transmission. As compared to HAV & HBV , chronic infection with HCV occurs in 85 % of infected individuals. In high risk population, the predictive value of Anti HCV for HCV infection is > 99% whereas in low risk populations it is only 25 %.

USES:

1. Indicator of past or present infection, but does not differentiate between Acute/ Chronic/Resolved Infection.
2. Routine screening of low and high prevelance population including blood donors.

NOTE:

1. False positive results are seen in Auto-immune disease, Rheumatoid Factor, HYpergammaglobulinemia, Paraproteinemia, Passive antibody transfer, Anti-idiotypes and Anti-superoxide dismutase.
2. False negative results are seen in early Acute infection, Immunosuppression and Immuno—incompetence.
3. *HCV-RNA PCR recommended in all reactive results to differentiate between past and present infection.*

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Test Name	Value	Unit	Biological Reference interval

ANTI HUMAN IMMUNODEFICIENCY VIRUS (HIV) DUO ULTRA WITH (P-24 ANTIGEN DETECTION)

HIV 1/2 AND P24 ANTIGEN: SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.11	S/CO	NEGATIVE: < 1.00 POSITIVE: > 1.00
HIV 1/2 AND P24 ANTIGEN RESULT *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	NON - REACTIVE		

INTERPRETATION:-

RESULT (INDEX)	REMARKS
< 1.00	NON - REACTIVE
> = 1.00	PROVISIONALLY REACTIVE

Non-Reactive result implies that antibodies to HIV 1/ 2 have not been detected in the sample . This menas that patient has either not been exposed to HIV 1/ 2 infection or the sample has been tested during the “window phase” i.e. before the development of detectable levels of antibodies. Hence a Non Reactive result does not exclude the possibility of exposure or infection with HIV 1/ 2.

RECOMMENDATIONS:

1. Results to be clinically correlated
2. Rarely falsenegativity/positivity may occur.

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Test Name	Value	Unit	Biological Reference interval

HEPATITIS B SURFACE ANTIGEN (HBsAg) ULTRA

HEPATITIS B SURFACE ANTIGEN (HBsAg): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.18	S/CO	NEGATIVE: < 1.0 POSITIVE: > 1.0
HEPATITIS B SURFACE ANTIGEN (HBsAg) RESULT *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	NON REACTIVE		

INTERPRETATION:

RESULT IN INDEX VALUE	REMARKS
< 1.30	NEGATIVE (-ve)
>=1.30	POSITIVE (+ve)

Hepatitis B Virus (HBV) is a member of the Hepadna virus family causing infection of the liver with extremely variable clinical features. Hepatitis B is transmitted primarily by body fluids especially serum and also spread effectively sexually and from mother to baby. In most individuals HBV hepatitis is self limiting, but 1-2 % normal adolescent and adults develop Chronic Hepatitis. Frequency of chronic HBV infection is 5-10% in immunocompromised patients and 80 % neonates. The initial serological marker of acute infection is HBsAg which typically appears 2-3 months after infection and disappears 12-20 weeks after onset of symtoms. Persistence of HBsAg for more than 6 months indicates carrier state or Chronic Liver disease.

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CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 20/Feb/2025 12:24PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**23.8[L]**	ng/mL	DEFICIENCY: < 20.0 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.

INCREASED:
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.*

*** End Of Report ***

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