

(A Unit of KOS Healthcare)



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

NAME : Mr. KIRAN PARKASH

AGE/ GENDER : 43 YRS/MALE **PATIENT ID** : 1553883

COLLECTED BY : REG. NO./LAB NO. : 012407190015

 REFERRED BY
 : 19/Jul/2024 08:19 AM

 BARCODE NO.
 : 01513408
 COLLECTION DATE
 : 19/Jul/2024 08:26 AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 19/Jul/2024 09:07 AM

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

Test Name Value Unit Biological Reference interval

SWASTHYA WELLNESS PANEL: GT COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by calorimetric	14.5	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	6.23 ^H	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	45.9	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	73.7 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by calculated by automated hematology analyzer	23.3 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by calculated by automated hematology analyzer	31.6 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	16.1 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	44.6	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	11.83	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by Calculated	19.07	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	8110	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER &	NIL	%	< 10 %

DIFFERENTIAL LEUCOCYTE COUNT (DLC)



MICROSCOPY

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Test Name	Value	Unit	Biological Reference interval
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	54	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	35	%	20 - 40
EOSINOPHILS by flow cytometry by sf cube & microscopy	6	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LEUKOCYTES (WBC) COUNT	0	%	0 - 1
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4379	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2838	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	487 ^H	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	406	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKE	0 RS.	/cmm	0 - 110
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	332000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.43 ^H	%	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	154000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	46.3 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.2	%	15.0 - 17.0



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

Test Name Value Unit Biological Reference interval

GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): 11.5^H % 4.0 - 6.4

WHOLE BLOOD

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE 283.35^H mg/dL 60.00 - 140.00

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

INTERPRETATION:

AS PER AMERICAN DIAI	BETES ASSOCIATION (ADA):		
REFERENCE GROUP	GLYCOSYLATED HEMOGL	OGIB (HBAIC) in %	
Non diabetic Adults >= 18 years	<5.7		
At Risk (Prediabetes)	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-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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

Value Unit **Biological Reference interval** Test Name

ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR) by MODIFIED WESTERGREN AUTOMATED METHOD 32H

mm/1st hr

0 - 20

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.

- NOTE:

- 1. ESR and C reactive protein (C-RP) are both markers of inflammation.
 2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
 3. CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.
 4. 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 services and quiping may decrease it. aspirin, cortisone, and quinine may decrease it

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Test Name Value Unit **Biological Reference interval**

CLINICAL CHEMISTRY/BIOCHEMISTRY **GLUCOSE FASTING (F)**

GLUCOSE FASTING (F): PLASMA 260.33H 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**

GLUCOSE POST PRANDIAL (PP)

GLUCOSE POST PRANDIAL (PP): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) 318.57H NORMAL: < 140.00

PREDIABETIC: 140.0 - 200.0 DIABETIC: > 0R = 200.0

INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A post-prandial plasma glucose level below 140 mg/dl is considered normal.

2. A post-prandial glucose level between 140 - 200 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 post-prandial plasma glucose level of 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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Test Name	Value	Unit	Biological Reference interval
	LIPID PROFILE :	BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	258.38 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	284.08 ^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	39.64	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	161.92 ^H	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	218.74 ^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	56.82 ^H	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	800.84 ^H	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	6.52 ^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: SERUM by CALCULATED, SPECTROPHOTOMETRY	4.08 ^H	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0



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Test Name Value Unit **Biological Reference interval**

TRIGLYCERIDES/HDL RATIO: SERUM 7.17^H **RATIO** 3.00 - 5.00by CALCULATED, SPECTROPHOTOMETRY

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	0.29	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.12	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.17	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	35.05	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	43.61	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.8	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para Nitrophenyl Phosphatase by Amino Meth' PROPANOL	143 YL	U/L	40.0 - 150.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	127 ^H	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.9	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.6	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.3	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.39	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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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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mg/dL

mg/dL

mmol/L

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9.4

3.33

142.1

4.11

106.57

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Test Name	Value	Unit	Biological Reference interval
К	IDNEY FUNCTION TI	EST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	14.76	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	0.62	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	6.9 ^L	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	11.13	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	23.81	RATIO	
URIC ACID: SERUM	5.3	mg/dL	3.60 - 7.70

PHOSPHOROUS: SERUM
by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY

ELECTROLYTES

SODIUM: SERUM
by ISE (ION SELECTIVE ELECTRODE)
POTASSIUM: SERUM

by ISE (ION SELECTIVE ELECTRODE)
CHLORIDE: SERUM

by ISE (ION SELECTIVE ELECTRODE)

by URICASE - OXIDASE PEROXIDASE

by ARSENAZO III, SPECTROPHOTOMETRY

ESTIMATED GLOMERULAR FILTERATION RATE

ESTIMATED GLOMERULAR FILTERATION RATE 121.6

(eGFR): SERUM
by CALCULATED

CALCIUM: SERUM

<u>INTERPRETATION:</u>

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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8.50 - 10.60

2.30 - 4.70

135.0 - 150.0

3.50 - 5.00

90.0 - 110.0

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KOS Diagnostic Lab (A Unit of KOS Healthcare)



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NAME : Mr. KIRAN PARKASH

AGE/ GENDER : 43 YRS/MALE **PATIENT ID** : 1553883

COLLECTED BY :012407190015 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 19/Jul/2024 08:19 AM BARCODE NO. :01513408 **COLLECTION DATE** : 19/Jul/2024 08:26AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 19/Jul/2024 10:47AM

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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:

THE CLOWLE CONTROL OF THE TENTH						
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

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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 : 21/Jul/2024 10:54AM

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

Test Name Value Unit Biological Reference interval

CREATININE PHOSPHOKINASE (CPK-NAC) TOTAL

CREATININE PHOSPHO KINASE (CPK-NAC)

by SPECTROPHOTOMETERY

36.22 IU/L 24 - 19

Interpretation:-

- 1. Serum creatine kinase (CK) activity is greatly elevated, at some time during the course of the disease, in all types of muscular dystrophy, and especially so in Duchenne type, in which levels up to 50 times the upper limit of normal may be encountered.
- 2.In progressive muscular dystrophy, enzyme activity in serum is highest in infancy and childhood (7-10 years of age) and may be elevated long before the disease is clinically apparent.
- 3. Quite high values of CK are noted in viral myositis, polymyositis, and similar muscle diseases.
- 4. However, in neurogenic Parkinsonism, serum enzyme activity is normal. Very high activity is also encountered in malignant hyperthermia.

Significance:-

- 1.An early rise in CK is also seen after an acute MI, with values peaking at 12 to 24 hours and falling back to normal in 3 to 4 days.
- 2.Although total CK activity has been used as a diagnostic test for MI, it has been replaced by the troponin T and I immunoassays, and is no longer the laboratory test choice for diagnosing and monitoring acute infarctions.
- 3.Serum CK activity may increase in patients with acute cerebrovascular disease or neurosurgical intervention and with cerebral ischemia.
- 4.Serum CK activity also demonstrates an inverse relationship with thyroid activity. About 60% of hypothyroid subjects show an average elevation of CK activity 5-fold over the upper reference limit; elevation of as high as 50-fold may also be found.

Note: Exercise and muscle trauma (contact sports, traffic accidents, intramuscular injections, surgery, convulsions, wasp or bee stings, and burns) can elevate serum creatine kinase values.



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

Test Name Value Unit **Biological Reference interval**

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM 1.012 ng/mL 0.35 - 1.93by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROXINE (T4): SERUM 8.52

4.87 - 12.60 μgm/dL

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROID STIMULATING HORMONE (TSH): SERUM 1.119 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	Т3	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

- 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
- 3. Serum T4 levies 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.

TRIIODOTHY	RONINE (T3)	THYROXINE (T4)		THYROID STIMULATING HORMON	
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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: KOS DIAGNOSTIC LAB **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name			Value	Unit		Biologi	cal 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		
	RECOI	MMENDATIONS OF TSH LI	EVELS DURING PRE	GNANCY (µIU/mL)			
	1st Trimester			0.10 - 2.50			
	2nd Trimester			0.20 - 3.00			
	3rd Trimester			0.30 - 4.10			

REPORTING DATE

INCREASED TSH LEVELS:

CLIENT CODE.

- 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



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 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 21/Jul/2024 09:25AM

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

Test Name Value Unit Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY GLUTAMIC ACID DECARBOXYLASE (GAD)-65 ANTIBODY

GLUTAMIC ACID DECARBOXYLASE
(GAD)-65 ANTIBODY IgG: SERUM

0.31 U/mL

NEGATIVE: < 1.00

POSITIVE: > 1.00

by ELISA (ENZYME LINKED IMMUNOASSAY)

<u> INTERPRETATION:</u>

Glutamic acid decarboxylase (GAD)-65 autoantibodies are detected in most newly diagnosed Type 1A Diabetes patients and in 80 % of prediabetic first degree relative of patients. Anti GAD are directed primarily at the GAD isoform which is found primarily in pancreatic islet cells and in the central nervous system. Presence of GAD autoantibodies is also associated with stiff man syndrome.

USES:

- 1.To diagnose Insulin Dependant Diabetes Mellitus (IDDM) and differentiate from Type 2 Diabetes (NDDM).
- 2.To assess risk and predict onset of development of IDDM specially in first degree relatives.
- 3.To assess risk of development of related endocrine disorders.



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

Test Name Value Unit **Biological Reference interval**

INSULIN ANTIBODIES/ANTI INSULIN ANTIBODIES

INSULIN ANTIBODIES: SERUM 0.14 U/mL NEGATIVE: < 0.9 by EIA (ENZYME IMMUNOASSAY) POSITIVE: > 1.0

INTERPRETATION:

NOTE:

1. This is only a screening test for the diagnosis of IDDM (Type I Diabetes mellitus). It should be correlated with clinical history and results of

2. This is a quantitative measurement of IgG class antibodies against Insulin

3. Positive results indicate the likelihood of development of future type 1 diabetes.

CLINICAL UTILITY:

1. Early diagnosis of patients with autoimmune diabetes type 1 during asymptomatic phase.
2. Predicting the future development of Type 1 Diabetes in asymptomatic children, adolescents and adults when used in conjunction with family

2. Predicting the tube development of Type 1 bladetes in asymptomatic children, addressents and address when used in conjunction with fair history, HLA typing & other autoantibodies.

3. To identify reason for no response to insulin therapy

4. To identify allergic response insulin

5. Differential diagnosis of Type 1 versus Type 2 Diabetes

6. Investigation of hypoglycemia in non-diabetic subjects

7. Presence of multiple autoantibodies in an individual i.e., GAD-65, ICA, IA-2, IAA, ZnT8 has a very high predictive value for type 1 diabetes. **ASSOCIATED TESTS:**

1. Islet cell antibody (I0287), GAD antibody (G0004), IA2 insulin autoantibody (I0001), Zinc Transporter 8 (ZnT8) Antibody (Z0015)



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

Test Name Value Unit **Biological Reference interval**

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE 8.52 NEGATIVE: < 20.0 ANTIBODY IgA POSITIVE: > 20.0

by ELISA (ENZYME LINKED IMMUNOASSAY)

INTERPRETATION:

1. Anti-transglutaminase antibodies (ATA) are autoantibodies against the transglutaminase protein.

2. Antibodies to tissue transglutaminas are found in patients with several conditions, including coeliac disease, juvenile diabetes, inflammatory bowel disease, and various forms of arthritis.

3.In coeliac disease, ATA are involved in the destruction of the villous extracellular matrix and target the destruction of intestinal villous epithelial cells by killer cells.

4. Deposits of anti-tTG in the intestinal epithelium predict coeliac disease.

5.Celiac disease (gluten-sensitive enteropathy, celiac sprue) results from an immune-mediated inflammatory process following ingestion of wheat, rye, or barley proteins that occurs in genetically susceptible individuals. The inflammation in celiac disease occurs primarily in the mucosa of the small intestine, which leads to villous atrophy.

CLINICAL MANIFESTATIONS RELATED TO GASTROINTESTINAL TRACT:

- 1. Abdominal pain
- 2.Malabsorption
- 3. Diarrhea and Constipation.

CLINICAL MANIFESTATION OF CELIAC DISEASE NOT RESTRICTED TO GIT:

- 1. Failure to grow (delayed puberty and short stature)
- 2.Iron deficiency anemia
- 3. Recurrent fetal loss
- 4. Osteoporosis and chronic fatigue
- 5. Recurrent aphthous stomatitis (canker sores)
- 6.Dental enamel hypoplasia, and dermatitis herpetiformis.
- 7. Patients with celiac disease may also present with neuropsychiatric manifestations including ataxia and peripheral neuropathy, and are at increased risk for development of non-Hodgkin lymphoma.
- 8. The disease is also associated with other clinical disorders including thyroiditis, type I diabetes mellitus, Down syndrome, and IgA deficiency.

NOTE:

- 1.The finding of tissue transglutaminase (tTG)-IgA antibodies is specific for celiac disease and possibly for dermatitis herpetiformis. For individuals with moderately to strongly positive results, a diagnosis of celiac disease is likely and the patient should undergo biopsy to confirm the diagnosis
- 2.If patients strictly adhere to a gluten-free diet, the unit value of IgA-anti-tTG should begin to decrease within 6 to 12 months of onset of dietary therapy

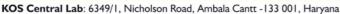
CAUTIÓN:

- 1. This test should not be solely relied upon to establish a diagnosis of celiac disease. It should be used to identify patients who have an increased probability of having celiac disease and in whom a small intestinal biopsy is recommended.
- 2. Affected individuals who have been on a gluten-free diet prior to testing may have a negative result.



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Test Name Value Unit Biological Reference interval

3.For individuals who test negative, IgA deficiency should be considered. If total IgA is normal and tissue transglutaminase (tTG)-IgA is negative there is a low probability of the patient having celiac disease and a biopsy may not be necessary.

4.If serology is negative or there is substantial clinical doubt remaining, then further investigation should be performed with endoscopy and bowel biopsy. This is especially important in patients with frank malabsorptive symptoms since many syndromes can mimic celiac disease. For the patient with frank malabsorptive symptoms, bowel biopsy should be performed regardless of serologic test results.

5.The antibody pattern in dermatitis herpetiformis may be more variable than in celiac disease; therefore, both endomysial and tTG antibody determinations are recommended to maximize the sensitivity of the serologic tests.

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

Test Name Value Unit **Biological Reference interval**

C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: 2.69 0.0 - 6.0mg/L

SERUM

by NEPHLOMETRY

INTERPRETATION:

C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.

2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant

rejection, and to monitor these inflammatory processes.

4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,

5. Elevated values are consistent with an acute inflammatory process.

NOTE:

1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
2. Oral contraceptives may increase CRP levels.

*** End Of Report



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