

(A Unit of KOS Healthcare)



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

NAME : Mrs. NIKITA

**AGE/ GENDER** : 33 YRS/FEMALE **PATIENT ID** : 1757731

COLLECTED BY : REG. NO./LAB NO. : 042502150001

 REFERRED BY
 : 15/Feb/2025 09:41 AM

 BARCODE NO.
 : A1260503
 COLLECTION DATE
 : 15/Feb/2025 04:19PM

 CLIENT CODE.
 : KOS DIAGNOSTIC SHAHBAD
 REPORTING DATE
 : 15/Feb/2025 05:03PM

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

Test Name Value Unit Biological Reference interval

### SWASTHYA WELLNESS PANEL: 1.2 COMPLETE BLOOD COUNT (CBC)

### **RED BLOOD CELLS (RBCS) COUNT AND INDICES**

PACKED CELL VOLU  by CALCULATED BY A  MEAN CORPUSCUL  by CALCULATED BY A  MEAN CORPUSCUL  by CALCULATED BY A  MEAN CORPUSCUL  by CALCULATED BY A	B)	9.7 <sup>L</sup>	am /dI	100 100
RED BLOOD CELL ( by HYDRO DYNAMIC F PACKED CELL VOLU by CALCULATED BY A MEAN CORPUSCUL by CALCULATED BY A MEAN CORPUSCUL by CALCULATED BY A MEAN CORPUSCUL by CALCULATED BY A	,	9.72	gm/dL	12.0 - 16.0
PACKED CELL VOLU  by CALCULATED BY A  MEAN CORPUSCUL  by CALCULATED BY A  MEAN CORPUSCUL  by CALCULATED BY A  MEAN CORPUSCUL  by CALCULATED BY A	RBC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	4.33	Millions/cmm	3.50 - 5.00
by CALCULATED BY A MEAN CORPUSCUL by CALCULATED BY A MEAN CORPUSCUL by CALCULATED BY A		32 <sup>L</sup>	%	37.0 - 50.0
by CALCULATED BY A MEAN CORPUSCUL by CALCULATED BY A	AR VOLUME (MCV) AUTOMATED HEMATOLOGY ANALYZER	73.8 <sup>L</sup>	fL	80.0 - 100.0
by CALCULATED BY A	AR HAEMOGLOBIN (MCH) AUTOMATED HEMATOLOGY ANALYZER	22.4 <sup>L</sup>	pg	27.0 - 34.0
	AR HEMOGLOBIN CONC. (MCHC) AUTOMATED HEMATOLOGY ANALYZER	30.4 <sup>L</sup>	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV) AUTOMATED HEMATOLOGY ANALYZER	17.5 <sup>H</sup>	%	11.00 - 16.00
	UTION WIDTH (RDW-SD) AUTOMATED HEMATOLOGY ANALYZER	48.5	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		17.04	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING IND by CALCULATED	DEX	29.82	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE	LLS (WBCS)			
TOTAL LEUCOCYTE	E COUNT (TLC) y by sf cube & microscopy	5250	/cmm	4000 - 11000
	BLOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
NUCLEATED RED B	BLOOD CELLS (nRBCS) %	NIL	%	< 10 %



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by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER



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DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	58	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES	31	%	20 - 40
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	31	70	20 - 40
EOSINOPHILS	6 <sup>H</sup>	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	_	0/	0 10
MONOCYTES by Flow cytometry by SF cube & microscopy	5	%	2 - 12
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	3045	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY  ABSOLUTE LYMPHOCYTE COUNT	1628	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1020	/ CIIIII	000 - 4000
ABSOLUTE EOSINOPHIL COUNT	315	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT	262	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	202	/ CIIIII	80 - 880
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			0.0.000.0
ABSOLUTE IMMATURE GRANULOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0.0 - 999.0
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT)	257000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.39 <sup>H</sup>	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV)	15 <sup>H</sup>	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	13	111	0.00 12.0
PLATELET LARGE CELL COUNT (P-LCC)	$165000^{\mathrm{H}}$	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR)	64 <sup>H</sup>	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	64**	/0	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW)	15.9	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			



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NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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**Value** Unit **Biological Reference interval Test Name** 

### **ERYTHROCYTE SEDIMENTATION RATE (ESR)**

ERYTHROCYTE SEDIMENTATION RATE (ESR)

mm/1st hr 62<sup>H</sup>

REPORTING DATE

by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

#### INTERPRETATION:

CLIENT CODE.

- 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:
- ESR and C reactive protein (C-RP) are both markers of inflammation.
   Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
   CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.
   If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
   Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
   Progs such as doubtern mathyldona, oral contracentives, popicillamino procesingmide, the only viling, and vitality in the orange of the

- 6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it



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

**Value** Unit **Biological Reference interval Test Name** 

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

GLUCOSE FASTING (F): PLASMA 94.91 NORMAL: < 100.0 mg/dL

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

INTERPRETATION
IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.

2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood

test (after consumption of 75 gms of glucose) is recommended for all such patients.

3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.



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Test Name	Value	Unit	Biological Reference interval
	LIPID PROFILE	: BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	162.29	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	75.25	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	53.54	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	93.7	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	108.75	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	15.05	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	399.83	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.03	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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LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.75	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED. SPECTROPHOTOMETRY	1.41 <sup>L</sup>	RATIO	3.00 - 5.00

#### **INTERPRETATION:**

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

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

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

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

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

5. Additional testing for Apolipoprotein B, hsCRP,Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement



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### **LIVER FUNCTION TEST (COMPLETE)**

BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.47	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	0.35	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	19.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	14.8	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM  by CALCULATED, SPECTROPHOTOMETRY	1.33	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	118.4	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	12.99	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.48	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.1	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.38	gm/dL	2.30 - 3.50
A: GRATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.21	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:**

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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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KIDN	EY FUNCTION TE	EST (COMPLETE)	
UREA: SERUM by urease - glutamate dehydrogenase (gldh)	23.84	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	0.91	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	11.14	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	12.24	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	26.2	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	3.23	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY	9.27	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY	2.9	mg/dL	2.30 - 4.70
ELECTROLYTES			
SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	145.3	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.23	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	108.98	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RATE			

ESTIMATED GLOMERULAR FILTERATION RATE 85.4

(eGFR): SERUM 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.
- 3. GI haemorrhage.



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**NAME** : Mrs. NIKITA

AGE/ GENDER : 33 YRS/FEMALE **PATIENT ID** : 1757731

**COLLECTED BY** :042502150001 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 15/Feb/2025 09:41 AM BARCODE NO. **COLLECTION DATE** : 15/Feb/2025 04:20PM : A1260502 CLIENT CODE. : KOS DIAGNOSTIC SHAHBAD REPORTING DATE : 15/Feb/2025 06:00PM

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

**Test Name** Value Unit **Biological Reference interval** 

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**:

EGITIVISTICE GEGINIERGESTICT	THE SECONDICTION OF THE PERSON						
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 creating 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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### **ENDOCRINOLOGY**

### THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM 1.021 ng/mL 0.35 - 1.93

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROXINE (T4): SERUM 8.07  $\mu$ gm/dL 4.87 - 12.60

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROID STIMULATING HORMONE (TSH): SERUM 2.161  $\mu$ IU/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 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.

TRIIODOTH	RONINE (T3)	THYROXINE (T4)		THYROID STIMULATING HORMONE (TS	
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range ( μΙU/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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: KOS DIAGNOSTIC SHAHBAD

Test Name		Value Unit	Biological Reference inter			
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50	
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20	11 – 19 Years	0.50 - 5.50	
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35- 5.50	
	RECOM	MENDATIONS OF TSH 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, 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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0.5

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: KOS DIAGNOSTIC SHAHBAD

Test Name Value Unit Biological Reference interval

# IMMUNOPATHOLOGY/SEROLOGY ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA

by ELISA (ENZYME LINKED IMMUNOASSAY)

IU/mL

REPORTING DATE

NEGATIVE: < 20.0

POSITIVE: > 20.0

: 16/Feb/2025 02:45AM

### **INTERPRETATION:**

CLIENT CODE.

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 fatique
- 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.



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Test Name Value Unit Biological Reference interval

2.Affected individuals who have been on a gluten-free diet prior to testing may have a negative result.

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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Test Name Value Unit Biological Reference interval

### VITAMINS VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM 508 pg/mL 190.0 - 890.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

#### INTERPRETATION:-

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

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

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



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# CLINICAL PATHOLOGY URINE ROUTINE & MICROSCOPIC EXAMINATION

### **PHYSICAL EXAMINATION**

QUANTITY RECIEVED 10 ml

COLOUR AMBER YELLOW PALE YELLOW

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

TRANSPARANCY HAZY CLEAR

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SPECIFIC GRAVITY 1.01 1.002 - 1.030

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

**CHEMICAL EXAMINATION** 

REACTION ACIDIC by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

PROTEIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

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

pH 5.0 - 7.5

BILIRUBIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NITRITE Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.

UROBILINOGEN Normal EU/dL 0.2 - 1.0 by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

KETONE BODIES Negative NEGATIVE (-ve)

BLOOD TRACE NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

ASCORBIC ACID

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NEGATIVE (-ve)

NEGATIVE (-ve)

MICROSCOPIC EXAMINATION

RED BLOOD CELLS (RBCs) 1-3 /HPF 0 - 3



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Test Name	Value	Unit	Biological Reference interval
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	5-6	/HPF	ABSENT
CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT		ABSENT

\*\*\* End Of Report \*\*\*



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