

(A Unit of KOS Healthcare)



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

NAME : Mr. BHUSHAN LAL GUPTA

AGE/ GENDER : 64 YRS/MALE PATIENT ID : 1825198

COLLECTED BY : REG. NO./LAB NO. : 012504100047

 REFERRED BY
 : 10/Apr/2025 10:57 AM

 BARCODE NO.
 : 01528744
 COLLECTION DATE
 : 10/Apr/2025 11:46AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 10/Apr/2025 12:22PM

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

Test Name Value Unit Biological Reference interval

SWASTHYA WELLNESS PANEL: DT COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	10.7^{L}	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.07	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	33.8 ^L	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by Calculated by automated hematology analyzer	83.2	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by Calculated by automated hematology analyzer	26.3 ^L	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	15.6	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	48.5	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	20.44	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	100.92	RATIO	BETA THALASSEMIA TRAIT: <= 74.1 IRON DEFICIENCY ANEMIA: >= 74.1
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) by Flow cytometry by sf cube & microscopy	11250 ^H	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by automated 6 part hematology analyzer	NIL		0.00 - 20.00



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NIL



< 10 %

NUCLEATED RED BLOOD CELLS (nRBCS) %



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Test Name	Value	Unit	Biological Reference interval
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER			
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	67	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
LYMPHOCYTES	27	%	20 - 40
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
EOSINOPHILS	2	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY		0.4	2 12
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	2 - 12
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	U	%0	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	U	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	7538 ^H	/CIIIII	2000 - 7300
ABSOLUTE LYMPHOCYTE COUNT	3038	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3030	, CI IIII	1700
ABSOLUTE EOSINOPHIL COUNT	225	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE MONOCYTE COUNT	450	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
PLATELETS AND OTHER PLATELET PREDICTIV	<u>E MARKERS.</u>		
PLATELET COUNT (PLT)	206000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT)	0.28	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	14 ^H	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE		,	20000 00000
PLATELET LARGE CELL COUNT (P-LCC)	103000^{H}	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR)	u au	0/	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	49.8 ^H	%	11.0 - 43.0
PLATELET DISTRIBUTION WIDTH (PDW)	16.2	%	15.0 - 17.0
Latitude Hotel (LDW)	10.2	/0	13.0 17.0



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Test Name Value Unit Biological Reference interval

by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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Value Unit Test Name **Biological Reference interval**

ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)

59H

mm/1st hr

0 - 20

by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

INTERPRETATION:

- 1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and autoimmune 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.

- 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 aspirin, cortisone, and quinine may decrease it



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Value Unit Test Name **Biological Reference interval**

CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA 106.92^{H} 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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	LIPID PROFILE	E: BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	119.25	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	203.63 ^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	50.17	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	28.35	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	69.08	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	40.73	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	442.13	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.38	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0



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Test Name	Value	Unit	Biological Reference interval
			MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.57	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	4.06	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

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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Test Name Value Unit Biological Reference interval

LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.65	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.25	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.4	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	23.76	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	21.14	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.12	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	58	U/L	40.0 - 150.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	17	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.44	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.3	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.14	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.37	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



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Test Name	Value	Unit	Biological Reference interval
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS		> 1.3 (Slightly Increased)	
DECDEASED.			

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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KIDNEY FUNCTION TEST (COMPLETE)

UREA: SERUM	31.03	mg/dL	10.00 - 50.00
by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)		8	
CREATININE: SERUM	1.01	mg/dL	0.40 - 1.40
by ENZYMATIC, SPECTROPHOTOMETERY			
BLOOD UREA NITROGEN (BUN): SERUM	14.5	mg/dL	7.0 - 25.0
by CALCULATED, SPECTROPHOTOMETRY	11.24	D. A. TOTA	10.0.20.0
BLOOD UREA NITROGEN (BUN)/CREATININE	14.36	RATIO	10.0 - 20.0
RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY			
UREA/CREATININE RATIO: SERUM	30.72	RATIO	
by CALCULATED, SPECTROPHOTOMETRY	30.72	MIIIO	
URIC ACID: SERUM	6.3	mg/dL	3.60 - 7.70
by URICASE - OXIDASE PEROXIDASE		· ·	
CALCIUM: SERUM	9.01	mg/dL	8.50 - 10.60
by ARSENAZO III, SPECTROPHOTOMETRY			
PHOSPHOROUS: SERUM	3.8	mg/dL	2.30 - 4.70
by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY			
ELECTROLYTES			
SODIUM: SERUM	138.4	mmol/L	135.0 - 150.0
by ISE (ION SELECTIVE ELECTRODE)	4.10	1.77	2.50 5.00
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.12	mmol/L	3.50 - 5.00
CHLORIDE: SERUM	103.8	mmol/L	90.0 - 110.0
by ISE (ION SELECTIVE ELECTRODE)	103.0	IIIIIOI/L	90.0 - 110.0

ESTIMATED GLOMERULAR FILTERATION RATE

ESTIMATED GLOMERULAR FILTERATION RATE 83

(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.



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REFERRED BY **REGISTRATION DATE** : 10/Apr/2025 10:57 AM BARCODE NO. :01528744 **COLLECTION DATE** : 10/Apr/2025 11:46AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 10/Apr/2025 01:40PM

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

Test Name Value Unit **Biological Reference interval**

- 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:

ESTIMATED GLOWENGEAR TIETERATION 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	
	Hormai or High GFR		Albumin of cast in unite	
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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(A Unit of KOS Healthcare)



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

NAME : Mr. BHUSHAN LAL GUPTA

AGE/ GENDER : 64 YRS/MALE **PATIENT ID** : 1825198

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

Value Unit Test Name **Biological Reference interval**

REPORTING DATE

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM 0.882 ng/mL 0.35 - 1.93by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROXINE (T4): SERUM 4.87 - 12.606.57 μgm/dL

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROID STIMULATING HORMONE (TSH): SERUM μIU/mL 0.35 - 5.501.408

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

CLIENT CODE.

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	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 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 (μΙυ/mL)
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 – 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 – 17.04	3 Days – 6 Months	0.70 - 8.40



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Test Name			Value	Unit	t	Biological Reference interval
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00	
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50	
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20	11 – 19 Years	0.50 - 5.50	
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35- 5.50	
	RECOM	IMENDATIONS OF TSH L	EVELS DURING PRE	GNANCY (µIU/mL)	•	
1st Trimester				0.10 - 2.50		
2nd Trimester				0.20 - 3.00		
3rd Trimester				0.30 - 4.10		

INCREASED TSH LEVELS:

- 1. Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.
- 2. Hypothyroid patients receiving insufficient thyroid replacement therapy.
- 3. Hashimotos thyroiditis
- 4.DRUGS: Amphetamines, 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.
- $2. Over \ replacement \ of \ thyroid \ hormone \ in \ treatment \ of \ hypothyroid ism.$
- 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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Value Unit Test Name **Biological Reference interval**

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

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

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0

TOXICITY: > 100.0

INTERPRETATION:

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

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

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

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

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

DECREASED:

- 1.Lack of sunshine exposure.
- 2.Inadequate intake, malabsorption (celiac disease)
 3.Depressed Hepatic Vitamin D 25- hydroxylase activity
- 4. Secondary to advanced Liver disease
- 5. Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- 6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

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

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

*** End Of Report ***



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