

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



		Chopra y & Microbiology) Consultant Pathologis	M	n Chopra D (Pathology) nt Pathologist
NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Mr. ABHISHEK : 27 YRS/MALE : : : 01528767 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROA	D, AMBALA CANTI	PATIENT ID REG. NO./LAB NO. REGISTRATION DATE COLLECTION DATE REPORTING DATE	: 1826176 : 012504100070 : 10/Apr/2025 06:37 PM : 10/Apr/2025 06:39PM : 10/Apr/2025 07:34PM
Test Name		Value	Unit	Biological Reference interval
RED BLOOD CELL	(S (RBCS) COUNT AND II		OOD COUNT (CBC)	
HAEMOGLOBIN (HE		14.6	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (by HYDRO DYNAMIC FC	RBC) COUNT cusing, electrical impeden	6.18 ^H	Millior	as/cmm 3.50 - 5.00
PACKED CELL VOL		45.2	%	40.0 - 54.0
MEAN CORPUSCUL by CALCULATED BY AU	AR VOLUME (MCV) TOMATED HEMATOLOGY ANAL	YZER 73.2 ^L	fL	80.0 - 100.0
MEAN CORPUSCUL	AR HAEMOGLOBIN (MCH TOMATED HEMATOLOGY ANAL	^I) 23.5 ^L	pg	27.0 - 34.0
MEAN CORPUSCUL	AR HEMOGLOBIN CONC	. (MCHC) 32.2	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV		%	11.00 - 16.00
RED CELL DISTRIB	UTION WIDTH (RDW-SD TOMATED HEMATOLOGY ANAL) 49.7	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		11.84	RATIC	D BETA THALASSEMIA TRAIT: - 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INE by CALCULATED		66.06	RATIC	D BETA THALASSEMIA TRAIT: <= 74.1 IRON DEFICIENCY ANEMIA: >= 74.1
WHITE BLOOD CE				1000 11000
TOTAL LEUCOCYT	E COUNT (TLC) by sf cube & microscopy	10110	/cmm	4000 - 11000
	LOOD CELLS (nRBCS)	NIL		0.00 - 20.00
NUCLEATED RED B	HEMATOLOGYANALYZER			





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	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. ABHISHEK			
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Test Name		Value	Unit	Biological Reference interval
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
DIFFERENTIAL LE	EUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY	BY SF CUBE & MICROSCOPY	68	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY	BY SF CUBE & MICROSCOPY	23	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY	BY SF CUBE & MICROSCOPY	3	%	1 - 6
MONOCYTES by FLOW CYTOMETRY	BY SF CUBE & MICROSCOPY	6	%	2 - 12
	BY SF CUBE & MICROSCOPY	0	%	0 - 1
	OCYTES (WBC) COUNT			
ABSOLUTE NEUTR by FLOW CYTOMETRY	OPHIL COUNT BY SF CUBE & MICROSCOPY	6875	/cmm	2000 - 7500
ABSOLUTE LYMPH by FLOW CYTOMETRY	OCYTE COUNT BY SF CUBE & MICROSCOPY	2325	/cmm	800 - 4900
ABSOLUTE EOSING	DPHIL COUNT " BY SF CUBE & MICROSCOPY	303	/cmm	40 - 440
ABSOLUTE MONO by FLOW CYTOMETRY	CYTE COUNT by SF cube & microscopy	607	/cmm	80 - 880
ABSOLUTE BASOP	HIL COUNT BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND (OTHER PLATELET PREDICTIV	<u>E MARKERS.</u>		
PLATELET COUNT	' (PLT) OCUSING, ELECTRICAL IMPEDENCE	338000	/cmm	150000 - 450000
PLATELETCRIT (P	CT) OCUSING, ELECTRICAL IMPEDENCE	0.3	%	0.10 - 0.36
MEAN PLATELET V		9	fL	6.50 - 12.0
PLATELET LARGE	CELL COUNT (P-LCC) OCUSING, ELECTRICAL IMPEDENCE	68000	/cmm	30000 - 90000
PLATELET LARGE	CELL RATIO (P-LCR) OCUSING, ELECTRICAL IMPEDENCE	20.3	%	11.0 - 45.0
•	BUTION WIDTH (PDW)	15.8	%	15.0 - 17.0



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



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Test Name		Value	Unit	Biological Reference interval
	GLYC	OSYLATED HAEMO	GLOBIN (HBA1C	<u>C)</u>
WHOLE BLOOD	EMOGLOBIN (HbA1c):	4.5	%	4.0 - 6.4
ESTIMATED AVERAG	GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY)	82.45	mg/dL	60.00 - 140.00
	AS PER AMERICAN DIAB	ETES ASSOCIATION (ADA):		
RE	FERENCE GROUP		EMOGLOGIB (HBAIC) i	n %
	etic Adults >= 18 years		<5.7	
	Risk (Prediabetes)	/	5.7 – 6.4	
Dia	gnosing Diabetes		>= 6.5	
			e > 19 Years	
TIME		Goals of Therapy:	< 7.0	-
Therapeutic	goals for glycemic control	Actions Suggested:	>8.0)
			e < 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

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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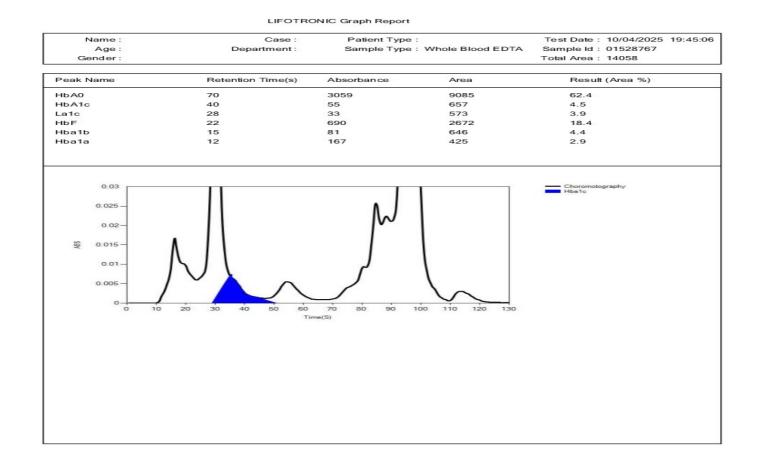
Page 4 of 21







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	Dr. Vinay Ch	opra	Dr. Yugam	n Chopra







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Test Name		Value	Unit	Biological Reference interval
	ERYTHROC	YTE SEDIM	IENTATION RATE	(ESR)
	EDIMENTATION RATE (ESR) gation by capillary photometry	37 ^H	mm/1st h	r 0 - 20
(polycythaemia), sigr as sickle cells in sick NOTE: 1. ESR and C - reactiv 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dexi	le cell anaemia) also lower the ESR. e protein (C-RP) are both markers of es not change as rapidly as does CRP I by as many other factors as is ESR, r ed, it is typically a result of two type we a higher ESR, and menstruation a	t (leucocytosis) inflammation. , either at the s naking it a bette ss of proteins, g nd pregnancy c es, penicillamine	, and some protein abnor tart of inflammation or as r marker of inflammation lobulins or fibrinogen. an cause temporary eleva	rmalities. Some changes in red cell shape (such it resolves.





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		Value	Unit	Biological Reference interval
Test Name				
Test Name	CLINI	CAL CHEMISTR	Y/BIOCHEMIS	STRY
Test Name	CLINI	CAL CHEMISTR GLUCOSE FA		STRY

KOS Diagnostic Lab (A Unit of KOS Healthcare)

A fasting plasma glucose level below 100 mg/dl is considered normal.
 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.
 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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Page 7 of 21





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Test Name		Value	Unit	Biological Reference interval
		LIPID PRO	OFILE : BASIC	
CHOLESTEROL TO by CHOLESTEROL OX		211.11 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: 5 by GLYCEROL PHOSP	SERUM HATE OXIDASE (ENZYMATIC)	203.52 ^H	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERC	DL (DIRECT): SERUM ion	45.9	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERO		124.51	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 CHOLES by CALCULATED, SPE		165.21 ^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 CHOLESTER		40.7	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEI by CALCULATED, SPE	RUM	625.74	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPE	DL RATIO: SERUM	4.6 ^H	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0
	AL .		Alapha	

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Page 8 of 21

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Test Name		Value	Unit	Biological Reference interval
				MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: S by CALCULATED, SPE		2.71	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	IDL RATIO: SERUM	4.43	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.

 Cow HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
 NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL

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





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		Value	Unit	Biological Reference interval
	LIVE	R FUNCTION T	EST (COMPLETE	
SFRUM		0.47	mg/dI	INFANT: 0.20 - 8.00

		,	
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	0.15	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.32	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	38.2	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	72.2 ^H	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.53	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para Nitrophenyl phosphatase by amino methyl propanol	59.49	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	38.9	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.89	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.42	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.47	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.27	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.

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MD (Pathology & Microbiology)

INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5





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Page 10 of 2

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HEPATOCELLULAR C	ARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Ir	ncreased)

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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		EXCELLENCE IN HEALTHCARE	& DIAGNOSTICS
o pra Microbiology) ultant Pathologist		Dr. Yugam MD CEO & Consultant	(Pathology)
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: 1826176
: 012504100070
: 10/Apr/2025 06:37 PM

: 10/Apr/2025 06:39PM

:10/Apr/2025 09:51PM

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

: KOS DIAGNOSTIC LAB

: Mr. ABHISHEK

: 27 YRS/MALE

:01528767

:

:

Dr. Vinay Cho MD (Pathology & Chairman & Const

Test Name	Value	Unit	Biological Reference interval
KIDNEY	FUNCTION T	EST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	15.59	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	1.16	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	7.29	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	6.28 ^L	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	13.44	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	7.99 ^H	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY	8.57	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY	3.99	mg/dL	2.30 - 4.70
ELECTROLYTES			
SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	143.2	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.15	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	107.4	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RAT	E		
ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM by CALCULATED	88.5		

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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NAME

AGE/ GENDER

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CLIENT CODE.





Chopra ogy & Microbiology) Consultant Pathologi		(Pathology)
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		. 10/ Api/ 2023 09.31FM
OAD, AMBALA CANTI		
Value	Unit	Biological Reference interval
ase. : diffuses out of extra absent in blood). narmone) due to tubi TININE: of creatine to creatin	ular secretion of urea. ine).	
i	o).	e increase in creatinine with certain methodolo b). he measurement).

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:

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.
 eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
 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 of CFD with the commended to measure

3. In patients, with eGFR cleaning between 45-59 minimit 1.73 m2 (G3) and without any marker of Kidney damage, it is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fulfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
7. A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).

ADVICE:

KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated



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Test Name		Value	Unit	Biological Reference interval
		IRON P	ROFILE	
IRON: SERUM by FERROZINE, SPEC	TROPHOTOMETRY	76.13	μg/dL	59.0 - 158.0
UNSATURATED II SERUM by FERROZINE, SPEC	RON BINDING CAPACITY (UIBC)) 204.01	μg/dL	150.0 - 336.0
•	DING CAPACITY (TIBC)	280.14	μg/dL	230 - 430
%TRANSFERRIN S	SATURATION: SERUM	27.18	%	15.0 - 50.0

by CALCULATED, SPECTROPHOTOMETERY (FERENE) 200.0 - 350.0 TRANSFERRIN: SERUM mg/dL 198.9^L by SPECTROPHOTOMETERY (FERENE) **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.

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):
 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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NAME	: Mr. ABHISHEK				
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Test Name		Value	Unit	Biological Reference inte	rval
		ENDOCRINO	DLOGY		
	Т	HYROID FUNCTION	TEST: TOTAL		
TRIIODOTHYRON by CMIA (CHEMILUMIN	INE (T3): SERUM IESCENT MICROPARTICLE IMMUN	0.95 OASSAY)	ng/mL	0.35 - 1.93	
THYROXINE (T4): by CMIA (CHEMILUMIN	SERUM IESCENT MICROPARTICLE IMMUN	9.34 OASSAY)	µgm/dL	4.87 - 12.60	
	ATING HORMONE (TSH): IESCENT MICROPARTICLE IMMUN		µIU/mL	0.35 - 5.50	
3rd GENERATION, ULT <u>INTERPRETATION</u> :	RASENSITIVE				
day has influence on the trilodothyronine (T3).Fai		s. TSH stimulates the productior	and secretion of the metab	ne variation is of the order of 50%.Hence tim olically active hormones, thyroxine (T4)and derproduction (hypothyroidism) or	

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

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 (TSI	
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range (μIU/mL)
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 - 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 - 17.04	3 Days – 6 Months	0.70 - 8.40





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Test Name	Value	Unit	Biological Reference interval

I est Maine			value	Om	6	Diological Reference interv
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	MENDATIONS OF TSH LI	EVELS DURING PREC	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 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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CLIENT ADDRESS	: 6349/1, NICHOLS	ON ROAD, AMBALA CANT	Т	
Test Name		Value	Unit	Biological Reference interv
		VI	TAMINS	
		VITAMIN D/25 H	YDROXY VITAMIN D)3
	DROXY VITAMIN		ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
INTERPRETATION:				
DEFI	CIENT:	< 20	ng	g/mL

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.



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Test Name		Value	Unit	Biological Reference interval
		VITAMIN B12/CO	BALAMIN	
VITAMIN B12/COB by CMIA (CHEMILUMIN INTERPRETATION:-	ALAMIN: SERUM	85 ^L	pg/mL	190.0 - 890.0
	SED VITAMIN B12		DECREASED VITAMIN	N B12
1.Ingestion of Vitan	nin C	1.Pregnancy		
2.Ingestion of Estro			in, Anti-convulsants	, Colchicine
3.Ingestion of Vitan 4.Hepatocellular in		3.Ethanol Igest 4. Contraceptiv		
5.Myeloproliferativ		5.Haemodialy		
6.Uremia	amin) is necessary for hematopo	6. Multiple My	eloma	
3. The body uses its v excreted. 4. Vitamin B12 deficie ileal resection, small 5. Vitamin B12 deficie proprioception, poor	ency may be due to lack of IF secr l intestinal diseases). ency frequently causes macrocyti coordination, and affective beha ts without macrocytic anemia. nic acid and homocysteine levels	ally, reabsorbing vitami etion by gastric mucosa c anemia, glossitis, per avioral changes. These r are also elevated in vit	n B12 from the ileun (eg, gastrectomy, g pheral neuropathy, nanifestations may o amin B12 deficiency dentify this potentia	n and returning it to the liver; very little is astric atrophy) or intestinal malabsorption (eg. weakness, hyperreflexia, ataxia, loss of occur in any combination; many patients have states. al cause of vitamin B12 malabsorption.





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A			1
Test Name		Value	Unit	Biological Reference interv
		CLINICAL PATH	IOLOGY	
		TINE & MICROSC		NATION
PHYSICAL EXAM	IINATION			
QUANTITY RECIE	VED CTANCE SPECTROPHOTOMETRY	10	ml	
COLOUR by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	AMBER YELLOW	Į.	PALE YELLOW
TRANSPARANCY	TANCE SPECTROPHOTOMETRY	CLEAR		CLEAR
SPECIFIC GRAVIT		1.01		1.002 - 1.030
CHEMICAL EXAM				
REACTION	TANCE SPECTROPHOTOMETRY	ACIDIC		
PROTEIN	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
pH by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	6		5.0 - 7.5
BILIRUBIN by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
NITRITE by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0
KETONE BODIES by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
BLOOD by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	CTANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
MICDOCODICE	X7 A X ATXI A (DTOX)			

MICROSCOPIC EXAMINATION



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Test Name		Value	Unit	Biological Reference interval	
RED BLOOD CELLS	(RBCs) ENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3	
PUS CELLS by MICROSCOPY ON CL	ENTRIFUGED URINARY SEDIMENT	2-3	/HPF	0 - 5	
EPITHELIAL CELLS	ENTRIFUGED URINARY SEDIMENT	0-2	/HPF	ABSENT	
CRYSTALS by MICROSCOPY ON CL	ENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
CASTS by MICROSCOPY ON CL	ENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
BACTERIA by MICROSCOPY ON CL	ENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
OTHERS		NEGATIVE (-ve)		NEGATIVE (-ve)	

OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT TRICHOMONAS VAGINALIS (PROTOZOA)

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

*** End Of Report ***

ABSENT





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