



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)	
NAME	: Miss. JAGRITI MANCHANDA				
AGE/ GENDER	: 38 YRS/FEMALE		PATIENT ID	: 1740870	
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	:012501310027	
REFERRED BY	:		REGISTRATION DATE	: 31/Jan/2025 10:16 AM	[
BARCODE NO.	: 01524701		COLLECTION DATE	: 31/Jan/2025 11:02AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 31/Jan/2025 11:21AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CANTI	Г		
Test Name		Value	Unit	Biological Re	ference interval
			ELLNESS PANEL: DI LOOD COUNT (CBC)	ſ	
RED BLOOD CELL	S (RBCS) COUNT AND INDICES				
HAEMOGLOBIN (H	B)	11.7 ^L	gm/dL	12.0 - 16.0	
by CALORIMETRIC RED BLOOD CELL (by HYDRO DYNAMIC F	(RBC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	4.23	Millions	/cmm 3.50 - 5.00	
PACKED CELL VOL		36.2 ^L	%	37.0 - 50.0	
	AR VOLUME (MCV) automated hematology analyzer	85.6	fL	80.0 - 100.0	
	AR HAEMOGLOBIN (MCH) AUTOMATED HEMATOLOGY ANALYZER	27.7	pg	27.0 - 34.0	
	AR HEMOGLOBIN CONC. (MCHC)	32.4	g/dL	32.0 - 36.0	
	UTION WIDTH (RDW-CV) automated hematology analyzer	15.1	%	11.00 - 16.00)
	SUTION WIDTH (RDW-SD) AUTOMATED HEMATOLOGY ANALYZER	48.4	fL	35.0 - 56.0	
MENTZERS INDEX by CALCULATED		20.24	RATIO	13.0	SSEMIA TRAIT: < ENCY ANEMIA:
GREEN & KING INI by CALCULATED WHITE BLOOD CE		30.6	RATIO	65.0	SSEMIA TRAIT:<= ENCY ANEMIA: >
TOTAL LEUCOCYTI		7250	/cmm	4000 - 11000)
NUCLEATED RED H	BLOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00	
NUCLEATED RED H	BLOOD CELLS (nRBCS) %	NIL	%	< 10 %	





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist



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

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	75 ^H	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	19 ^L	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5438	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1378	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	72	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	362	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	190000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.29	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	15 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	119000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	62.7 ^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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	Dr. Vinay Cho MD (Pathology & M Chairman & Consu			(Pathology)
JAME	: Miss. JAGRITI MANCHANDA			
GE/ GENDER	: 38 YRS/FEMALE	PAT	FIENT ID	: 1740870
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ARCODE NO.	: 01524701	COL	LECTION DATE	: 31/Jan/2025 11:02AM
LIENT CODE.	: KOS DIAGNOSTIC LAB	REI	PORTING DATE	: 31/Jan/2025 11:42AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	MBALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
	ERYTHRO	CYTE SEDIME	NTATION RATE (1	ESR)
mmune disease, but An ESR can be affe s C-reactive protein this test may also ystemic lupus erythe CONDITION WITH LOV low ESR can be see polycythaemia), sigr s sickle cells in sickl IOTE: . ESR and C - reactive cenerally, ESR doe CRP is not affected for the ESR is elevate to Women tend to ha b. Drugs such as dext	does not tell the health practitione cted by other conditions besides in be used to monitor disease activity ematosus W ESR n with conditions that inhibit the n ificantly high white blood cell cour e cell anaemia) also lower the ESR e protein (C-RP) are both markers of s not change as rapidly as does CRI by as many other factors as is ESR , ed, it is typically a result of two typ ve a higher ESR, and menstruation	er exactly where the iflammation. For thi y and response to the normal sedimentation nt (leucocytosis), a conf inflammation. P, either at the star making it a better r bes of proteins, glob and pregnancy can	e inflammation is in the is reason, the ESR is typ nerapy in both of the a on of red blood cells, si nd some protein abno t of inflammation or as narker of inflammatior vulins or fibrinogen. cause temporary eleva	bicallý used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count rmalities. Some changes in red cell shape (such s it resolves.





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BARCODE NO.	:01524701			COLLECTION DATE	: 31/Jan/2025 11:02AM
CLIENT CODE.	: KOS DIAGNOS	TIC LAB		REPORTING DATE	: 31/Jan/2025 02:36PM
CLIENT ADDRESS	: 6349/1, NICH	OLSON ROAD, A	AMBALA CANTT		
Test Name			Value	Unit	Biological Reference interval
		CLINIC	AL CHEMIS	TRY/BIOCHEMIST	'RY
			GLUCOSE	FASTING (F)	
GLUCOSE FASTING	G (F): PLASMA Se - peroxidase (g	OD-POD)	104.9 ^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0

IN ACCRDANCE 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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		C hopra y & Microbiology) consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Miss. JAGRITI MANCHAN : 38 YRS/FEMALE : SURJESH : : 01524701 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROA	PA RI RI CC RI	ATIENT ID EG. NO./LAB NO. EGISTRATION DATE DLLECTION DATE EPORTING DATE	: 1740870 : 012501310027 : 31/Jan/2025 10:16 AM : 31/Jan/2025 11:02AM : 31/Jan/2025 02:59PM
Test Name		Value	Unit	Biological Reference interval
		LIPID PROF	II F · BASIC	
CHOLESTEROL TOT by CHOLESTEROL OX		165.69	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SI by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	71.97	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROI by SELECTIVE INHIBITI		55.22	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL by CALCULATED, SPE		96.08	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 CHOLEST by CALCULATED, SPE		110.47	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 CHOLESTERC	CTROPHOTOMETRY	14.39	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER by CALCULATED, SPE CHOLESTEROL/HD by CALCULATED, SPE	<i>сткорнотометку</i> L RATIO: SERUM	403.35 3	mg/dL RATIO	350.00 - 700.00 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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	Dr. Vinay Cho MD (Pathology & I Chairman & Const	Microbiology)	Dr. Yugam MD (CEO & Consultant	(Pathology)
NAME	: Miss. JAGRITI MANCHANDA			
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BARCODE NO.	: 01524701	C	COLLECTION DATE	: 31/Jan/2025 11:02AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	F	REPORTING DATE	: 31/Jan/2025 02:59PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		1.74	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		1.3 ^L	RATIO	3.00 - 5.00

<u>INTERPRETATION:</u> 1. Measurements in the same patient can show physiological& analytical variations. Three serial samples 1 week apart are recommended for

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

3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues. 4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL

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





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Test Name	Value	Unit	Biological Reference interval
LIVER	FUNCTION TH	EST (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	0.15	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	19.6	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	23.6	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.83	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	74.56	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	17.12	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.87	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.46	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.41	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.85	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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	Dr. Vinay Chopra	l Dr Yugar	n Chopra

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).

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6



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Test Name		Value	Unit	Biological Reference interval
	KIDNE	Y FUNCTION 1	TEST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAN	IATE DEHYDROGENASE (GLDH)	20.24	mg/dL	10.00 - 50.00
CREATININE: SERU	JM	0.99	mg/dL	0.40 - 1.20
BLOOD UREA NITE by CALCULATED, SPE	COGEN (BUN): SERUM	9.46	mg/dL	7.0 - 25.0
BLOOD UREA NITE RATIO: SERUM by CALCULATED, SPE	COGEN (BUN)/CREATININE	9.56 ^L	RATIO	10.0 - 20.0
UREA/CREATININ by CALCULATED, SPE	E RATIO: SERUM	20.44	RATIO	
URIC ACID: SERUM	[4.19	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.3	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE by PHOSPHOMOLYBE	ERUM DATE, SPECTROPHOTOMETRY	3.42	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	'E ELECTRODE)	138.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUE by ISE (ION SELECTIV		4.26	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIV	'E ELECTRODE)	103.88	mmol/L	90.0 - 110.0
ESTIMATED GLON	IERULAR FILTERATION RATE			
(eGFR): SERUM by CALCULATED	ERULAR FILTERATION RATE	74.8		
INTERPRETATION:	een nre- and nost renal azotemia			

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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Page 9 of 14

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Test Name		Value	Uni	t Bio	ological Reference interv
 Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia 	ass (subnormal creatinine pro tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal diseas	NE LEVELS: y more than creatinine) (e.g. obstructive	uropathy).	
 Certain drugs (e.g., NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia CECREASED RATIO (< Acute tubular necr Low protein diet and Severe liver diseas Other causes of definition of the second dialysis Severe liver diseas Other causes of definition of the second dialysis Severe liver diseas Other causes of definition of the second dialysis Severe liver diseas Other causes of definition of the second dialysis Severe liver diseas Pregnancy. DECREASED RATIO (Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido cephalosporin the second dialysis CKD STAGE G1 	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATIN a (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. a d starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually all of inappropiate antidiuretic ha 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine <u>JLAR FILTERATION RATE:</u> <u>DESCRIPTION</u> Normal kidney fu	NE LEVELS: y more than creatinine ise. offuses out of extracell osent in blood). rmone) due to tubular VINE: creatine to creatinine increase in creatinine e measurement). M GFR (mL/	ular fluid). secretion of urea. with certain meth <u>min/1.73m2)</u> >90	nodologies,resulting in ASSOCIATED FINDI No proteinuria	NGS
Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Perenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin the <u>STIMATED GLOMERI</u> CKD STAGE	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATIN a (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. a d starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic ha 10:1) WITH INCREASED CREATII py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine) 10:1) CREASED CREATION 10:1) CREASED CREATION 10:1) WITH INCREASED CREATION py (accelerates conversion of eleases muscle creatinine). who develop renal failure. 10:1) CREASED CREATION 10:1) CREASED CREA	NE LEVELS: y more than creatinine ise. offuses out of extracell osent in blood). rmone) due to tubular VINE: creatine to creatinine) increase in creatinine e measurement). Mathematical Content Mathematical Content with	ular fluid). secretion of urea. with certain meth min/1.73m2)	nodologies,resulting in ASSOCIATED FINDI No proteinuria Presence of Prote	NGS a ein ,
Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin thei STIMATED GLOMERI G1 G2	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATIN a (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. a d starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually all of inappropiate antidiuretic ha 10:1) WITH INCREASED CREATII py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine) LAR FILTERATION RATE: DESCRIPTION Normal kidney fu Kidney damage normal or high	NE LEVELS: y more than creatinine ise. offuses out of extracell osent in blood). rmone) due to tubular vine: creatine to creatinine increase in creatinine e measurement). M GFR (mL/ motion	ular fluid). secretion of urea. with certain meth <u>min/1.73m2)</u> >90 >90	nodologies,resulting in ASSOCIATED FINDI No proteinuria	NGS a ein ,
. Certain drugs (e.g. VCREASED RATIO (>2 . Postrenal azotemia Prerenal azotemia VECREASED RATIO (< . Acute tubular necr . Low protein diet al . Severe liver diseas . Other causes of de . Repeated dialysis . Inherited hyperam . SIADH (syndrome of . Pregnancy. VECREASED RATIO (< . Phenacimide thera . Rhabdomyolysis (r . Muscular patients VAPPROPIATE RATIO . Diabetic ketoacido hould produce an in . Cephalosporin thei STIMATED GLOMERI CKD STAGE G1	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATIN a (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. a d starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic ha 10:1) WITH INCREASED CREATII py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine) 10:1) CREASED CREATION 10:1) CREASED CREATION 10:1) WITH INCREASED CREATION py (accelerates conversion of eleases muscle creatinine). who develop renal failure. 10:1) CREASED CREATION 10:1) CREASED CREA	NE LEVELS: y more than creatinine ie. offuses out of extracell osent in blood). rmone) due to tubular NINE: creatine to creatinine increase in creatinine e measurement). N GFR GFR GFR	ular fluid). secretion of urea. with certain meth <u>min/1.73m2)</u> >90	nodologies,resulting in ASSOCIATED FINDI No proteinuria Presence of Prote	NGS a ein ,
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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS , MD (PATHOLOGY)









	Dr. Vinay Chopra MD (Pathology & Microbiolo Chairman & Consultant Path		(Pathology)
NAME	: Miss. JAGRITI MANCHANDA		
AGE/ GENDER	: 38 YRS/FEMALE	PATIENT ID	: 1740870
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012501310027
REFERRED BY	:	REGISTRATION DATE	: 31/Jan/2025 10:16 AM
BARCODE NO.	: 01524701	COLLECTION DATE	: 31/Jan/2025 11:02AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 31/Jan/2025 02:59PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CA	ANTT	
Test Name	Valu	e 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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CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 31/Jan/2025 12:43PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTI	r		
Test Name		Value	Unit	Biological Reference interval	
		ENDOC	CRINOLOGY		
	T	HYROID FUN	CTION TEST: TOTAL		
TRIIODOTHYRONII	NE (T3): SERUM ESCENT MICROPARTICLE IMMUNO	0.715 ASSAY)	ng/mL	0.35 - 1.93	
THYROXINE (T4): S	ERUM ESCENT MICROPARTICLE IMMUNO	6.52 ASSAY)	μgm/d	L 4.87 - 12.60	
	TING HORMONE (TSH): SEE		µIU/ml	0.35 - 5.50	
by CMIA (CHEMILUMIN 3rd GENERATION, ULT <u>INTERPRETATION</u> :	ESCENT MICROPARTICLE IMMUNO. RASENSITIVE	ASSAY)			
day has influence on the i	neasured serum TSH concentrations. ure at any level of regulation of the	TSH stimulates the p	roduction and secretion of the	<i>pm. The variation is of the order of 50%.Hence time of t</i> metabolically active hormones, thyroxine (T4)and her underproduction (hypothyroidism) or	
CLINICAL CONDITION	Т3		T4	TSH	
Primary Hypothyroidis			Reduced	Increased (Significantly)	
Subclinical Hypothyroi	lism: Normal or Lo	w Normal	Normal or Low Normal	High	

LIM	ΙΤΑΤ	IONS:-	

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

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.

Increased

Normal or High Normal

Reduced (at times undetectable)

Reduced

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	DDOTHYRONINE (T3) THYROXINE (T4)		(INE (T4)	THYROID STIMULATING HORMONE (TSH)		
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range (µIU/mL)	
0-7 Days	0.20 - 2.65	0 - 7 Days	5.90 - 18.58	0 - 7 Days	2.43 - 24.3	
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00	
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 - 17.04	3 Days – 6 Months	0.70 - 8.40	
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00	

Increased

Normal or High Normal





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Test Name			Value	Unit		Biological Reference interva
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	
	RECON	/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		

INCREASED TSH LEVELS:

1. Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.

2. Hypothyroid patients receiving insufficient thyroid replacement therapy.

3. Hashimotos thyroiditis

4.DRUGS: Amphetamines, iodine containing agents & dopamine antagonist.

5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

DECREASED TSH LEVELS:

1.Toxic multi-nodular goiter & Thyroiditis.

2. Over replacement of thyroid hormone in treatment of hypothyroidism.

3. Autonomously functioning Thyroid adenoma

4. Secondary pituitary or hypothalamic hypothyroidism

5. Acute psychiatric illness

6.Severe dehydration.

7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

8.Pregnancy: 1st and 2nd Trimester





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Ch MD (Pathology & Chairman & Cor			Dr. Yugam MD CEO & Consultant	(Pathology)	
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LIENT ADDRESS	: 6349/1, NICHOLS	SON ROAD, AMBALA CAN	NTT			
Fest Name		Value		Unit	Biological Reference interval	
		L L	/ITAM	INS		
		VITAMIN D/25	HYDR	OXY VITAMIN D	3	
by CLIA (CHEMILUMIN	DROXY VITAMIN D ESCENCE IMMUNOASSA			ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0	
<u>NTERPRETATION:</u> DFFI	CIENT:	< 20		n	ı/mL	
INSUF	FICIENT:	21 - 29	21 - 29		ng/mL	
	ED RANGE: CATION:	<u> </u>	9			
2.25-OHVitamin D r issue and tightly bou Vitamin D plays a p shosphate reabsorpt Severe deficiency n DECREASED: .Lack of sunshine ex .lnadeguate intake, Depressed Hepatic Secondarv to advar 0.Osteoporosis and S DEnzyme Inducing di NCREASED: . Hypervitaminosis I evere hypercalcemia cAUTION: Replaceme iypervitaminosis D	epresents the main b und by a transport pr rimary role in the ma- ion, skeletal calcium nay lead to failure to posure. malabsorption (celia Vitamin D 25- hydrox need Liver disease econdary Hyperpara- rugs: anti-epileptic dr D is Rare, and is seen a and hyperphophate ent therapy in deficier <i>individuals as compar</i>	otein while in circulation aintenance of calcium ho deposition, calcium mob mineralize newly formed ac disease) xylase activity throidism (Mild to Moder rugs like phenytoin, phen only after prolonged exp mia. nt individuals must be mo	ort form c n. omeostati ilization, d osteoid rate defic obarbital oosure to onitored l	of Vitamin D and trans s. It promotes calciun mainly regulated by p in bone, resulting in r ciency) and carbamazepine, extremely high doses by periodic assessmen	port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH). ickets in children and osteomalacia in adults. that increases Vitamin D metabolism. of Vitamin D. When it occurs, it can result in t of Vitamin D levels in order to prevent <i>iency due to excess of melanin pigment which</i>	
		*** End Of	f Repor	t ***		
	an	2	Ghof	bra		

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