

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar	obiology)	Dr. Yugam MD ( CEO & Consultant	(Pathology)
IAME	: Mrs. NARINDER KAUR			
AGE/ GENDER	: 37 YRS/FEMALE	J	PATIENT ID	: 1666236
COLLECTED BY	: SURJESH	I	REG. NO./LAB NO.	: 012411090021
REFERRED BY	:	l	REGISTRATION DATE	: 09/Nov/2024 10:04 AM
BARCODE NO.	: 01520410	-	COLLECTION DATE	: 09/Nov/2024 10:18AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 09/Nov/2024 10:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	ALA CANT I		
Fest Name		Value	Unit	<b>Biological Reference interval</b>
	SM/A ST	HVA WFI	LNESS PANEL: DT	
			OD COUNT (CBC)	
DED BLOOD CELL	(RBCS) COUNT AND INDICES	LEIE DLU	OD COUNT (CBC)	
HAEMOGLOBIN (H		11.9 <sup>L</sup>	gm/dL	12.0 - 16.0
by CALORIMETRIC			ů l	
ED BLOOD CELL (	RBC) COUNT OCUSING, ELECTRICAL IMPEDENCE	4.39	Millions/	cmm 3.50 - 5.00
PACKED CELL VOLU	JME (PCV) utomated hematology analyzer	38.5	%	37.0 - 50.0
	AR VOLUME (MCV)	87.7	fL	80.0 - 100.0
	utomated hematology analyzer AR HAEMOGLOBIN (MCH)	27.1	nd	27.0 - 34.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER		pg	
	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	30.9 <sup>L</sup>	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV)	14.5	%	11.00 - 16.00
-	utomated hematology analyzer UTION WIDTH (RDW-SD)	47.5	fL	35.0 - 56.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
MENTZERS INDEX by CALCULATED		19.98	RATIO	BETA THALASSEMIA TRAIT: < 13.0
				IRON DEFICIENCY ANEMIA:
GREEN & KING IND	DEX	28.96	RATIO	>13.0 BETA THALASSEMIA TRAIT:<
by CALCULATED				65.0
				IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE	LLS (WBCS)			
	COUNT (TLC)	7560	/cmm	4000 - 11000
		NIL		0.00 - 20.00
by flow cytometry NUCLEATED RED B	LOOD CELLS (nRBCS)	1111		
by FLOW CYTOMETRY NUCLEATED RED B by AUTOMATED 6 PAF	LOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER LOOD CELLS (nRBCS) %	NIL	%	< 10 %

KOS Diagnostic Lab (A Unit of KOS Healthcare)





DR.VINAY CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY) DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)

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

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Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Test Name	Value	Unit	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	57	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	35	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	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	4309	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2646	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	151	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	454	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	171000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.27	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	16 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	118000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	69.1 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	15.9	%	15.0 - 17.0



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Cest Name		Value	Unit	<b>Biological Reference interval</b>
in mis cost may also	ematosus	ing and response to t	nerapy in both of the a	bove diseases as well as some others, such as





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Test Name		Value	Unit	<b>Biological Reference interval</b>
	CLINI	CAL CHEMISTR GLUCOSE FA	Y/BIOCHEMIST STING (F)	TRY
GLUCOSE FASTING by GLUCOSE OXIDAS	(F): PLASMA E - PEROXIDASE (GOD-POD)	107.07 <sup>H</sup>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

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**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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Page 4 of 14





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Test Name		Value	Unit	<b>Biological Reference interval</b>
		I IPID PRO	FILE : BASIC	
CHOLESTEROL TO	TAL SERIM	169.87	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		109.87	ing/ dL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S. by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	118.87	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERO	L (DIRECT): SERUM ION	51.45	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		94.65	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		118.42	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		23.77	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER by CALCULATED, SPE	CUM	458.61	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPE	L RATIO: SERUM	3.3	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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Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		1.84	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	2.31 <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.

 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.
 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	<b>Biological Reference interval</b>
	LIVER	FUNCTION 7	TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI	: SERUM PECTROPHOTOMETRY	0.55	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.14	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE by CALCULATED, SPE	CT (UNCONJUGATED): SERUM	0.41	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	20.5	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	22.8	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE		0.9	RATIO	0.00 - 46.00
ALKALINE PHOSPI by para nitrophen propanol	HATASE: SERUM yl phosphatase by amino methyl	80.34	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTROF	L TRANSFERASE (GGT): SERUM	17.76	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		7.4	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G	REEN	4.16	gm/dL	3.50 - 5.50
GLOBULIN: SERUM	1	3.24	gm/dL	2.30 - 3.50
A : G RATIO: SERUI		1.28	RATIO	1.00 - 2.00

A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY

INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

# **INCREASED:**

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)



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### DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)

2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

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	<b>Biological Reference interva</b>	1
	KIDNI	EY FUNCTION 7	FEST (COMPLETE)		
UREA: SERUM	IATE DEHYDROGENASE (GLDH)	21.81	mg/dL	10.00 - 50.00	
CREATININE: SERU	UM	0.94	mg/dL	0.40 - 1.20	
•	ROGEN (BUN): SERUM	10.19	mg/dL	7.0 - 25.0	
	ROGEN (BUN)/CREATININE	10.84	RATIO	10.0 - 20.0	
by CALCULATED, SPE					
UREA/CREATININ by CALCULATED, SPE		23.2	RATIO		
URIC ACID: SERUM		4.78	mg/dL	2.50 - 6.80	
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.89	mg/dL	8.50 - 10.60	
PHOSPHOROUS: SE	ERUM DATE, SPECTROPHOTOMETRY	3.33	mg/dL	2.30 - 4.70	
<b>ELECTROLYTES</b>					
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	143.6	mmol/L	135.0 - 150.0	
POTASSIUM: SERU		4.01	mmol/L	3.50 - 5.00	
CHLORIDE: SERUM	1	107.7	mmol/L	90.0 - 110.0	
	IERULAR FILTERATION RATE				
(eGFR): SERUM by CALCULATED	ERULAR FILTERATION RATE	80.1			
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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Fest Name			Value	Un	it	Biolog	ical Referen	ce interva
9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< <sup>-</sup> 1. Acute tubular necr	kia, high fever (e.g. ureter cc ass (subnorma tetracycline, g 0:1) WITH ELE (BUN rises dis superimposed 0:1) WITH DEC osis.	lostomy) Il creatinine productior lucocorticoids) /ATED CREATININE LEVI sproportionately more t on renal disease.	) LS:			Cushing's synd	rome, high pr	otein diet,
7. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. <b>INCREASED RATIO (&gt;2</b> 1. Postrenal azotemia 2. Prerenal azotemia <b>DECREASED RATIO (</b> 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. <b>DECREASED RATIO (</b> 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients <b>INAPPROPIATE RATIO</b> 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther <b>ESTIMATED GLOMERL</b> <b>OKD STAGE</b>	kia, high fever (e.g. ureter cc ass (subnorma tetracycline, g <b>D:1) WITH ELE</b> (BUN rises dis superimposed <b>0:1) WITH DEC</b> osis. d starvation. creased urea s urea rather th monemias (ur f inappropiate <b>0:1) WITH INC</b> oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/o apy (interfere: LAR FILTERATI	). Iostomy) I creatinine productior Iucocorticoids) /ATED CREATININE LEVI sproportionately more to on renal disease. REASED BUN : ynthesis. an creatinine diffuses of the asses of the second antidiuretic harmone) REASED CREATININE: s conversion of creating the causes false increases creatinine ratio). s with creatinine measu ON RATE: DESCRIPTION	) LS: han creatinine ut of extracell blood). due to tubular to creatinine) e in creatinine rement). GFR ( mL/	) (e.g. obstructive ular fluid). secretion of urea with certain met	e uropathy). a. hodologies,r	esulting in no TED FINDINGS	rmal ratio wh	
7. Urine reabsorption     8. Reduced muscle m     9. Certain drugs (e.g.     NCREASED RATIO (>2     1. Postrenal azotemia     2. Prerenal azotemia     2. Prerenal azotemia     2. Certain drugs (e.g.     1. Acute tubular necr     2. Low protein diet ar     3. Severe liver disease     4. Other causes of de     5. Repeated dialysis (     5. Inherited hyperam     7. SIADH (syndrome of     8. Pregnancy.     7. SIADH (syndrome of     7. SIADH (syndrome of	kia, high fever (e.g. ureter cc ass (subnorma tetracycline, g <b>0:1) WITH ELE</b> (BUN rises dis superimposed <b>0:1) WITH DEC</b> osis. d starvation. creased urea s urea rather th monemias (ur f inappropiate <b>0:1) WITH INC</b> oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/o apy (interfere: LAR FILTERATI	). Iostomy) I creatinine productior Iucocorticoids) /ATED CREATININE LEVI sproportionately more to on renal disease. REASED BUN : ynthesis. an creatinine diffuses of the antidiuretic harmone) REASED CREATININE: s conversion of creating the causes false increases creatinine ratio). s with creatinine measu ON RATE: DESCRIPTION prmal kidney function	) LS: han creatinine ut of extracell blood). due to tubular to creatinine) e in creatinine rement). GFR ( mL/	) (e.g. obstructive ular fluid). secretion of urea with certain met <u>'min/1.73m2 )</u> >90	e uropathy). a. hodologies,r <b>ASSOCIA</b> No p	esulting in no TED FINDINGS roteinuria	rmal ratio wh	
A. Urine reabsorption     Reduced muscle m     Certain drugs (e.g.     NCREASED RATIO (>2     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Acute tubular necr     Severe liver disease     Other causes of de     Severe liver disease     Acute tubular necr     Severe liver disease     Other causes of de     Severe liver disease     Other causes of de     Severe liver disease     Other causes of de     Severe liver disease     Naperated dialysis (     Diabetic disease     Nappropiate RATIO     Diabetic ketoacido     should produce an in     Cephalosporin ther     STIMATED GLOMERL     OKD STAGE	kia, high fever (e.g. ureter cc ass (subnorma tetracycline, g <b>D:1) WITH ELE</b> (BUN rises dis superimposed <b>0:1) WITH DEC</b> osis. d starvation. creased urea s urea rather th monemias (ur f inappropiate <b>0:1) WITH INC</b> oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/o apy (interfere: LAR FILTERATI	). Iostomy) I creatinine productior Iucocorticoids) /ATED CREATININE LEVI sproportionately more to on renal disease. REASED BUN : ynthesis. an creatinine diffuses of the asses as virtually absent in the antidiuretic harmone) REASED CREATININE: s conversion of creatine the causes false increases creatinine ratio). s with creatinine measu ON RATE: DESCRIPTION ormal kidney function Kidney damage with normal or high GFR	) LS: han creatinine ut of extracell blood). due to tubular to creatinine) e in creatinine rement). GFR ( mL/	) (e.g. obstructive ular fluid). secretion of urea with certain met	e uropathy). a. hodologies,r ASSOCIA Presence	esulting in no TED FINDINGS	rmal ratio wh	
7. Urine reabsorption     8. Reduced muscle m     9. Certain drugs (e.g.     NCREASED RATIO (>2     1. Postrenal azotemia     2. Prerenal azotemia     2. Prerenal azotemia     2. Cow protein diet ar     3. Severe liver disease     4. Other causes of de     5. Repeated dialysis (     5. Inherited hyperam     7. SIADH (syndrome of     8. Pregnancy.     7. Pregnancy.     7. Phenacimide thera     7. Rhabdomyolysis (r     7. Diabetic ketoacido     5. hould produce an in     7. Cephalosporin ther     5. STIMATED GLOMERL     61     62	kia, high fever (e.g. ureter cc ass (subnorma tetracycline, g <b>D:1) WITH ELE</b> (BUN rises dis superimposed <b>0:1) WITH DEC</b> osis. d starvation. creased urea s urea rather th monemias (ur f inappropiate <b>0:1) WITH INC</b> oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/c apy (interfere: LAR FILTERATI	). Iostomy) I creatinine productior Iucocorticoids) /ATED CREATININE LEVI sproportionately more to on renal disease. REASED BUN : ynthesis. an creatinine diffuses of the asses of the second antidiuretic harmone) REASED CREATININE: s conversion of creatine the causes false increases creatinine ratio). s with creatinine measu ON RATE: DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION DESCRIPTION A decrease in GFR /ild decrease in GFR	) LS: han creatinine ut of extracell blood). due to tubular to creatinine rement). GFR (mL/	) (e.g. obstructive ular fluid). secretion of urea with certain met <u>(min/1.73m2)</u> >90 >90	e uropathy). a. hodologies,r ASSOCIA Presence	esulting in no TED FINDINGS roteinuria e of Protein ,	rmal ratio wh	
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. <b>INCREASED RATIO (&gt;2</b> 1. Postrenal azotemia <b>DECREASED RATIO (</b> 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. <b>DECREASED RATIO (</b> 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. <b>DECREASED RATIO (</b> 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients <b>INAPPROPIATE RATIO</b> 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther <b>ESTIMATED GLOMERU</b> <b>G1</b> <b>G2</b>	kia, high fever (e.g. ureter cc ass (subnorma tetracycline, g <b>D:1) WITH ELE</b> (BUN rises dis superimposed <b>0:1) WITH DEC</b> osis. d starvation. creased urea s urea rather th monemias (ur f inappropiate <b>0:1) WITH INC</b> oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/a apy (interfere: LAR FILTERATI	). Iostomy) I creatinine productior Iucocorticoids) /ATED CREATININE LEVI sproportionately more to on renal disease. REASED BUN : ynthesis. an creatinine diffuses of the asses as virtually absent in the antidiuretic harmone) REASED CREATININE: s conversion of creatine the causes false increases creatinine ratio). s with creatinine measu ON RATE: DESCRIPTION ormal kidney function Kidney damage with normal or high GFR	) LS: han creatinine ut of extracell blood). due to tubular to creatinine rement). GFR (mL/	<ul> <li>(e.g. obstructive</li> <li>ular fluid).</li> <li>secretion of urea</li> <li>with certain met</li> <li><u>/min/1.73m2 )</u></li> <li>&gt;90</li> <li>&gt;90</li> </ul>	e uropathy). a. hodologies,r ASSOCIA Presence	esulting in no TED FINDINGS roteinuria e of Protein ,	rmal ratio wh	





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	<b>Dr. Vinay Chopra</b> MD (Pathology & Micro Chairman & Consultant	obiology) ME	n Chopra 9 (Pathology) 1t Pathologist
NAME	: Mrs. NARINDER KAUR		
AGE/ GENDER	: 37 YRS/FEMALE	PATIENT ID	: 1666236
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012411090021
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 09/Nov/2024 10:04 AM
BARCODE NO.	: 01520410	COLLECTION DATE	:09/Nov/2024 10:18AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	:09/Nov/2024 12:03PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	LA CANTT	
Test Name		Value Unit	<b>Biological Reference interval</b>

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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	Dr. Vinay Cho MD (Pathology & Chairman & Cons	Microbiology)		m Chopra D (Pathology) nt Pathologist	
NAME	: Mrs. NARINDER KAUR				
AGE/ GENDER	: 37 YRS/FEMALE	I	PATIENT ID	: 1666236	
COLLECTED BY	: SURJESH	I	REG. NO./LAB NO.	:012411090021	
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT			
Test Name		Value	Unit	Biological Refe	rence interval
		ENDOCR	INOLOGY		
	THY	ROID FUNCT	ION TEST: TOTAL		
TRIIODOTHYRONI	NE (T3): SERUM IESCENT MICROPARTICLE IMMUNOAS	0.587 SAY)	ng/mL	0.35 - 1.93	
THYROXINE (T4): S	SERUM IESCENT MICROPARTICLE IMMUNOAS	6.78 SAY)	µgm/d]	L 4.87 - 12.60	
	TING HORMONE (TSH): SERU		µIU/mI	L 0.35 - 5.50	
3rd GENERATION, ULT <u>INTERPRETATION</u> :	RASENSITIVE				
day has influence on the triiodothyronine (T3).Fai	circadian variation, reaching peak levels I measured serum TSH concentrations. TSH lure at any level of regulation of the hyp rroidism) of T4 and/or T3.	I stimulates the prod	uction and secretion of the	, metabolically active hormones, thyr	oxine (T4)and
CLINICAL CONDITION	Т3		T4	TSH	]
Primary Hypothyroidis	m: Reduced		Reduced	Increased (Significantly)	

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	YRONINE (T3)	THYROX	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





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	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Patholog		(Pathology)
NAME	: Mrs. NARINDER KAUR		
AGE/ GENDER	: 37 YRS/FEMALE	PATIENT ID	: 1666236
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Test Name			Value	Unit	t	Biological Reference interval
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	
	RECO	MMENDATIONS 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 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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		Dr. Vinay Cho MD (Pathology & Chairman & Cons	Microbiology)		(Pathology)
NAME	: Mrs. NARI	NDER KAUR			
AGE/ GENDER	: 37 YRS/FEN	<b>MALE</b>		PATIENT ID	: 1666236
COLLECTED BY	: SURJESH			REG. NO./LAB NO.	: 012411090021
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CLIENT ADDRESS	: 6349/1, NI	CHOLSON ROAD, A	MBALA CANTT		
Test Name			Value	Unit	<b>Biological Reference interval</b>
			VIT	AMINS	
		VITA	MIN D/25 HY	YDROXY VITAMIN D	3
VITAMIN D (25-HY by CLIA (CHEMILUMIN			16.076 <sup>L</sup>	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
<u>NTERPRETATION:</u> DEFI	CIENT:		< 20	n	g/mL
	FICIENT:		21 - 29		g/mL
	ED RANGE: CATION:		30 - 100 > 100		g/mL g/mL
issue and tightly boo 3. Vitamin D plays a p shosphate reabsorpt 4. Severe deficiency r DECREASED: 1. Lack of sunshine ex 2. Inadequate intake, 3. Depressed Hepatic 4. Secondary to advar 5. Osteoporosis and S 5. Enzyme Inducing di NCREASED: 1. Hypervitaminosis I severe hypercalcemia CAUTION: Replaceme hypervitaminosis D	und by a transp primary role in t ion, skeletal ca nay lead to failu posure. malabsorption Vitamin D 25-1 need Liver disea econdary Hype rugs: anti-epile D is Rare, and is a and hyperpho ent therapy in d <i>individuals as co</i>	port protein while the maintenance of licium deposition, ure to mineralize r (celiac disease) hvdroxylase activit ase erparathroidism (N ptic drugs like phe s seen only after prophatemia. leficient individuals compare to whites, i	in circulation. If calcium homeo calcium mobiliza ewly formed ost y lild to Moderate nytoin, phenoba rolonged exposu s must be monito	ostatis. It promotes calcium ition, mainly regulated by p teoid in bone, resulting in r deficiency) rbital and carbamazepine, re to extremely high doses pred by periodic assessmen	port form of Vitamin D, being stored in adipos n absorption, renal calcium absorption and barathyroid harmone (PTH). ickets in children and osteomalacia in adults. that increases Vitamin D metabolism. of Vitamin D. When it occurs, it can result in it of Vitamin D levels in order to prevent <i>iency due to excess of melanin pigment which</i>
		*	** End Of Re	eport ***	
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