



	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultan	obiology)	ME	m Chopra D (Pathology) ht Pathologist	
NAME : Mr	rs. AARTI				
AGE/ GENDER : 46	YRS/FEMALE		PATIENT ID	: 168459	99
COLLECTED BY :			REG. NO./LAB NO.	:01241	1280027
<b>REFERRED BY</b> :			<b>REGISTRATION DATE</b>	:28/Nov	v/2024 10:59 AM
	521600		COLLECTION DATE		v/2024 12:40PM
	S DIAGNOSTIC LAB		REPORTING DATE	: 28/Nov	v/2024 11:32AM
CLIENT ADDRESS : 63	49/1, NICHOLSON ROAD, AMBA	ALA CANTI	ſ		
Test Name		Value	Unit		Biological Reference interval
	СОМР		ELLNESS PANEL: D .00D COUNT (CBC)	т	
	<u>CS) COUNT AND INDICES</u>		(17		
HAEMOGLOBIN (HB) by CALORIMETRIC		8.7 <sup>L</sup>	gm/dL		12.0 - 16.0
RED BLOOD CELL (RBC)	COUNT NG, ELECTRICAL IMPEDENCE	3.86	Millions	s/cmm	3.50 - 5.00
PACKED CELL VOLUME (		29.7 <sup>L</sup>	%		37.0 - 50.0
MEAN CORPUSCULAR VO		77 <sup>L</sup>	fL		80.0 - 100.0
MEAN CORPUSCULAR HA		22.7 <sup>L</sup>	pg		27.0 - 34.0
	EMOGLOBIN CONC. (MCHC) ATED HEMATOLOGY ANALYZER	29.4 <sup>L</sup>	g/dL		32.0 - 36.0
RED CELL DISTRIBUTION by CALCULATED BY AUTOM	N WIDTH (RDW-CV) ATED HEMATOLOGY ANALYZER	16.1 <sup>H</sup>	%		11.00 - 16.00
RED CELL DISTRIBUTION	N WIDTH (RDW-SD) ated hematology analyzer	46.2	fL		35.0 - 56.0
MENTZERS INDEX by CALCULATED		19.95	RATIO		BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED WHITE BLOOD CELLS (1	WR(S)	32.35	RATIO		BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
TOTAL LEUCOCYTE COUL	NT (TLC)	4710	/cmm		4000 - 11000
NUCLEATED RED BLOOI by AUTOMATED 6 PART HEM	O CELLS (nRBCS)	NIL			0.00 - 20.00
NUCLEATED RED BLOOI		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

NAME	: Mrs. AARTI		
AGE/ GENDER	: 46 YRS/FEMALE	PATIENT ID	: 1684599
COLLECTED BY	:	REG. NO./LAB NO.	: 012411280027
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 28/Nov/2024 10:59 AM
BARCODE NO.	: 01521600	<b>COLLECTION DATE</b>	: 28/Nov/2024 12:40PM
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by flow cytometry by sf cube & microscopy	47 <sup>L</sup>	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	42 <sup>H</sup>	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1	%	1 - 6
MONOCYTES by flow cytometry by SF cube & microscopy	10	%	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	2214	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1978	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by flow cytometry by sf cube & microscopy	47	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	471	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	316000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.4 <sup>H</sup>	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	13 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	146000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	46.3 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	15.6	%	15.0 - 17.0



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CLIENT CODE.	: KOS DIAGNOS	TIC LAB		<b>REPORTING DATE</b>	: 28/Nov/2024 11:47AM
CLIENT ADDRESS	: 6349/1, NICH	OLSON ROAD, AM	IBALA CANTT		
Test Name			Value	Unit	Biological Reference interval
ERYTHROCYTE SE by red cell aggre NTERPRETATION:			12	mm/1st	hr 0 - 20





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		Chopra y & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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BARCODE NO.	:01521600	COLLE	CTION DATE	: 28/Nov/2024 12:40PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 28/Nov/2024 01:26PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMISTRY/	BIOCHEMIST	RY
		GLUCOSE FAST	'ING (F)	

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





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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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<b>LIENT CODE.</b> : KOS DIAG	NOSTIC LAB		REPORTING DATE	: 28/Nov/2024 01:56PM
<b>LIENT ADDRESS</b> : 6349/1, N	ICHOLSON ROAD, AMBA	LA CANTT		
Fest Name		Value	Unit	<b>Biological Reference interval</b>
	Т	IPID PRO	FILE : BASIC	
CHOLESTEROL TOTAL: SERUM		124.41	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OXIDASE PAP		1 ~ 1, 1 1	ing, ut	BORDERLINE HIGH: 200.0 -
				239.0 HIGH CHOLESTEROL: > OR =
				1000000000000000000000000000000000000
RIGLYCERIDES: SERUM		83.42	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	E (ENZYMATIC)			BORDERLINE HIGH: 150.0 - 199.0
				HIGH: 200.0 - 499.0
				VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): by SELECTIVE INHIBITION	SERUM	53.69	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0
				60.0
		00.04	. / 17	HIGH HDL: $> OR = 60.0$
DL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOM	ETRY	60.04	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129
				BORDERLINE HIGH: 130.0 -
				159.0 HIGH: 160.0 - 189.0
				VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERU		70.72	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPECTROPHOTOM	EIRY			ABOVE OPTIMAL: 130.0 - 159 BORDERLINE HIGH: 160.0 -
				189.0
				HIGH: 190.0 - 219.0
/LDL CHOLESTEROL: SERUM		16.68	mg/dL	VERY HIGH: > OR = 220.0 0.00 - 45.00
by CALCULATED, SPECTROPHOTOM	ETRY			
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOM	ETRY	338.24 <sup>L</sup>	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SE	CRUM	2.32	RATIO	LOW RISK: 3.30 - 4.40
by CALCULATED, SPECTROPHOTOM	ETRY			AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0
				HIGH RISK: > 11.0
15285429466		/	n	



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA	CANTT	
Test Name	Va	lue Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		12 RATIO	D LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		55 <sup>L</sup> RATIO	3.00 - 5.00

## INTERPRETATION:

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

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

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

Dr. Yugam Chopra

MD (Pathology)

:1684599

:012411280027

: 28/Nov/2024 10:59 AM

: 28/Nov/2024 12:40PM

: 28/Nov/2024 01:56PM

**Biological Reference interval** 

Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** : Mrs. AARTI AGE/ GENDER : 46 YRS/FEMALE **PATIENT ID COLLECTED BY** REG. NO./LAB NO. : **REFERRED BY REGISTRATION DATE** : **BARCODE NO.** :01521600 **COLLECTION DATE** CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Value

LIVER FUNCTION TEST (COMPLETE)							
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	1.44 <sup>H</sup>	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20				
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.41 <sup>H</sup>	mg/dL	0.00 - 0.40				
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	1.03 <sup>H</sup>	mg/dL	0.10 - 1.00				
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	28	U/L	7.00 - 45.00				
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	20.8	U/L	0.00 - 49.00				
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.35	RATIO	0.00 - 46.00				
ALKALINE PHOSPHATASE: SERUM by Para Nitrophenyl phosphatase by amino methyl propanol	72.87	U/L	40.0 - 130.0				
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	10.43	U/L	0.00 - 55.0				
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.51	gm/dL	6.20 - 8.00				
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.56	gm/dL	3.50 - 5.50				
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.95	gm/dL	2.30 - 3.50				
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.55	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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NAME

Test Name





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Test Name		Value Unit	Biological Reference interval

### 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).

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
	KIDNI	EY FUNCTIO	N TEST (COMPLETE)	
UREA: SERUM		19.23	mg/dL	10.00 - 50.00
•	MATE DEHYDROGENASE (GLDH)	0.91	ma/dI	0.40 - 1.20
CREATININE: SERI		0.91	mg/dL	0.40 - 1.20
BLOOD UREA NITE by CALCULATED, SPE	ROGEN (BUN): SERUM	8.99	mg/dL	7.0 - 25.0
	ROGEN (BUN)/CREATININE	9.88 <sup>L</sup>	RATIO	10.0 - 20.0
UREA/CREATININ by CALCULATED, SPE	E RATIO: SERUM	21.13	RATIO	
URIC ACID: SERUM		2.51	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	10.04	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE by PHOSPHOMOLYBL	ERUM DATE, SPECTROPHOTOMETRY	3.08	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	141	mmol/L	135.0 - 150.0
POTASSIUM: SERU by ISE (ION SELECTIV		3.99	mmol/L	3.50 - 5.00
CHLORIDE: SERUN by ISE (ION SELECTIV	1 /E ELECTRODE)	105.75	mmol/L	90.0 - 110.0
ESTIMATED GLON	<b>IERULAR FILTERATION RATE</b>			

# <u>ESTIMATED GLOMERULAR FILTERATION RATE</u>

ESTIMATED GLOMERULAR FILTERATION RATE (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.

78.8

2. Catabolic states with increased tissue breakdown.

3. GI haemorrhage.



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CLIENT ADDRESS	: 6349/1, NIC	HOLSON ROAD, AM	IBALA CANT'	Г						
Test Name			Value	Un	uit	Bie	ological	Refere	nce inte	erval
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2	ass (subnormal tetracycline, glu <b>0:1) WITH ELEV/</b> (BUN rises disp	ostomy) creatinine producti cocorticoids) I <b>TED CREATININE LE</b> roportionately mor	on) E <b>VELS</b> :	tion, GI bleeding, thy nine) (e.g. obstructive		-	syndrom	e, high p	protein c	liet,
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI CKD STAGE G1	(e.g. ureter cold ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. Id starvation. 2: creased urea sy urea rather tha monemias (urea f inappropiate a 0:1) WITH INCR oy (accelerates eleases muscle who develop re sis (acetoacetat creased BUN/cr apy (interferes LAR FILTERATIO	estomy) creatinine producti cocorticoids) <b>ITED CREATININE LE</b> roportionately mor n renal disease. <b>EASED BUN :</b> The creatinine diffuse is virtually absent is virtually absent intidiuretic harmon <b>EASED CREATININE:</b> conversion of creat creatinine). nal failure. e causes false increat creatinine ratio). with creatinine meat <u>N RATE: DESCRIPTION</u> mal kidney functio	on) EVELS: e than creating s out of extra in blood). e) due to tub ine to creating ease in creating surement).	nine) (e.g. obstructive acellular fluid). ular secretion of urea nine). hine with certain met <u>mL/min/1.73m2 )</u> >90	e uropath a. hodologi ASSO	y). es,resulting i <u>CIATED FIND</u> o proteinuri	n norma INGS			
7. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. <b>NCREASED RATIO (&gt;2</b> 4. Postrenal azotemia 5. Prerenal azotemia <b>DECREASED RATIO (</b> 4. Acute tubular necr 5. Low protein diet an 6. Severe liver diseas 6. Other causes of de 6. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. 5. Pregnancy. 5. Pregnancy. 6. Phenacimide thera 6. Rabdomyolysis (r 8. Muscular patients <b>NAPPROPIATE RATIO</b> 6. Diabetic ketoacido 6. Nould produce an in 6. Cephalosporin ther 5. STIMATED GLOMERL CKD STAGE	(e.g. ureter cold ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. Id starvation. 2: creased urea sy urea rather tha monemias (urea f inappropiate a 0:1) WITH INCR oy (accelerates eleases muscle who develop re sis (acetoacetat creased BUN/cr apy (interferes of LAR FILTERATIO	estomy) creatinine producti cocorticoids) <b>ITED CREATININE LE</b> roportionately mor n renal disease. <b>EASED BUN :</b> The creatinine diffuse is virtually absent is virtually absent intidiuretic harmon <b>EASED CREATININE:</b> conversion of creat creatinine). nal failure. e causes false increat creatinine ratio). with creatinine meat <u>N RATE:</u> <u>DESCRIPTION</u> mal kidney functio dney damage with	on) EVELS: e than creating s out of extra in blood). e) due to tub ine to creating ease in creating surement).	nine) (e.g. obstructive acellular fluid). ular secretion of urea nine). hine with certain met mL/min/1.73m2 )	e uropath a. hodologi ASSO	y). es,resulting i <u>CIATED FIND</u> o proteinuria ence of Prote	n norma INGS a			
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 an     3. Severe liver diseas     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 there     7. STATED GLOMERI     7. CALL     7. CALL	(e.g. ureter cold ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. Id starvation. 2: creased urea sy urea rather tha monemias (urea f inappropiate a 0:1) WITH INCR oy (accelerates eleases muscle who develop re sis (acetoacetat creased BUN/cr apy (interferes LAR FILTERATIO	estomy) creatinine producti icocorticoids) <b>ITED CREATININE LE</b> roportionately mor n renal disease. <b>EASED BUN :</b> The creatinine diffuse is virtually absent is virtually absent intidiuretic harmon <b>EASED CREATININE:</b> conversion of creat creatinine). nal failure. te causes false increat creatinine ratio). with creatinine meat <b>N RATE:</b> DESCRIPTION mal kidney function dney damage with prmal or high GFR	on) EVELS: the than creation is out of extra in blood). e) due to tub ine to creation ease in creation surement). GFR (	nine) (e.g. obstructive acellular fluid). ular secretion of urea nine). hine with certain met <u>mL/min/1.73m2 ) &gt;90 &gt;90</u>	e uropath a. hodologi ASSO	y). es,resulting i <u>CIATED FIND</u> o proteinuri	n norma INGS a			
7. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. <b>NCREASED RATIO (&gt;2</b> 1. Postrenal azotemia <b>DECREASED RATIO (</b> 2. Prerenal azotemia <b>DECREASED RATIO (</b> 2. Low protein diet and 3. Severe liver diseas 4. Other causes of dec 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> 6. Muscular patients <b>NAPPROPIATE RATIO</b> 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther <b>ESTIMATED GLOMERI</b> <b>G1</b> <b>G2</b> <b>G3a</b>	(e.g. ureter cold ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. Id starvation. 2: creased urea sy urea rather tha monemias (urea f inappropiate a 0:1) WITH INCR oy (accelerates eleases muscle who develop re sis (acetoacetat creased BUN/cr apy (interferes of LAR FILTERATIO	estomy) creatinine producti icocorticoids) <b>ITED CREATININE LE</b> roportionately mor in renal disease. <b>EASED BUN :</b> The creatinine diffuse is virtually absent intidiuretic harmon <b>EASED CREATININE:</b> conversion of creat creatinine). hal failure. to causes false increat creatinine ratio). with creatinine meat <b>N RATE:</b> DESCRIPTION mal kidney functio dney damage with prmal or high GFR Id decrease in GFR	on) EVELS: the than creating in blood). e) due to tub ine to creating ease in creating surement). GFR (	nine) (e.g. obstructive acellular fluid). ular secretion of urea nine). hine with certain met <u>mL/min/1.73m2 ) &gt;90 &gt;90 60 -89</u>	e uropath a. hodologi ASSO	y). es,resulting i <u>CIATED FIND</u> o proteinuria ence of Prote	n norma INGS a			
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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS , MD (PATHOLOGY)









: 6349/1, NICHOLSON ROAD, A	MBALA CANTT	
: 6349/1, NICHOLSON ROAD, A	MBALA CANTT	
: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 28/Nov/2024 01:56PM
:01521600	COLLECTION DATE	: 28/Nov/2024 12:40PM
:	<b>REGISTRATION DATE</b>	: 28/Nov/2024 10:59 AM
:	<b>REG. NO./LAB NO.</b>	:012411280027
: 46 YRS/FEMALE	PATIENT ID	: 1684599
: Mrs. AARTI		
· · · · · · · · · · · · · · · · · · ·	G, /	9 (Pathology) t Pathologist
	MD (Pathology & N Chairman & Consu : Mrs. AARTI : 46 YRS/FEMALE : : : 01521600	MD (Pathology & Microbiology) Chairman & Consultant Pathologist CEO & Consultant : Mrs. AARTI : 46 YRS/FEMALE PATIENT ID :

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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	<b>Dr. Vinay Ch</b> MD (Pathology & Chairman & Con		Dr. Yugam Chopra MD (Pathology) t CEO & Consultant Pathologist		
NAME	: Mrs. AARTI				
AGE/ GENDER	: 46 YRS/FEMALE	P	ATIENT ID	: 1684599	
COLLECTED BY	:	R	EG. NO./LAB NO.	:012411280027	
REFERRED BY	:	R	EGISTRATION DATE	: 28/Nov/2024 10:59 AM	
BARCODE NO.	: 01521600	C	OLLECTION DATE	: 28/Nov/2024 12:40PM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	R	EPORTING DATE	: 28/Nov/2024 03:56PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT			
Test Name		Value	Unit	Biological Referen	ce interval
	ТН		INOLOGY ION TEST: TOTAL		
TRIIODOTHYRONI	NE (T3): SERUM	1.073 SSAY)	ng/mL	0.35 - 1.93	
THYROXINE (T4): S	SERUM iescent microparticle immunoas	6.23 SSAY)	µgm/dl	L 4.87 - 12.60	
	ATING HORMONE (TSH): SERU		µIU/mI	0.35 - 5.50	
<b>3rd GENERATION, ULT</b>	RASENSITIVE				
INTERPRETATION:					
day has influence on the triiodothyronine (T3).Fai	measured serum TSH concentrations. TS	H stimulates the prod	uction and secretion of the	pm. The variation is of the order of 50%. metabolically active hormones, thyroxin her underproduction (hypothyroidism) o	e (T4)and
CLINICAL CONDITION	T3		T4	TSH	
Primary Hypothyroidis			Reduced	Increased (Significantly)	
Subclinical Hypothyroi	dism: Normal or Low	Normal No	ormal or Low Normal	High	

#### LIMITATIONS:-

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT

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	YRONINE (T3)	NE (T3) THYROXINE (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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	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologi		(Pathology)
NAME	: Mrs. AARTI		
AGE/ GENDER	: 46 YRS/FEMALE	PATIENT ID	: 1684599
COLLECTED BY	:	REG. NO./LAB NO.	: 012411280027
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 28/Nov/2024 10:59 AM
BARCODE NO.	: 01521600	COLLECTION DATE	: 28/Nov/2024 12:40PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 28/Nov/2024 03:56PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT	ſ	

1 - 10 Years 11 - 19 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50		
11 - 10 Voars					
II - I/ Ical3	4.87-13.20	11 – 19 Years	0.50 - 5.50		
> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35-5.50		
MENDATIONS OF TSH LE	VELS DURING PRE	GNANCY ( µIU/mL)			
		0.10 - 2.50			
		0.20 - 3.00			
		0.30 - 4.10			
	. ,		MENDATIONS OF TSH LEVELS DURING PREGNANCY ( μIU/mL) 0.10 - 2.50 0.20 - 3.00	MENDATIONS OF TSH LEVELS DURING PREGNANCY (μIU/mL) 0.10 - 2.50 0.20 - 3.00	MENDATIONS OF TSH LEVELS DURING PREGNANCY ( μIU/mL) 0.10 - 2.50 0.20 - 3.00

### **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.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)







		Chopra / & Microbiology) onsultant Pathologist		(Pathology)
NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Mrs. AARTI : 46 YRS/FEMALE : : : 01521600 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROA		PATIENT ID REG. NO./LAB NO. REGISTRATION DATE COLLECTION DATE REPORTING DATE	: 1684599 <b>: 012411280027</b> : 28/Nov/2024 10:59 AM : 28/Nov/2024 12:40PM : 28/Nov/2024 01:26PM
Test Name		Value	Unit	<b>Biological Reference interval</b>
		VIT	AMINS	
	VI		DROXY VITAMIN D	3
by CLIA (CHEMILUMINE	DROXY VITAMIN D3): SERU SCENCE IMMUNOASSAY)	M <b>18.569<sup>L</sup></b>	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
<u>Nterpretation:</u> Defic	IENT:	< 20	n	g/mL
INSUFF		21 - 29		g/mL
PREFFERE INTOXIC		30 - 100 > 100		g/mLg/mL
2.25-OHVitamin D re- issue and tightly bou 3. Vitamin D plays a pr shosphate reabsorpti- 4. Severe deficiency m <b>DECREASED:</b> 1. Lack of sunshine ext 2. Inadequate intake, 1 3. Depressed Hepatic N 4. Secondary to advance 5. Enzyme Inducing dru <b>NCREASED:</b> 1. Hypervitaminosis D severe hypercalcemia <b>CAUTION:</b> Replacemen hypervitaminosis D	nd by a transport protein wh imary role in the maintenanc on, skeletal calcium depositio ay lead to failure to mineraliz oosure. malabsorption (celiac disease /itamin D 25- hydroxylase act ced Liver disease econdary Hyperparathroidism ugs: anti-epileptic drugs like p is Rare, and is seen only afte and hyperphophatemia. It therapy in deficient individ mdividuals as compare to white	voir and transport fo ile in circulation. e of calcium homeo n, calcium mobiliza e newly formed ost ivity (Mild to Moderate henytoin, phenobal r prolonged exposur uals must be monito	orm of Vitamin D and trans ostatis. It promotes calciun tion, mainly regulated by p eoid in bone, resulting in r deficiency) rbital and carbamazepine, re to extremely high doses ored by periodic assessmen	port form of Vitamin D, being stored in adipose 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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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS , MD (PATHOLOGY)

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