

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mrs. SHARDA			
AGE/ GENDER	: 32 YRS/FEMALE		PATIENT ID	: 1749641
COLLECTED BY	:		REG. NO./LAB NO.	: 012502080028
<b>REFERRED BY</b>	:		<b>REGISTRATION DATE</b>	: 08/Feb/2025 11:29 AM
BARCODE NO.	: 01525148		COLLECTION DATE	:08/Feb/202511:32AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 08/Feb/2025 12:35PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
			LLNESS PANEL: 1.2	2
		PLETE BL	OOD COUNT (CBC)	
	S (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H) by CALORIMETRIC	B)	13.6	gm/dL	12.0 - 16.0
RED BLOOD CELL (	RBC) COUNT	4.6	Millions/	cmm 3.50 - 5.00
PACKED CELL VOLU	JME (PCV) utomated hematology analyzer	41.1	%	37.0 - 50.0
MEAN CORPUSCUL		89.3	fL	80.0 - 100.0
MEAN CORPUSCUL	AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	29.5	pg	27.0 - 34.0
MEAN CORPUSCUL	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	33	g/dL	32.0 - 36.0
RED CELL DISTRIB	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	14	%	11.00 - 16.00
RED CELL DISTRIB	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	46.9	fL	35.0 - 56.0
MENTZERS INDEX		19.41	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING IND		27.12	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE		0000		1000 11000
TOTAL LEUCOCYTE by FLOW CYTOMETRY	COUNT (TLC) / by sf cube & microscopy	8960	/cmm	4000 - 11000
NUCLEATED RED B	LOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
NUCLEATED RED B	LOOD CELLS (nRBCS) % UTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			





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

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 0171-2643898, +91 99910 43898
 care@koshealthcare.com

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 www.koshealthcare.com



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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	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	64	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2.2	04	22.12
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	28	%	20 - 40
EOSINOPHILS	3	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			1.0
MONOCYTES	5	%	2 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 1
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	5734	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0.01	,	
ABSOLUTE LYMPHOCYTE COUNT	2509	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT	900	/	10 110
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	269	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT	448	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	144000 <sup>L</sup>	/cmm	150000 - 450000
PLATELETCRIT (PCT) by Hydro Dynamic Focusing, Electrical Impedence	0.22	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV)	15 <sup>H</sup>	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	86000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR)	60.1 <sup>H</sup>	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	10.4	0/	15.0 17.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence	16.4	%	15.0 - 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			



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

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

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	MD		opraDr. Yugam ChopraMicrobiology)MD (Pathology)sultant PathologistCEO & Consultant Pathologist		
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CLIENT CODE.	: KOS DIAGNOSTI	C LAB	R	EPORTING DATE	: 08/Feb/2025 12:58PM
LIENT ADDRESS	: 6349/1, NICHOI	SON ROAD, A	AMBALA CANTT		
Fest Name		_	Value	TT \$4	
NTERPRETATION: 1. ESR is a non-specif mmune disease, but 2. An ESR can be affe as C-reactive protein	GATION BY CAPILLAR) ic test because an e does not tell the he cted by other condit	TE (ESR) Y PHOTOMETR' levated result alth practition tions besides	OCYTE SEDIM 3 Y t often indicates th ner exactly where t inflammation. For	the inflammation is in the this reason, the ESR is ty	





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	AD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMISTRY	BIOCHEMISTR	Y
		GLUCOSE FAST	TING (F)	
GLUCOSE FASTING	F (F): PLASMA E - PEROXIDASE (GOD-POD)	113.08 <sup>H</sup>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

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.





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFI	F · BASIC	
CHOLESTEROL TOT				OPTIMAL: < 200.0
by CHOLESTEROL TO		122.96	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)	178.21 <sup>H</sup>	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
				VERY HIGH: > OR = 500.0
HDL CHOLESTEROI		43.13	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		44.19	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		79.83	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		35.64	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER by CALCULATED, SPE	UM	424.13	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPE	L RATIO: SERUM	2.85	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	Biological Reference interval	
LDL/HDL RATIO: S by Calculated, spe		1.02	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0	
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY		4.13	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)

:1749641

:012502080028

:08/Feb/2025 11:29 AM

:08/Feb/202511:32AM

:08/Feb/202502:25PM

**Biological Reference interval** 

Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** : Mrs. SHARDA AGE/ GENDER : 32 YRS/FEMALE **PATIENT ID COLLECTED BY** REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE BARCODE NO.** :01525148 **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 mg/dL INFANT: 0.20 - 8.00 1.54<sup>H</sup> by DIAZOTIZATION, SPECTROPHOTOMETRY ADULT: 0.00 - 1.20 0.00 - 0.40 BILIRUBIN DIRECT (CONJUGATED): SERUM 0.28 mg/dL by DIAZO MODIFIED, SPECTROPHOTOMETRY BILIRUBIN INDIRECT (UNCONJUGATED): SERUM 1.26<sup>H</sup> mg/dL 0.10 - 1.00 by CALCULATED, SPECTROPHOTOMETRY 34.27.00 - 45.00 SGOT/AST: SERUM U/L by IFCC, WITHOUT PYRIDOXAL PHOSPHATE SGPT/ALT: SERUM U/L 0.00 - 49.00 50.5<sup>H</sup> by IFCC, WITHOUT PYRIDOXAL PHOSPHATE AST/ALT RATIO: SERUM 0.68 RATIO 0.00 - 46.00 by CALCULATED, SPECTROPHOTOMETRY ALKALINE PHOSPHATASE: SERUM 107.01 U/L 40.0 - 130.0 by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM 39.61 U/L 0.00 - 55.0 by SZASZ, SPECTROPHTOMETRY TOTAL PROTEINS: SERUM 8.02<sup>H</sup> gm/dL 6.20 - 8.00 by BIURET, SPECTROPHOTOMETRY ALBUMIN: SERUM 4.43gm/dL 3.50 - 5.50 by BROMOCRESOL GREEN 2.30 - 3.50 **GLOBULIN: SERUM** gm/dL 3.59<sup>H</sup> by CALCULATED, SPECTROPHOTOMETRY A : G RATIO: SERUM 1.23 RATIO 1.00 - 2.00 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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NAME

Test Name





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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).

PROGNOSTIC SIGNIFICANCE:

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



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MBBS, MD (PATHOLOGY)

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	KIDN	EY FUNCTION 7	FEST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAM	IATE DEHYDROGENASE (GLDH)	15.75	mg/dL	10.00 - 50.00
CREATININE: SERU	JM	0.84	mg/dL	0.40 - 1.20
	OGEN (BUN): SERUM	7.36	mg/dL	7.0 - 25.0
BLOOD UREA NITE RATIO: SERUM by CALCULATED, SPE	COGEN (BUN)/CREATININE	8.76 <sup>L</sup>	RATIO	10.0 - 20.0
UREA/CREATININ	E RATIO: SERUM	18.75	RATIO	
URIC ACID: SERUM by URICASE - OXIDAS		2.82	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	10.01	mg/dL	8.50 - 10.60
-	RUM DATE, SPECTROPHOTOMETRY	2.92	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	E ELECTRODE)	141.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUE by ISE (ION SELECTIV	M	4.17	mmol/L	3.50 - 5.00
CHLORIDE: SERUM	ſ	106.13	mmol/L	90.0 - 110.0
	ERULAR FILTERATION RATE	94.6		

Dr. Vinay Chopra

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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Test Name		_	Value	Un	it	Biologic	al Reference inter
<ol> <li>Excess protein inta burns, surgery, cache</li> <li>Urine reabsorption</li> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>INCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> </ol>	kia, high fever (e.g. ureter co ass (subnorma tetracycline, <u>c</u> <b>D:1) WITH ELE</b> (BUN rises dis superimposec	blostomy) al creatinine productior llucocorticoids) <b>/ATED CREATININE LEVE</b> sproportionately more t on renal disease.	) LS:			hing's syndro	me, high protein die
5. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. <b>INCREASED RATIO (&gt;2</b> 1. Postrenal azotemia 2. Prerenal azotemia 2. Prerenal azotemia 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome c 3. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (ro 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in	se or producti kia, high fever (e.g. ureter co ass (subnorma tetracycline, co <b>D:1) WITH ELE</b> (BUN rises di superimposed <b>D:1) WITH DEC</b> osis. d starvation. creased ureas urea rather th nonemias (ur f inappropiate <b>D:1) WITH INC</b> oy (accelerate eleases musch who develop r sis (acetoacet treased BUN/ apy (interfere <b>LAR FILTERAT</b>	). blostomy) al creatinine production plucocorticoids) <b>VATED CREATININE LEVE</b> sproportionately more to on renal disease. <b>CREASED BUN :</b> creatinine diffuses of ea is virtually absent in e antidiuretic harmone) <b>REASED CREATININE:</b> s conversion of creating e creatinine). renal failure. ate causes false increas creatinine ratio). s with creatinine measu	) LS: han creatinine blood). due to tubular to creatinine e in creatinine rement).	e) (e.g. obstructive lular fluid). secretion of urea	e uropathy). hodologies,res ASSOCIATE	ulting in norn	
5. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. NCREASED RATIO (>2 9. Postrenal azotemia 9. Prerenal azotemia 9. Prerenal azotemia 9. CertaSED RATIO (<1 9. Acute tubular necr 9. Low protein diet ar 9. Severe liver disease 9. Other causes of de 10. Repeated dialysis ( 10. Inherited hyperam 11. SIADH (syndrome c 12. Pregnancy. 12. Phenacimide thera 13. Muscular patients 14. Phenacimide thera 15. Muscular patients 16. Muscular patients 17. Diabetic ketoacido 15. Diabetic ketoacido 16. Diabetic ketoacido 17. Cephalosporin ther 17. STAGE	e or producti kia, high fever (e.g. ureter co ass (subnorma tetracycline, g <b>D:1) WITH ELE</b> (BUN rises di- superimposed <b>D:1) WITH DEC</b> osis. d starvation. creased ureas urea rather th nonemias (ur f inappropiate <b>D:1) WITH INC</b> oy (accelerate eleases muscl- who develop n sis (acetoacet treased BUN/ apy (interfere LAR FILTERATI	). blostomy) al creatinine production plucocorticoids) <b>VATED CREATININE LEVE</b> sproportionately more to on renal disease. <b>CREASED BUN :</b> block and the second creatinine diffuses of the antidiuretic harmone) <b>REASED CREATININE:</b> s conversion of creatine the creatinine). renal failure. ate causes false increases creatinine ratio). s with creatinine measu <b>ON RATE:</b> <b>DESCRIPTION</b> ormal kidney function Kidney damage with	) LS: han creatinine blood). due to tubular to creatinine e in creatinine rement).	e) (e.g. obstructive lular fluid). r secretion of urea ). e with certain met /min/1.73m2 )	e uropathy). hodologies,res <u>ASSOCIATE</u> <u>No pro</u> Presence o	ulting in norn D FINDINGS teinuria of Protein ,	
5. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. NCREASED RATIO (>2 9. Postrenal azotemia 0. Postrenal azotemia 0. Prerenal azotemia 0. Acute tubular necr 9. Low protein diet ar 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 c 6. Pregnancy. 0. Pregnancy. 0. Pregnancy. 0. Phenacimide thera 2. Rhabdomyolysis (r 6. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther 1. STATED GLOMERL 0. CKD STAGE 1. G1 1. G2	e or producti kia, high fever (e.g. ureter co ass (subnorma tetracycline, co <b>D:1) WITH ELE</b> (BUN rises di- superimposed <b>D:1) WITH DEC</b> osis. d starvation. creased ureas urea rather th nonemias (ur f inappropiate <b>D:1) WITH INC</b> oy (accelerate eleases muscl- who develop r sis (acetoacet treased BUN/ apy (interfere LAR FILTERATI	). blostomy) al creatinine production plucocorticoids) <b>VATED CREATININE LEVE</b> sproportionately more to on renal disease. <b>CREASED BUN :</b> block and creatinine diffuses of the antidiuretic harmone) <b>REASED CREATININE:</b> s conversion of creatine antidiuretic harmone) <b>REASED CREATININE:</b> s conversion of creatine the creatinine). renal failure. ate causes false increases creatinine ratio). s with creatinine measu <b>ON RATE:</b> <b>DESCRIPTION</b> ormal kidney function Kidney damage with normal or high GFR.	) LS: han creatinine ut of extracel blood). due to tubular to creatinine e in creatinine rement). GFR ( mL	e) (e.g. obstructive lular fluid). r secretion of urea ). e with certain met / <u>min/1.73m2 ) &gt;90 &gt;90</u>	e uropathy). hodologies,res <u>ASSOCIATE</u> No pro	ulting in norn D FINDINGS teinuria of Protein ,	
5. Excess protein inta purns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. NCREASED RATIO (>2 9. Postrenal azotemia 0. Prerenal azotemia 0. Certased RATIO (<1 9. Acute tubular necr 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 c 8. Pregnancy. 0. Pregnancy. 0. Pregnancy. 0. Phenacimide thera 1. Phenacimide thera 1. Phenacimide thera 1. Diabetic ketoacido 1. Cephalosporin ther 1. STIMATED GLOMERL 0. CKD STAGE 0. G1 0. G2 0. G3a	e or producti kia, high fever (e.g. ureter co ass (subnorma tetracycline, g <b>D:1) WITH ELE</b> (BUN rises di- superimposed <b>D:1) WITH DEC</b> osis. d starvation. creased ureas urea rather th nonemias (ur f inappropiate <b>D:1) WITH INC</b> oy (accelerate eleases muscl- who develop n sis (acetoacet treased BUN/ apy (interfere LAR FILTERATI	). blostomy) al creatinine production plucocorticoids) <b>VATED CREATININE LEVE</b> sproportionately more to on renal disease. <b>CREASED BUN :</b> synthesis. an creatinine diffuses of the artidiuretic harmone) <b>REASED CREATININE:</b> s conversion of creatine the creatinine net increase creatinine ratio). s with creatinine measu <b>ON RATE:</b> <b>DESCRIPTION</b> ormal kidney function Kidney damage with normal or high GFR Mild decrease in GFR	) LS: han creatinine ut of extracel blood). due to tubular to creatinine e in creatinine rement). GFR ( mL	e) (e.g. obstructive lular fluid). r secretion of urea ). e with certain met /min/1.73m2) >90 >90	e uropathy). hodologies,res <u>ASSOCIATE</u> <u>No pro</u> Presence o	ulting in norn D FINDINGS teinuria of Protein ,	
5. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. <b>INCREASED RATIO (&gt;2</b> 1. Postrenal azotemia 2. Prerenal azotemia <b>DECREASED RATIO (&lt;1</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 c 6. Pregnancy. <b>DECREASED RATIO (&lt;1</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>G1</b> <b>G2</b>	se or producti kia, high fever (e.g. ureter co ass (subnorma tetracycline, g <b>D:1) WITH ELE</b> (BUN rises di- superimposed <b>D:1) WITH DEC</b> osis. d starvation. creased ureas urea rather th nonemias (ur f inappropiate <b>D:1) WITH INC</b> oy (accelerate eleases muscl- who develop n sis (acetoacet treased BUN/ apy (interfere LAR FILTERATI	). blostomy) al creatinine production plucocorticoids) <b>VATED CREATININE LEVE</b> sproportionately more to on renal disease. <b>CREASED BUN :</b> block and creatinine diffuses of the antidiuretic harmone) <b>REASED CREATININE:</b> s conversion of creatine antidiuretic harmone) <b>REASED CREATININE:</b> s conversion of creatine the creatinine). renal failure. ate causes false increases creatinine ratio). s with creatinine measu <b>ON RATE:</b> <b>DESCRIPTION</b> ormal kidney function Kidney damage with normal or high GFR.	) LS: han creatinine blood). due to tubular to creatinine e in creatinine rement). GFR ( mL	e) (e.g. obstructive lular fluid). r secretion of urea ). e with certain met / <u>min/1.73m2 ) &gt;90 &gt;90</u>	e uropathy). hodologies,res <u>ASSOCIATE</u> <u>No pro</u> Presence o	ulting in norn D FINDINGS teinuria of Protein ,	





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

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







	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologi		(Pathology)
NAME	: Mrs. SHARDA		
AGE/ GENDER	: 32 YRS/FEMALE	PATIENT ID	: 1749641
<b>COLLECTED BY</b>	:	REG. NO./LAB NO.	: 012502080028
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 08/Feb/2025 11:29 AM
BARCODE NO.	: 01525148	<b>COLLECTION DATE</b>	:08/Feb/202511:32AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	:08/Feb/202501:18PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Г	
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



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

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt - 133 001, Haryana 0171-2643898, +91 99910 43898 care@koshealthcare.com www.koshealthcare.com







	М	r. Vinay Chopra D (Pathology & Micro nairman & Consultan	obiology)		gam Chopr MD (Patholog ultant Pathologi	у)	
NAME	: Mrs. SHARDA						
AGE/ GENDER	: 32 YRS/FEMAL	Æ		PATIENT ID	: 1749	641	
COLLECTED BY	:			REG. NO./LAB NO.	:0125	502080028	
REFERRED BY	:			<b>REGISTRATION DAT</b>	<b>FE</b> : 08/Fe	eb/2025 11:29 AM	
BARCODE NO.	:01525148			COLLECTION DATE	:08/Fe	eb/2025 11:32AM	
CLIENT CODE.	: KOS DIAGNOS	FIC LAB		<b>REPORTING DATE</b>	:08/Fe	eb/202501:31PM	
CLIENT ADDRESS	: 6349/1, NICHO	DLSON ROAD, AMBA	ALA CANTI	ſ			
Test Name			Value	Unit		Biological Referen	nce interval
			0.412	CTION TEST: TOT ng/1		0.35 - 1.93	
by CMIA (CHEMILUMIN THYROXINE (T4): S	iescent micropar SERUM		8.02	μgm		4.87 - 12.60	
by CMIA (CHEMILUMIN THYROID STIMULA by CMIA (CHEMILUMIN 3rd GENERATION, ULT INTERPRETATION:	TING HORMONE	C (TSH): SERUM	6.065 <sup>H</sup>	μIU,	/mL	0.35 - 5.50	
TSH levels are subject to a day has influence on the l	<i>measured serum TSH c</i> lure at any level of reg	<i>oncentrations</i> . TSH stim gulation of the hypothal	ulates the p	nd at a minimum between a oduction and secretion of ry-thyroid axis will result in	the metabolically	active hormones, thyroxir	ne (T4)and
CLINICAL CONDITION		T3		T4	TSI	-	
Primary Hypothyroidis		Reduced		Reduced	Increased (S	a .	
Subclinical Hypothyroi		Normal or Low Norma	1	Normal or Low Normal	Hiç		
Primary Hyperthyroidis		Increased Normal or High Norma		Increased Normal or High Normal		times undetectable)	
Subclinical Hyperthyro							

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.

TRIIODOTHYRONINE (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	





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

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





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	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Patholog		(Pathology)
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AGE/ GENDER	: 32 YRS/FEMALE	PATIENT ID	: 1749641
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Т	

Test Name			Value	Unit	t	<b>Biological Reference interval</b>
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	VIMENDATIONS 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





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





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	<b>Dr. Vinay Ch</b> MD (Pathology & Chairman & Con		Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. SHARDA			
AGE/ GENDER	: 32 YRS/FEMALE	PA	TIENT ID	: 1749641
COLLECTED BY	:	RE	G. NO./LAB NO.	: 012502080028
<b>REFERRED BY</b>	:	RE	GISTRATION DATE	: 08/Feb/2025 11:29 AM
BARCODE NO.	: 01525148	CO	LLECTION DATE	:08/Feb/202511:32AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		PORTING DATE	: 08/Feb/2025 12:25PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		CLINICAL PA	THOLOGY	
	URINE RO		SCOPIC EXAMINA	ATION
PHYSICAL EXAMI	NATION			
QUANTITY RECIEV	ED STANCE SPECTROPHOTOMETRY	10	ml	
COLOUR		PALE YELLO	W	PALE YELLOW
TRANSPARANCY	TANCE SPECTROPHOTOMETRY	HAZY		CLEAR
SPECIFIC GRAVITY		1.02		1.002 - 1.030
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
REACTION		ACIDIC		
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	-		
SUGAR by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
pH	TANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5
BILIRUBIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
NITRITE		Negative		NEGATIVE (-ve)
UROBILINOGEN	TANCE SPECTROPHOTOMETRY.	Normal	EU/dL	0.2 - 1.0
KETONE BODIES		Negative		NEGATIVE (-ve)
BLOOD	TANCE SPECTROPHOTOMETRY	TRACE		NEGATIVE (-ve)
ASCORBIC ACID by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	NEGATIVE (-	ve)	NEGATIVE (-ve)
MICROSCOPIC EX			/////	
RED BLOOD CELLS	(KBUS)	3-4	/HPF	0 - 3

57  $\infty$ n



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

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KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana 0171-2643898, +91 99910 43898 | care@koshealthcare.com | www.koshealthcare.com





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

NAME	: Mrs. SHARDA				
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Test Name		Value	Unit	Biological Reference interval	
	CENTRIFUGED URINARY SEDIMENT	· · · · · · · · · · · · · · · · · · ·	Child		
PUS CELLS	CENTRIFUGED URINARY SEDIMENT	1-2	/HPF	0 - 5	
EPITHELIAL CELL by MICROSCOPY ON	S CENTRIFUGED URINARY SEDIMENT	0-3	/HPF	ABSENT	
CRYSTALS		NEGATIVE (-ve)		NEGATIVE (-ve)	

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	NEGATIVE (-ve)
BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	NEGATIVE (-ve)
OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT	ABSENT

\*\*\* End Of Report \*\*



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

MBBS, MD (PATHOLOGY)

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