



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultan	obiology)		(Pathology)
NAME	: Mrs. SHANTA NARANG			
AGE/ GENDER	: 84 YRS/FEMALE		PATIENT ID	: 1709130
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	:012412260022
REFERRED BY	:		REGISTRATION DATE	: 26/Dec/2024 10:50 AM
	: 01523029		COLLECTION DATE	: 26/Dec/2024 11:07AM
	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMB/		REPORTING DATE	: 26/Dec/2024 11:18AM
Test Name		Value	Unit	<b>Biological Reference interval</b>
			LLNESS PANEL: 1.( )OD COUNT (CBC)	
RED BLOOD CELLS (	(RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB)		11.9 <sup>L</sup>	gm/dL	12.0 - 16.0
by CALORIMETRIC RED BLOOD CELL (R		4.56	Millions/	′cmm 3.50 - 5.00
PACKED CELL VOLUN	CUSING, ELECTRICAL IMPEDENCE ME (PCV) TOMATED HEMATOLOGY ANALYZER	38.7	%	37.0 - 50.0
MEAN CORPUSCULAI		84.9	fL	80.0 - 100.0
	R HAEMOGLOBIN (MCH) TOMATED HEMATOLOGY ANALYZER	26 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCULA	R HEMOGLOBIN CONC. (MCHC) TOMATED HEMATOLOGY ANALYZER	30.6 <sup>L</sup>	g/dL	32.0 - 36.0
	TION WIDTH (RDW-CV) TOMATED HEMATOLOGY ANALYZER	14.1	%	11.00 - 16.00
RED CELL DISTRIBU	TION WIDTH (RDW-SD) TOMATED HEMATOLOGY ANALYZER	44.8	fL	35.0 - 56.0
MENTZERS INDEX		18.62	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDE by calculated	X	26.15	RATIO	BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELI		0100		1000 11000
TOTAL LEUCOCYTE (		6180	/cmm	4000 - 11000
TOTAL LEUCOCYTE ( by flow cytometry e NUCLEATED RED BL	COUNT (TLC) BY SF CUBE & MICROSCOPY	6180 NIL	/cmm	4000 - 11000 0.00 - 20.00

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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Yugam Chopra

**CEO & Consultant Pathologist** 

MD (Pathology)

NAME : Mrs. SHANTA NARANG **AGE/ GENDER** : 84 YRS/FEMALE **PATIENT ID** :1709130 **COLLECTED BY** : SURJESH :012412260022 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 26/Dec/2024 10:50 AM : **BARCODE NO.** :01523029 **COLLECTION DATE** : 26/Dec/2024 11:07AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 26/Dec/2024 11:18AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC) NEUTROPHILS** 62 % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 26% 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 4 % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 8 % 2 - 12by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE LEUKOCYTES (WBC) COUNT** ABSOLUTE NEUTROPHIL COUNT 3832 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 1607 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 247/cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 494 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 355000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.34 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 10 fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 30000 - 90000 PLATELET LARGE CELL COUNT (P-LCC) 83000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE % PLATELET LARGE CELL RATIO (P-LCR) 23.511.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 15.0 - 17.0 16.2% by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

Dr. Vinay Chopra

MD (Pathology & Microbiology)

Chairman & Consultant Pathologist

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CAN	NTT	
The set NI server	V-L	TL.**	
Test Name	Value	Unit	<b>Biological Reference interval</b>





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

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





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



AME GE/ GENDER OLLECTED BY EFERRED BY	<b>: Mrs. SHANTA NARANG</b> : 84 YRS/FEMALE			Pathologist
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ARCODE NO.	: 01523029	C	OLLECTION DATE	: 26/Dec/2024 11:07AM
LIENT CODE.	: KOS DIAGNOSTIC LAB	F	EPORTING DATE	: 26/Dec/2024 12:04PM
IENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT		
est Name		Value	Unit	<b>Biological Reference interval</b>
stemic lupus erythe DNDITION WITH LOV low ESR can be seen olycythaemia), sign sickle cells in sickle DTE: ESR and C - reactive Generally, ESR doe CRP is not affected	ematosus <b>N ESR</b> 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 o s not change as rapidly as does CRI <b>by as many other factors as is ESR</b> , ed, it is typically a result of two typ	ormal sedimenta nt (leucocytosis) f inflammation. P, either at the s <b>making it a bette</b> es of proteins, g	ation of red blood cells, si , and some protein abno art of inflammation or as r marker of inflammation obulins or fibrinogen. an cause temporary eleva	rmalities. Šome changes in red cell shape (suc s it resolves.





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		ology & Microbiology) & Consultant Pathologist	MD (P CEO & Consultant P	Pathology) athologist	
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CLIENT CODE.			ORTING DATE		
CLIENT ADDRESS	: 6349/1, NICHOLSON	ROAD, AMBALA CANTT			
Test Name		Value	Unit	<b>Biological Reference interval</b>	
	C	LINICAL CHEMISTRY	//BIOCHEMISTR	2Y	
		GLUCOSE FAS	TING (F)		
GLUCOSE FASTING	G (F): PLASMA e - peroxidase (god-pod)	83.6	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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		Chopra & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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Test Name		Value	Unit	<b>Biological Reference interval</b>
			FILE : BASIC	
CHOLESTEROL TO	TAL · SERIM	113.28	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		113.20	nig/ uL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSP	ERUM PHATE OXIDASE (ENZYMATIC)	93.79	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
		07.01	. / 11	VERY HIGH: $> OR = 500.0$
HDL CHOLESTERO	L (DIRECT): SERUM	37.61	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		56.91	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0
NON HDL CHOLEST by calculated, spe		75.67	mg/dL	VERY HIGH: > OR = 190.0 OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0
VLDL CHOLESTER		18.76	mg/dL	VERY HIGH: > OR = 220.0 0.00 - 45.00
by CALCULATED, SPE TOTAL LIPIDS: SER by CALCULATED, SPE	RUM	320.35 <sup>L</sup>	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPE	DL RATIO: SERUM	3.01	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		1.51	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 ECTROPHOTOMETRY	2.49 <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	Biological Reference interval
	LIVER	FUNCTION	N TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI	: SERUM PECTROPHOTOMETRY	0.7	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.22	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE	CT (UNCONJUGATED): SERUM	0.48	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	31.3	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	19.8	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE		1.58	RATIO	0.00 - 46.00
ALKALINE PHOSPI by PARA NITROPHEN PROPANOL	HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL	62.3	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTROF	L TRANSFERASE (GGT): SERUM	19.09	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.2	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		4.64	gm/dL	3.50 - 5.50
GLOBULIN: SERUM	1	1.56 <sup>L</sup>	gm/dL	2.30 - 3.50
by CALCULATED, SPE A : G RATIO: SERUI	M	2.97 <sup>H</sup>	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:**

> 2
> 2 (Highly Suggestive)
1.4 - 2.0
> 1.5
> 1.3 (Slightly Increased)



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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).

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



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







0 9001.2000 CENT				
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Test Name		Value	Unit	Biological Reference interva
	KIDN	EY FUNCTION T	EST (COMPLETE)	
UREA: SERUM		34.96	mg/dL	10.00 - 50.00
by UREASE - GLUTAN CREATININE: SER	/ATE DEHYDROGENASE (GLDH)	0.91	mg/dL	0.40 - 1.20
by ENZYMATIC, SPEC		0.91	iiig/ uL	0.40 - 1.20
	ROGEN (BUN): SERUM	16.34	mg/dL	7.0 - 25.0
by CALCULATED, SPECTROPHOTOMETRY BLOOD UREA NITROGEN (BUN)/CREATININE		17.96	RATIO	10.0 - 20.0
RATIO: SERUM				
by CALCULATED, SPI UREA/CREATININ	ECTROPHOTOMETRY	38.42	RATIO	
	ECTROPHOTOMETRY	30.42	KATIO	
URIC ACID: SERUM		8.16 <sup>H</sup>	mg/dL	2.50 - 6.80
by URICASE - OXIDAS CALCIUM: SERUM	SE PEROXIDASE	10.34	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE			Ū.	
PHOSPHOROUS: SI	ERUM DATE, SPECTROPHOTOMETRY	2.65	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM		139.7	mmol/L	135.0 - 150.0
by ISE (ION SELECTIN		4.9		250 500
POTASSIUM: SERU by ISE (ION SELECTIN		4.3	mmol/L	3.50 - 5.00
CHLORIDE: SERUM		104.78	mmol/L	90.0 - 110.0
by ISE (ION SELECTIN FSTIMATED CI ON	/E ELECTRODE) MERULAR FILTERATION RATE			
	IERULAR FILTERATION RATE	62.2		
(eGFR): SERUM	ILIVOLAN FILTENATION NATE	04.4		
by CALCULATED				
<u>INTERPRETATION:</u>	con pro, and post ropal azatamia			

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 PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





ACC / GENDER       :84 YRS/FEMALE       PATIENT ID       :1709130         XXDLECTED BY       :SURJESH       REG. NO./LAB NO.       :012412260022         REFERED BY       :       :26/Dec/2024 10:50 AM         AARCODE NO.       :01523029       COLLECTION DATE       :26/Dec/2024 11:07AM         XLENT CODE       :KOS DIAGNOSTIC LAB       REPORTING DATE       :26/Dec/2024 01:15PM         XLENT ADDRESS       :6349/1, NICHOLSON ROAD, AMBALA CANTT       :26/Dec/2024 01:15PM         Fest Name       Value       Unit       Biological Reference interemention         1. High protein intake.       :Impaired renal function plus       :       :Kress protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein die purns, surgery, cacheakia, high fever).       :       !/// intereabsorption (e.g., ureter colostomy)         2. Reduced muscle mass (subnormal creatinine production)       :		Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist			Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist				
COLLECTED BY       SURJESH       REG. NO./LAB NO.       : 012412260022         REFERED BY       :       REGISTRATION DATE       : 26/Dec/2024 10:50 AM         BARCODE NO.       : 01523029       COLLECTION DATE       : 26/Dec/2024 10:50 AM         SLIENT CODE       : KOS DIAGNOSTIC LAB       REPORTING DATE       : 26/Dec/2024 01:15PM         CLIENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTT       Biological Reference interv         1. High portein intake.       :       :       :       :         1. Impaired renal function plus       :       :       :       :         2. Urber cabsorption (e.g. ureter colostomy)       :       :       :       :         3. Medued muscle mass (subnormal creatinine production)       :       :       :       :         0. Certain drugs (e.g. tetrazycline glucocortinolately more than creatinine) (e.g. obstructive uropathy).       :       :       :         1. Postrenal azotemia (SUP thromostinately more than creatinine) (e.g. obstructive uropathy).       :       :       :         2. Ordernal azotemia (Superoprotinolately more than creatinine) (e.g. obstructive uropathy).       :       :       :         3. Pregnary.       :       :       :       :       :       :         0. Ceruston SUG (cortinoste)       :	NAME	: Mrs. SHANT	A NARANG						
REFERED BY       ::       REGISTRATION DATE       ::	AGE/ GENDER	: 84 YRS/FEM	ALE	Р	ATIENT ID	: 17	09130		
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BARCODE NO.       10.1523029       COLLECTION DATE       26./Dec/2024 11:07AM         SLIENT CODE       KOS DIAGNOSTIC LAB       REPORTING DATE       26./Dec/2024 01:15PM         SLIENT ADDRESS       6349/1, NICHOLSON ROAD, AMBALA CANTT       Image: Collega C									
Element CODE       KENS DIAGNOSTIC LAB       REPORTING DATE       : 26/Dec/2024 01:15PM         SILENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTE         Test Name       Value       Unit       Biological Reference internet         1. High protein intake.       :									
CLENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTT         Test Name       Value       Unit       Biological Reference internerner         1. High protein intake.       Impaired renal function plus         3. Excess protein intake or production or tissue breakdown (e.g. infection, Gi bleeding, thyrotoxicosis, Cushing's syndrome, high protein die borns, surgery, cachexia, high fever).       Impaired renal function plus         1. Urine readsorption (e.g. ureter colostomy)       Reduced muscle mass (subnormal creatinine production).         2. Ortical cargotilen, affuccorricolds)       NOREASED RATIO (>02-01) WITH ELEVATED CREATININE LEVELS:         1. Ortical azotemia (BUH reset disproprotionately more than creatinine) (e.g. obstructive uropathy).         2. Prerenal azotemia superimposed on renal disease.         PECKASED RATIO (>0-01) WITH DECREASED BUN :         3. Aduet tubular necrosis.         9. Severe liver disease.         9. Ow protein diet and starvation.         9. Severe liver disease.         9. Other causes of decreased urea synthesis.         9. Other causes of decreased urea synthesis.         9. Shored King disease muscle creatinne).         9. Shored liver disease.         9. Other causes of decreased urea synthesis.         9. Other causes of decreases duver asynthesis.         9. Aduet tubular hercrosis.         9. Matore disease.         9. Other									
Test Name       Value       Unit       Biological Reference intervent         4. High protein intake.       5. Impaired renal function plus       5. Excess protein intake or production or tissue breakdown (e.g. infection, Gl bleeding, thyrotoxicosis, Cushing's syndrome, high protein die burns, surgery, cacheka, high fever).       7. Urine reabsorption (e.g. ureter colostomy)         8. Reduced muscle mass (subnormal creatinine production)       9. Certain drugs (e.g. tetracycline, glucocorticoids)         NRCREASED RATIO (>201) WITH ELEVATED CREATINNE LEVELS:       1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         3. Porter liver disease.       0. Other causes of decreased urea synthesis.       5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).         6. Inherited hyperammonemias (urea is virtually absent in blood).       7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.         8. Pregnancy.       9. Pregnancy.       9. Pregnancy.         DECKEASED RATIO (<10.1) WITH INCREASED CREATINNE:					EPORTING DATE	20	5/Dec/2024 0.	1:15PM	
4. High protein intake.         5. Impaired renal function plus         6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein die burns, surgery, cachexia, high fever).         7. Urine reabsorption (e.g. ureter colostomy)         8. Reduced muscle mass (subnormal creatinine production)         9. Certain drugs (e.g. tetracycline, glucocorticoids)         INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:         1. Postrenal azotemia (Blur rises disproportionately more than creatinine) (e.g. obstructive uropathy).         2. Prerenal azotemia superimposed on renal disease.         DECREASED RATIO (>10:1) WITH DECREASED BUN :         1. Acute tubular necrosis.         2. Low protein diet and starvation.         3. Severe liver disease.         4. Other causes of decreased urea synthesis.         5. Inherited hyperammonemias (urea is virtually absent in blood).         7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.         8. Pregnancy.         DECREASED RATIO (<10:1) WITH INCREASED CREATININE:	LLIENT ADDRESS	: 6349/1, NIC	HOLSON ROAD, AMB.	ALA CANTI					
5. Impaired renal function plus 6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein die 2. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein die 2. Urine reabsorption (e.g. ureter colostomy) 7. Urine reabsorption (e.g. ureter colostomy) 7. Certain drugs (e.g. lettracycline, glucocorticoids) 7. Orine reabsorption (e.g. ureter colostomy) 7. Orien reabsorption (e.g. ureter colostomy) 7. Orien reabsorption (e.g. ureter colostomy) 7. Orien at azotemia (BUN rises disproprionately more than creatinine) (e.g. obstructive uropathy). 7. Percenal azotemia Superimposed on renal disease. 7. Orien at azotemia superimposed on renal disease. 7. Acute tubular necrosis. 7. Low protein diet and starvation. 7. Severe liver disease. 7. Orien at astrvation. 7. SiADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea. 7. SiADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea. 7. Penacimide therapy (accelerates conversion of creatine to creatinine). 7. Auster tubular patients who develop renal failure. 7. Nappropriate RATIO 7. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehyd should produce an increased BUN/creatinine ratio). 7. CKO STAGE 7. Discussed Conversion Articles 7. Oriental kidney function 7. Signal patients who develop renal failure: 7. CKO STAGE 7. Discussed cause false increase in creatinine with certain methodologies, resulting in normal ratio when dehyd should produce an increased BUN/creatinine ratio). 7. Construction appropriate antidiume ratio. 7. CKO STAGE 7. Discussed cause false increase in GFR 7. Oriental Albumin or cast in urine 7. CKO STAGE 7. D	Fest Name			Value	Un	it	Biologi	ical Reference	ce interva
normal or high GFRAlbumin or cast in urineG3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59	9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< <sup>-</sup> 1. Acute tubular necr	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed c 0:1) WITH DECR osis.	icocorticoids) <b>TED CREATININE LEVI</b> roportionately more to in renal disease.	LS:	e) (e.g. obstructive	e uropathy).			
normal or high GFRAlbumin or cast in urineG3aMild decrease in GFR60 - 89G3bModerate decrease in GFR30-59	<ol> <li>Certain drugs (e.g.,</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;'</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin there</li> <li>ESTIMATED GLOMERI</li> <li>CKD STAGE</li> </ol>	tetracycline, glu <b>0:1) WITH ELEV/</b> (BUN rises disp superimposed of <b>0:1) WITH DECR</b> osis. ad starvation. e. creased urea sy urea rather than monemias (urea of inappropiate a <b>10:1) WITH INCR</b> py (accelerates eleases muscle who develop re : sis (acetoacetat creased BUN/cr apy (interferes <b>JLAR FILTERATIO</b>	acocorticoids) <b>ATED CREATININE LEVI</b> roportionately more to in renal disease. <b>EASED BUN :</b> A creatinine diffuses of its virtually absent in antidiuretic harmone) <b>EASED CREATININE:</b> conversion of creatine creatinine). hal failure. the causes false increase eatinine ratio). with creatinine measu <b>N RATE:</b> <b>DESCRIPTION</b>	ELS: han creatinine but of extracel blood). due to tubular e to creatinine e in creatinine rement).	Ilular fluid). r secretion of urea e). e with certain met /min/1.73m2 )	hodologies,re			en dehydra
G3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59	<ol> <li>Certain drugs (e.g.,</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Perenal azotemia</li> <li>CECREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet and</li> <li>Severe liver diseas</li> <li>Other causes of degination of the second dialysis of the second</li></ol>	tetracycline, glu <b>0:1) WITH ELEV/</b> (BUN rises disp superimposed of <b>10:1) WITH DECR</b> osis. ad starvation. creased urea sy urea rather than monemias (urea of inappropiate a <b>10:1) WITH INCRI</b> py (accelerates eleases muscle who develop re : sis (acetoacetat creased BUN/cr apy (interferes <b>JLAR FILTERATIO</b> Nor	Accorticoids) ATED CREATININE LEVI roportionately more to in renal disease. EASED BUN : Acceatinine diffuses of is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). hal failure. the causes false increase eatinine ratio). with creatinine measu N RATE: DESCRIPTION mal kidney function	ELS: han creatinine but of extracel blood). due to tubular e to creatinine e in creatinine rement).	Ilular fluid). r secretion of urea e). e with certain met /min/1.73m2 ) >90	hodologies,re ASSOCIA No pi	TED FINDINGS		en dehydra
	<ol> <li>Certain drugs (e.g., NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>PecREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet and</li> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>SIADH (syndrome of Barden and the second Construction of the second Barden and the second and the second Barden and the second and the second Barden and the second and the seco</li></ol>	tetracycline, glu <b>0:1) WITH ELEV/</b> (BUN rises disp superimposed of <b>0:1) WITH DECR</b> osis. ad starvation. creased urea sy urea rather than monemias (urea of inappropiate a <b>10:1) WITH INCRI</b> py (accelerates eleases muscle who develop re : sis (acetoacetat creased BUN/cr apy (interferes <b>JLAR FILTERATIO</b> Nor Ki	Accorticoids) ATED CREATININE LEVI roportionately more to in renal disease. EASED BUN : Acceatinine diffuses of its virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). hal failure. the causes false increase eatinine ratio). with creatinine measu N RATE: DESCRIPTION mal kidney function dney damage with	ELS: han creatinine but of extracel blood). due to tubular e to creatinine e in creatinine rement).	Ilular fluid). r secretion of urea e). e with certain met /min/1.73m2 ) >90	hodologies,re ASSOCIA No pr Presence	TED FINDINGS Toteinuria		en dehydra
G4 Severe decrease in GER 15-29	<ul> <li>P. Certain drugs (e.g.,</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;'</li> <li>Acute tubular necr</li> <li>Low protein diet and</li> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis (r</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;'</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>cephalosporin ther</li> <li>STIMATED GLOMERI</li> <li>G1</li> <li>G2</li> </ul>	tetracycline, glu 0:1) WITH ELEV/ 0 (BUN rises disp superimposed of 0:1) WITH DECR 0:1) WITH DECR 0:1) WITH DECR 0:10 WITH INCR 10:1)	Accordition of the second seco	ES: han creatinine but of extracel blood). due to tubular e to creatinine e in creatinine rement).	Ilular fluid). r secretion of urea e). e with certain met /min/1.73m2) >90 >90	hodologies,re ASSOCIA No pr Presence	TED FINDINGS Toteinuria		en dehydra
G5 Kidney failure <15	<ul> <li>P. Certain drugs (e.g.,</li> <li>INCREASED RATIO (&gt;2</li> <li>1. Postrenal azotemia</li> <li>2. Prerenal azotemia</li> <li>DECREASED RATIO (&lt;'</li> <li>1. Acute tubular necr</li> <li>2. Low protein diet an</li> <li>3. Severe liver diseas</li> <li>4. Other causes of de</li> <li>5. Repeated dialysis (</li> <li>6. Inherited hyperam</li> <li>7. SIADH (syndrome of</li> <li>8. Pregnancy.</li> <li>DECREASED RATIO (&lt;'</li> <li>1. Phenacimide thera</li> <li>2. Rhabdomyolysis (r</li> <li>3. Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>1. Diabetic ketoacido</li> <li>should produce an in</li> <li>2. Cephalosporin there</li> <li>ESTIMATED GLOMERI</li> <li>G1</li> <li>G2</li> </ul>	tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. ad starvation. creased urea sy urea rather that monemias (urea of inappropiate a 10:1) WITH INCRI py (accelerates eleases muscle who develop re : sis (acetoacetat creased BUN/cr apy (interferes y <u>LAR FILTERATIO</u> Nor Ki     	According of the second	but of extracel blood). due to tubular e to creatinine e in creatinine <b>e in creatinine GFR ( mL</b>	Ilular fluid). r secretion of urea e). e with certain met <u>/min/1.73m2 )</u> >90 >90 60 -89 30-59	hodologies,re ASSOCIA No pr Presence	TED FINDINGS Toteinuria		en dehydra





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

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







: 6349/1, NICHOLSON ROAD,	AMBALA CANTI		
COAO /1 NICHOLCON DOAD	AMDALA CANTT		
: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 26/Dec/2024 01:15PM
: 01523029	COLL	ECTION DATE	: 26/Dec/2024 11:07AM
:	REGI	STRATION DATE	: 26/Dec/2024 10:50 AM
: SURJESH	REG.	NO./LAB NO.	: 012412260022
: 84 YRS/FEMALE	PATI	ENT ID	: 1709130
: Mrs. SHANTA NARANG			
		CEO & Consultant	
			(Pathology)
	MD (Pathology & Chairman & Cor : Mrs. SHANTA NARANG : 84 YRS/FEMALE : SURJESH : : 01523029 : KOS DIAGNOSTIC LAB	: 84 YRS/FEMALE PATE : SURJESH REG. : 01523029 COLL	MD (Pathology & Microbiology) Chairman & Consultant Pathologist       MD CEO & Consultant         : Mrs. SHANTA NARANG       :         : 84 YRS/FEMALE       PATIENT ID         : SURJESH       REG. NO./LAB NO.         :       .         : 01523029       COLLECTION DATE         : KOS DIAGNOSTIC LAB       REPORTING DATE

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)







	<b>Dr. Vinay Cho</b> MD (Pathology & Chairman & Cons	Microbiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. SHANTA NARANG			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		CLINICAL PA	THOLOGY	
	URINE ROI	UTINE & MICRO	SCOPIC EXAMINA	ATION
PHYSICAL EXAMIN	ATION			
QUANTITY RECIEV		10	ml	
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	AMBER YELL	ow	PALE YELLOW
-	TANCE SPECTROPHOTOMETRY			
TRANSPARANCY by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	HAZY		CLEAR
SPECIFIC GRAVITY	TANCE SPECTROPHOTOMETRY	1.01		1.002 - 1.030
CHEMICAL EXAMI				
REACTION		ACIDIC		
by DIP STICK/REFLEC PROTEIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
SUGAR	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
pH		<=5.0		5.0 - 7.5
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
NITRITE by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN	TANCE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0
KETONE BODIES	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
BLOOD		Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	NEGATIVE (-v		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY	MEGATIVE (-)		NEGATIVE (-Ve)
MICROSCOPIC EXA				
RED BLOOD CELLS	(RBCs)	NEGATIVE (-v	ve) /HPF	0 - 3





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





NANGE



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

CITANTA NADANO



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

NAME	: Mrs. SHANTA NARANG			
AGE/ GENDER	: 84 YRS/FEMALE	P	ATIENT ID	: 1709130
COLLECTED BY	: SURJESH	F	REG. NO./LAB NO.	: 012412260022
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
by MICROSCOPY ON	CENTRIFUGED URINARY SEDIMENT			
PUS CELLS		2-3	/HPF	0 - 5

EPITHELIAL CELLS       5-7       /HPF       ABSENT         by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT       NEGATIVE (-ve)       NEGATIVE (-ve)         by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT       NEGATIVE (-ve)       NEGATIVE (-ve)         by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT       NEGATIVE (-ve)       NEGATIVE (-ve)         CASTS       NEGATIVE (-ve)       NEGATIVE (-ve)	
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	
CASTS NECATIVE (-vo) NECATIVE (-vo)	
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	
BACTERIA NEGATIVE (-ve) NEGATIVE (-ve)	
OTHERS NEGATIVE (-ve) NEGATIVE (-ve)	
TRICHOMONAS VAGINALIS (PROTOZOA)       ABSENT       ABSENT         by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT       ABSENT       ABSENT	

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





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