



	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant		Dr. Yugam Chop MD (Patholo CEO & Consultant Patholo	ogy)
NAME	: Mrs. USHA WALIA			
AGE/ GENDER	: 53 YRS/FEMALE	PATIE	NT ID : 181	3651
COLLECTED BY	: SURJESH	REG. N	0./LAB NO. : 012	2504010023
REFERRED BY	:	REGIST	FRATION DATE : 01/	′Apr/2025 10:16 AM
BARCODE NO.	:01528137	COLLE	CTION DATE : 01/	Apr/2025 10:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPOR	TING DATE : 01/	Apr/2025 11:04AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	LA CANTT		
Fest Name		Value	Unit	Biological Reference interval
	SWASTH	IYA WELLNI	ESS PANEL: G	
	COMPL	ETE BLOOD (COUNT (CBC)	
RED BLOOD CELI	LS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H	B)	10 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL	(RBC) COUNT OCUSING, ELECTRICAL IMPEDENCE	4.93	Millions/cmm	3.50 - 5.00
PACKED CELL VOI	LUME (PCV)	33.5 ^L	%	37.0 - 50.0
MEAN CORPUSCUI	UTOMATED HEMATOLOGY ANALYZER LAR VOLUME (MCV)	67.8 ^L	fL	80.0 - 100.0
MEAN CORPUSCUI	UTOMATED HEMATOLOGY ANALYZER LAR HAEMOGLOBIN (MCH)	20.2 ^L	pg	27.0 - 34.0
	UTOMATED HEMATOLOGY ANALYZER LAR HEMOGLOBIN CONC. (MCHC		g/dL	32.0 - 36.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER	20.1	-	
	BUTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	15.7	%	11.00 - 16.00
RED CELL DISTRI	BUTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	39.7	fL	35.0 - 56.0
MENTZERS INDEX		13.75	RATIO	BETA THALASSEMIA TRAIT:
by CALCULATED				13.0
				IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING IN	DEX	72.17	RATIO	BETA THALASSEMIA TRAIT:
by CALCULATED				<= 65.0
				IRON DEFICIENCY ANEMIA: 65.0
	ELLS (WBCS)			55.0
<u>WHITE BLOOD CI</u>		7010	/cmm	4000 - 11000
TOTAL LEUCOCY				
NUCLEATED RED	IE COUNT (IEC) ['] BY SF CUBE & MICROSCOPY BLOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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NAME	: Mrs. USHA WALIA			
AGE/ GENDER	: 53 YRS/FEMALE	РАТ	TIENT ID	: 1813651
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Test Name		Value	Unit	Biological Reference interval
	AUTOMATED HEMATOLOGY ANALYZER			
	<u>EUCOCYTE COUNT (DLC)</u>			
NEUTROPHILS	Y BY SF CUBE & MICROSCOPY	59	%	50 - 70
LYMPHOCYTES		32	%	20 - 40
by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY			
EOSINOPHILS	Y BY SF CUBE & MICROSCOPY	4	%	1 - 6
MONOCYTES	T BT SF COBE & MICKOSCOFT	5	%	2 - 12
-	Y BY SF CUBE & MICROSCOPY			
BASOPHILS	Y BY SF CUBE & MICROSCOPY	0	%	0 - 1
	COCYTES (WBC) COUNT			
ABSOLUTE NEUTI		4136	/cmm	2000 - 7500
	Y BY SF CUBE & MICROSCOPY	4150	/clilli	2000 - 7500
ABSOLUTE LYMP		2243	/cmm	800 - 4900
by FLOW CYTOMETR ABSOLUTE EOSIN	Y BY SF CUBE & MICROSCOPY	280	/cmm	40 - 440
	Y BY SF CUBE & MICROSCOPY	280	/ciiiii	40 - 440
ABSOLUTE MONC		350	/cmm	80 - 880
by FLOW CYTOMETR ABSOLUTE BASOI	PY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
	Y BY SF CUBE & MICROSCOPY	0	/ciiiii	0 - 110
PLATELETS AND	OTHER PLATELET PREDICTIV	<u>E MARKERS.</u>		
PLATELET COUN		158000	/cmm	150000 - 450000
	FOCUSING, ELECTRICAL IMPEDENCE	0.17	0/	0.10 0.26
PLATELETCRIT (I by HYDRO DYNAMIC I	FOCUSING, ELECTRICAL IMPEDENCE	0.17	%	0.10 - 0.36
MEAN PLATELET	VOLUME (MPV)	15 ^H	fL	6.50 - 12.0
	FOCUSING, ELECTRICAL IMPEDENCE		(20000 00000
	E CELL COUNT (P-LCC) FOCUSING, ELECTRICAL IMPEDENCE	79000	/cmm	30000 - 90000
PLATELET LARGE	E CELL RATIO (P-LCR)	68.9 ^H	%	11.0 - 45.0
	FOCUSING, ELECTRICAL IMPEDENCE			15.0 17.0
PLATELET DISTR	IBUTION WIDTH (PDW)	15	%	15.0 - 17.0



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Test Name		Value Unit	Biological Reference interval

by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AI				
Test Name		Value	Unit	Biological Reference inter	rval
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVER.	IAEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	5.2 102.54	% mg/dL	4.0 - 6.4 60.00 - 140.00	
	AS PER AMERICAN D	ABETES ASSOCIA	TION (ADA):		
	REFERENCE GROUP		YCOSYLATED HEMOGLOGIB	(HBAIC) in %	
	abetic Adults >= 18 years	1	<5.7		
	t Risk (Prediabetes)		5.7 – 6.4		
D	iagnosing Diabetes		>= 6.5		
Therapeut	ic goals for glycemic control		Age > 19 Years of Therapy: s Suggested:	< 7.0 >8.0	
			Age < 19 Years		
		01	of therapy:	<7.5	

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients. 2.Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

3. Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropriate.

4. High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications 5. Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	/IBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
by RED CELL AGGRE	EDIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETRY	7	mm/1st ł	nr 0 - 20
immune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus eryth CONDITION WITH LO' A low ESR can be see (polycythaemia), sigr	does not tell the health practitione octed by other conditions besides in be used to monitor disease activity ematosus W ESR n with conditions that inhibit the n	er exactly where flammation. Fo and response to ormal sedimen nt (leucocytosis	e the inflammation is in the r this reason, the ESR is ty to therapy in both of the a tation of red blood cells, s	picallý used in conjunction with other test such bove diseases as well as some others, such as
NOTE: 1. ESR and C - reactiv 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dext	e protein (C-RP) are both markers o so not change as rapidly as does CRI by as many other factors as is ESR, ed, it is typically a result of two typ we a higher ESR, and menstruation	of inflammation. P, either at the making it a bet t es of proteins, and pregnancy	start of inflammation or a ter marker of inflammation globulins or fibrinogen. can cause temporary eleva	n.

aspirin, cortisone, and quinine may decrease it





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	ME	: Vinay Choj 9 (Pathology & M airman & Consul	licrobiology)	Dr. Yugan MD CEO & Consultant	(Pathology)	
IAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Mrs. USHA WA : 53 YRS/FEMAL : SURJESH : : 01528137 : KOS DIAGNOST : 6349/1, NICHO	E IC LAB	REGIS COLLE REPOI	NT ID 10./LAB NO. FRATION DATE CTION DATE RTING DATE	: 1813651 : 012504010023 : 01/Apr/2025 10:16 AM : 01/Apr/2025 10:34AM : 01/Apr/2025 12:24PM	
Fest Name			Value	Unit	Biological Reference interv	al
GLUCOSE FASTIN	G (F): PLASMA		GLUCOSE FAS ⁷ 108.36 ^H	FING (F) mg/dL	NORMAL: < 100.0	
est (after consumpt 3. A fasting plasma g	lucose level below lucose level betwee on of 75 gms of glu lucose level of aboy	100 mg/dl is cor en 100 - 125 mg cose) is recomm re 125 mg/dl is t	nsidered normal. /dl is considered as glu lended for all such pat highly suggestive of di	ients. abetic state. A repe	DIABETIC: > 0R = 126.0 prediabetic. A fasting and post-prandial bl at post-prandial is strongly recommended atory for diabetic state.	

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PRO	OFILE : BASIC	
CHOLESTEROL TO by CHOLESTEROL OX		142.93	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
TRIGLYCERIDES: 5 by GLYCEROL PHOSE	SERUM PHATE OXIDASE (ENZYMATIC)	125.27	mg/dL	240.0 OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERC by SELECTIVE INHIBIT	DL (DIRECT): SERUM 70N	37.87	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERC by CALCULATED, SPE		80.01	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 CHOLES by CALCULATED, SPE		105.06	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 VERY HIGH: > OR = 220.0
VLDL CHOLESTER by CALCULATED, SPE		25.05	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SE by CALCULATED, SPE	RUM	411.13	mg/dL	350.00 - 700.00
CHOLESTEROL/HI by CALCULATED, SPE	DL RATIO: SERUM	3.77	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0
	Ch		Shopra	

DR.YUGAM CHOPRA

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- N







		Chopra y & Microbiology) consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S		2.11	RATIO	MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0
TRIGLYCERIDES/I by CALCULATED, SPE	HDL RATIO: SERUM	3.31	RATIO	HIGH RISK: > 6.0 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.

 Cow 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
	I IVER F		N TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI		0.83	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	T (CONJUGATED): SERUM	0.23	mg/dL	0.00 - 0.40
BILIRUBIN INDIRI by CALCULATED, SPE	ECT (UNCONJUGATED): SERUM	0.6	mg/dL	0.10 - 1.00
SGOT/AST: SERUN by IFCC, WITHOUT PY	Л /RIDOXAL PHOSPHATE	19.55	U/L	7.00 - 45.00
SGPT/ALT: SERUN by IFCC, WITHOUT PY	1 (RIDOXAL PHOSPHATE	17.34	U/L	0.00 - 49.00
AST/ALT RATIO: S	ERUM ECTROPHOTOMETRY	1.13	RATIO	0.00 - 46.00
ALKALINE PHOSP by PARA NITROPHEN PROPANOL	HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL	159.74 ^H	U/L	40.0 - 130.0
GAMMA GLUTAM by SZASZ, SPECTRO	YL TRANSFERASE (GGT): SERUM PHTOMETRY	18.46	U/L	0.00 - 55.0
TOTAL PROTEINS		6.42	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		4.1	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	IN	2.32	gm/dL	2.30 - 3.50
A : G RATIO: SERU		1.77	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





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HEPATOCELLULAR C	ARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly In	ncreased)

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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Test Name		Value	Unit	Biological Reference interval
	KIDNEY	FUNCTIO	N TEST (COMPLET)	E)
UREA: SERUM		29.91	mg/dL	10.00 - 50.00
	ATE DEHYDROGENASE (GLDH)			
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY		0.67	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM		13.98	mg/dL	7.0 - 25.0
by CALCULATED, SPECTROPHOTOMETRY				
BLOOD UREA NIT RATIO: SERUM	ROGEN (BUN)/CREATININE	20.87 ^H	RATIO	10.0 - 20.0
	ECTROPHOTOMETRY			
UREA/CREATININ		44.64	RATIO	
URIC ACID: SERUN	ECTROPHOTOMETRY M	4.24	mg/dL	2.50 - 6.80
by URICASE - OXIDAS			ing all	2.00 0.00
CALCIUM: SERUM		8.57	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE PHOSPHOROUS: S		3.15	mg/dL	2.30 - 4.70
by PHOSPHOMOLYBL	DATE, SPECTROPHOTOMETRY			
<u>ELECTROLYTES</u>				
SODIUM: SERUM by ISE (ION SELECTIV		143.5	mmol/L	135.0 - 150.0
POTASSIUM: SERU		4.22	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV	/E ELECTRODE)			
CHLORIDE: SERUN by ISE (ION SELECTIV		107.63	mmol/L	90.0 - 110.0
	MERULAR FILTERATION RATE	2		
ESTIMATED GLON	MERULAR FILTERATION RATE	104.4		
(eGFR): SERUM				
by CALCULATED INTERPRETATION:				
	een pre- and post renal azotemia.			

To differentiate between pre- and post renal azotemia. INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased glomerular filtration rate.



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





		Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant			m Chopra D (Pathology) nt Pathologist	
NAME	: Mrs. USHA	WALIA				
AGE/ GENDER	: 53 YRS/FEN	IALE	PAT	ENT ID	: 1813651	
COLLECTED BY	: SURJESH		RFG	NO./LAB NO.	:012504010023	
REFERRED BY				STRATION DATE	: 01/Apr/2025 10:16	6 AM
BARCODE NO.	:01528137			LECTION DATE	: 01/Apr/2025 10:14	
					1	
CLIENT CODE.	: KOS DIAGN			DRTING DATE	:01/Apr/2025 11:39	9AM
CLIENT ADDRESS	: 6349/1, NI	CHOLSON ROAD, AMBA	LA CANTT			
Test Name			Value	Unit	Biological	l Reference interval
burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2	ke or production xia, high fever) (e.g. ureter col ass (subnorma tetracycline, gl 20:1) WITH ELEV	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL	S:		cosis, Cushing's syndrom	ne, high protein diet,
6. Excess protein inta burns, surgery, cache 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 ar 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome (8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin thera	ke or production xia, high fever) (e.g. ureter colloass (subnorma tetracycline, gl co:1) WITH ELEV a (BUN rises dis superimposed to:1) WITH DEC osis. Ind starvation. e. creased urea sy furea rather that monemias (urea of inappropiate to:1) WITH INCF py (accelerates eleases muscle who develop ro- sis (acetoaceta creased BUN/c rapy (interferes	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more th on renal disease. REASED BUN : (nthesis. an creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d REASED CREATININE: conversion of creatine f creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measure	S: han creatinine) (e ut of extracellula blood). lue to tubular se to creatinine). in creatinine wi	e.g. obstructive urop r fluid). cretion of urea.		
6. Excess protein inta burns, surgery, cache 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 ar 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome (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	ke or production xia, high fever) (e.g. ureter colloass (subnorma tetracycline, gl co:1) WITH ELEV a (BUN rises dis superimposed to:1) WITH DEC osis. Ind starvation. e. creased urea sy furea rather that monemias (urea of inappropiate to:1) WITH INCF py (accelerates eleases muscle who develop ro- sis (acetoaceta creased BUN/c rapy (interferes	ostomy) I creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more th on renal disease. REASED BUN : In creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d REASED CREATININE: conversion of creatine to creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measure DN RATE:	S: ban creatinine) (e ut of extracellula blood). lue to tubular set to creatinine). in creatinine wi ement).	r fluid). cretion of urea.	bathy). logies,resulting in norma	
6. Excess protein inta burns, surgery, cache 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 ar 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome (8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin thera	ke or production xia, high fever) (e.g. ureter coll ass (subnorma tetracycline, gl co:1) WITH ELEV a (BUN rises dis superimposed to:1) WITH DEC osis. Ind starvation. e. creased urea s furea rather that monemias (urea finappropiate to:1) WITH INCF py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/c apy (interferes JLAR FILTERATION Not	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more th on renal disease. REASED BUN : (nthesis. an creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d REASED CREATININE: conversion of creatine f creatinine). enal failure. te causes false increase reatinine ratio). with creatinine measure	S: han creatinine) (e ut of extracellula blood). lue to tubular se to creatinine). in creatinine wi	r fluid). cretion of urea. th certain methodo	pathy).	

G2	Kidney damage with	>90	Presence of Protein ,
	normal or high GFR		Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	
<u> </u>		10 27	



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	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultan	obiology) MI	m Chopra D (Pathology) ht Pathologist
NAME	: Mrs. USHA WALIA		
AGE/ GENDER	: 53 YRS/FEMALE	PATIENT ID	: 1813651
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012504010023
REFERRED BY	:	REGISTRATION DATE	: 01/Apr/2025 10:16 AM
BARCODE NO.	: 01528137	COLLECTION DATE	: 01/Apr/2025 10:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 01/Apr/2025 11:39AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA CANTT	
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

End Of Report ***





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