

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mrs. SUDESH KUMARI			
AGE/ GENDER	: 75 YRS/FEMALE		PATIENT ID	: 1793984
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012503170022
REFERRED BY	:		REGISTRATION DATE	: 17/Mar/2025 09:14 AM
BARCODE NO.	: 01527245		COLLECTION DATE	: 17/Mar/2025 09:22AM
CLIENT CODE. CLIENT ADDRESS	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMB/		REPORTING DATE	: 17/Mar/2025 09:43AM
Test Name		Value	Unit	<b>Biological Reference interval</b>
	SWAST	THYA WE	ELLNESS PANEL: G	
	COMP	PLETE BLO	DOD COUNT (CBC)	
RED BLOOD CELLS	(RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HI	3)	10.2 <sup>L</sup>	gm/dL	12.0 - 16.0
RED BLOOD CELL (I		4.9	Millions/	cmm 3.50 - 5.00
by HYDRO DYNAMIC FO	DCUSING, ELECTRICAL IMPEDENCE	33.5 <sup>L</sup>	%	37.0 - 50.0
by CALCULATED BY A	JTOMATED HEMATOLOGY ANALYZER		fL	80.0 - 100.0
	JTOMATED HEMATOLOGY ANALYZER	68.4 <sup>L</sup>	IL	
	AR HAEMOGLOBIN (MCH) JTOMATED HEMATOLOGY ANALYZER	20.9 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCUL	AR HEMOGLOBIN CONC. (MCHC) JTOMATED HEMATOLOGY ANALYZER	30.5 <sup>L</sup>	g/dL	32.0 - 36.0
	JTION WIDTH (RDW-CV) JTOMATED HEMATOLOGY ANALYZER	17.7 <sup>H</sup>	%	11.00 - 16.00
RED CELL DISTRIBU	JTION WIDTH (RDW-SD) JTOMATED HEMATOLOGY ANALYZER	45.4	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		13.96	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING IND by CALCULATED		24.81	RATIO	BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CEI		7.102		
TOTAL LEUCOCYTE by FLOW CYTOMETRY	COUNT (TLC) By SF CUBE & MICROSCOPY	7430	/cmm	4000 - 11000
NUCLEATED RED B	LOOD CELLS (nRBCS) t hematology analyzer	NIL		0.00 - 20.00

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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Dr Vinay Ch



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	Chairman & Consu		CEO & Consultant		
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Test Name		Value	Unit	Biological Reference interval	
	UCOCYTE COUNT (DLC)				
NEUTROPHILS		57	%	50 - 70	
by FLOW CYTOMETRY LYMPHOCYTES	Y BY SF CUBE & MICROSCOPY	34	%	20 - 40	
	Y BY SF CUBE & MICROSCOPY	34	%	20 - 40	
EOSINOPHILS	Y BY SF CUBE & MICROSCOPY	2	%	1 - 6	
MONOCYTES	Y BY SF CUBE & MICROSCOPY	7	%	2 - 12	
BASOPHILS	Y BY SF CUBE & MICROSCOPY	0	%	0 - 1	
•	CYTES (WBC) COUNT				
ABSOLUTE NEUTR by FLOW CYTOMETR	OPHIL COUNT y by sf cube & microscopy	4235	/cmm	2000 - 7500	
ABSOLUTE LYMPH by FLOW CYTOMETRY	OCYTE COUNT ( by sf cube & microscopy	2526	/cmm	800 - 4900	
ABSOLUTE EOSINC		149	/cmm	40 - 440	
ABSOLUTE MONOC	YTE COUNT	520	/cmm	80 - 880	
	Y BY SF CUBE & MICROSCOPY D <b>THER PLATELET PREDICTIV</b>	E MARKERS			
PLATELET COUNT		277000	/cmm	150000 - 450000	
	OCUSING, ELECTRICAL IMPEDENCE	0.0	0/	0.10, 0.00	
PLATELETCRIT (PC by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE	0.3	%	0.10 - 0.36	
MEAN PLATELET V	OLUME (MPV) OCUSING, ELECTRICAL IMPEDENCE	11	fL	6.50 - 12.0	
PLATELET LARGE	CELL COUNT (P-LCC) CCUSING, ELECTRICAL IMPEDENCE	97000 <sup>H</sup>	/cmm	30000 - 90000	
PLATELET LARGE	CELL RATIO (P-LCR)	35	%	11.0 - 45.0	
PLATELET DISTRIE	BUTION WIDTH (PDW)	15.7	%	15.0 - 17.0	
-	CTED ON EDTA WHOLE BLOOD				





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A			. 177 Mai, 2020 01.001 M
	, , , , , , , , , , , , , , , , , , , ,			
Test Name		Value	Unit	Biological Reference interva
WHOLE BLOOD by HPLC (HIGH PERFOR	EMOGLOBIN (HbA1c):	7.8 <sup>H</sup>	%	4.0 - 6.4
	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	177.16 <sup>H</sup>	mg/dL	60.00 - 140.00
INTERPRETATION:	,			
	AS PER AMERICAN E	DIABETES ASSOCIATION	I (ADA):	
	REFERENCE GROUP		YLATED HEMOGLOGIB	(HBAIC) in %
Non dia	abetic Adults >= 18 years	/	<5.7	
	t Risk (Prediabetes)		5.7 – 6.4	
D	iagnosing Diabetes		>= 6.5	
			Age > 19 Years	
These l	in and a few shares in a sector i	Goals of Th		< 7.0
Inerapeut	ic goals for glycemic control	Actions Sugg		>8.0
1		Goal of the	Age < 19 Years	
				<7.5

**KOS Diagnostic Lab** 

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## COMMENTS:

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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	ERYTHROC	CYTE SEDIME	NTATION RATE (	ESR)
INTERPRETATION: 1. ESR is a non-specifimmune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythe CONDITION WITH LOV A low ESR can be see (polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactive 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dext	does not tell the health practitioner cted by other conditions besides infl be used to monitor disease activity a ematosus <b>W ESR</b> n with conditions that inhibit the no ificantly high white blood cell count e cell anaemia) also lower the ESR. e protein (C-RP) are both markers of s not change as rapidly as does CRP, <b>by as many other factors as is ESR</b> , n ed, it is typically a result of two type ye a higher ESR. and menstruation al	exactly where the lammation. For the and response to the prmal sedimentation (leucocytosis), a finflammation. , either at the stan making it a better se of proteins, glo nd pregnancy car	e inflammation is in the his reason, the ESR is typ herapy in both of the a on of red blood cells, su and some protein abno rt of inflammation or as marker of inflammatior bulins or fibrinogen. i cause temporary eleva	pically used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count irmalities. Some changes in red cell shape (such s it resolves. <b>n</b> .





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLINI	CAL CHEMISTRY GLUCOSE FAS		TRY
GLUCOSE FASTING by glucose oxidas	(F): PLASMA E - PEROXIDASE (GOD-POD)	136.1 <sup>H</sup>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

**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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CLIENT CODE. CLIENT ADDRESS	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROA		RTING DATE	: 17/Mar/2025 11:45AM
Test Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PROFILE	·BASIC	
CHOLESTEROL TO		154.48	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		134.40	ing/ uL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S. by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	132.4	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
HDL CHOLESTEROI by SELECTIVE INHIBIT.	L (DIRECT): SERUM Ion	53.14	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		74.86	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		101.34	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0
VLDL CHOLESTER		26.48	mg/dL	VERY HIGH: > OR = 220.0 0.00 - 45.00
by CALCULATED, SPE TOTAL LIPIDS: SER by CALCULATED, SPE	CUM	441.36	mg/dL	350.00 - 700.00
by CALCULATED, SPE CHOLESTEROL/HD by CALCULATED, SPE	DL RATIO: SERUM	2.91	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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT	,	
Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		1.41	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	IDL RATIO: SERUM	2.49 <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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	LIVER	FUNCTION	TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI	: SERUM PECTROPHOTOMETRY	0.62	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	Г (CONJUGATED): SERUM spectrophotometry	0.15	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE by CALCULATED, SPE	CCT (UNCONJUGATED): SERUM	0.47	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	[ /RIDOXAL PHOSPHATE	22.3	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	[ /RIDOXAL PHOSPHATE	15.8	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE		1.41	RATIO	0.00 - 46.00
ALKALINE PHOSPI by PARA NITROPHEN PROPANOL	HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL	82.36	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTROF	L TRANSFERASE (GGT): SERUM	18.41	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.72	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		3.87	gm/dL	3.50 - 5.50
GLOBULIN: SERUM	1	2.85	gm/dL	2.30 - 3.50
by CALCULATED, SPE A : G RATIO: SERUI		1.36	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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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).

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 interva
	KIDNE	EY FUNCTION	N TEST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAN	IATE DEHYDROGENASE (GLDH)	43.01	mg/dL	10.00 - 50.00
CREATININE: SERI		0.99	mg/dL	0.40 - 1.20
-	ROGEN (BUN): SERUM	20.1	mg/dL	7.0 - 25.0
	ROGEN (BUN)/CREATININE	20.3 <sup>H</sup>	RATIO	10.0 - 20.0
UREA/CREATININ by CALCULATED, SPE	E RATIO: SERUM	43.44	RATIO	
URIC ACID: SERUM	1	4.05	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPE		9.58	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE by PHOSPHOMOLYBE		3.46	mg/dL	2.30 - 4.70
<u>ELECTROLYTES</u>				
SODIUM: SERUM by ISE (ION SELECTIV	(F ELECTRODE)	140.9	mmol/L	135.0 - 150.0
POTASSIUM: SERU by ISE (ION SELECTIV	M	5.06 <sup>H</sup>	mmol/L	3.50 - 5.00
CHLORIDE: SERUN by ISE (ION SELECTIV	1	105.68	mmol/L	90.0 - 110.0
	ERULAR FILTERATION RATE	59.5		

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.





	Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist						
IAME	: Mrs. SUDESH KUMARI						
GE/ GENDER	: 75 YRS/FEMALE	1	PATIENT ID	: 1793	984		
<b>OLLECTED BY</b>	: SURJESH	I	REG. NO./LAB NO	. : 012;	503170022		
REFERRED BY			REGISTRATION D		lar/2025 09:1	1 <i>4</i> AM	
ARCODE NO.	: 01527245		COLLECTION DAT		lar/2025 09:2		
LIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DAT		lar/2025 12:2		
LIENT CODE.	: 6349/1, NICHOLSON ROAI		LF OK I ING DA I	<b>E</b> . 1771VI	idi / 2023 12.2		
Fest Name		Value	Ur	nit	Biologica	l Reference i	nterval
9. Certain drugs (e.g. NCREASED RATIO (>2 . Postrenal azotemia	hass (subnormal creatinine pro tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately supprimposed on ronal disease	<b>NE LEVELS:</b> / more than creatinin	e) (e.g. obstructiv	e uropathy).			
Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Low protein diet al     Severe liver diseas     Other causes of de     Repeated dialysis     Inherited hyperam     SIADH (syndrome of     Pregnancy.     Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido     hould produce an in     SETIMATED GLOMERI     CKD STAGE	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false creased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION	NE LEVELS: / more than creatinin e. ffuses out of extrace bsent in blood). rmone) due to tubula VINE: creatine to creatinine increase in creatinine measurement). J	Ilular fluid). r secretion of urea e). e with certain me ./min/1.73m2 )	a. thodologies,resu	FINDINGS	al ratio when c	dehydratio
Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Low protein diet al     Severe liver diseas     Other causes of de     Repeated dialysis     Inherited hyperam     SIADH (syndrome of     Pregnancy.     DECREASED RATIO (<         Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido hould produce an in     Cephalosporin thei     STIMATED GLOMERI     CKD STAGE     G1	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun	NE LEVELS: y more than creatinin e. ffuses out of extrace bsent in blood). rmone) due to tubula JINE: creatine to creatinine increase in creatinine measurement). J GFR (mil nction	Ilular fluid). r secretion of urea e). e with certain me <u>/min/1.73m2 )</u> >90	a. thodologies,resu ASSOCIATED No prote	FINDINGS	al ratio when c	dehydratio
Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Low protein diet al     Severe liver diseas     Other causes of de     Repeated dialysis     Inherited hyperam     SIADH (syndrome of     Pregnancy.     Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido     hould produce an in     Cephalosporin thei     STADE GLOMERI     STAGE	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage normal or high	NE LEVELS: more than creatinin e. ffuses out of extrace bsent in blood). rmone) due to tubula MINE: creatine to creatinine increase in creatinine measurement). M GFR (mil GFR	Ilular fluid). r secretion of urea e). e with certain me ./min/1.73m2 )	a. thodologies,resu	FINDINGS einuria Protein ,	al ratio when c	dehydratio
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Certain drugs (e.g.     VCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Low protein diet al     Severe liver diseas     Other causes of de     Repeated dialysis     Inherited hyperam     SIADH (syndrome of     Pregnancy.     DECREASED RATIO (<         Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Loid produce an in     Cephalosporin thee     STIMATED GLOMERNI     G1     G2     G3a     G3b	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATINI a (BUN rises disproportionately superimposed on renal diseas 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han 10:1) WITH INCREASED CREATIN py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage normal or high Moderate decrease	NE LEVELS:         y more than creatining         e.         offuses out of extrace         osent in blood).         rmone) due to tubula         JINE:         creatine to creatining         increase in creatining         measurement).         M         GFR         GFR         in GFR	Ilular fluid). r secretion of urea e). e with certain me <u>/min/1.73m2 )</u> >90 >90 60 -89 30-59	a. thodologies,resu ASSOCIATED No prote Presence of	FINDINGS einuria Protein ,	al ratio when c	dehydratio
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NAME	: Mrs. SUDESH KUMARI				
AGE/ GENDER	: 75 YRS/FEMALE	PATIENT ID	: 1793984		
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012503170022		
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 17/Mar/2025 09:14 AM		
BARCODE NO.	: 01527245	COLLECTION DATE	: 17/Mar/2025 09:22AM		
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 17/Mar/2025 12:20PM		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBAL				
Test Name	v	alue 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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