



	Dr. Vinay Chop MD (Pathology & Mic Chairman & Consulta	crobiology)	MD	m Chopra D (Pathology) nt Pathologist
NAME	: Mr. GURMEJ SINGH			
AGE/ GENDER	: 86 YRS/MALE		PATIENT ID	: 1683556
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	:012411270025
REFERRED BY	:		REGISTRATION DATE	: 27/Nov/2024 10:15 AM
BARCODE NO.	:01521527		COLLECTION DATE	: 27/Nov/2024 10:22AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 27/Nov/2024 10:36AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMI	BALA CANTI	Γ	
Test Name		Value	Unit	Biological Reference interval
	СОМ		ELLNESS PANEL: 1.0 .00D COUNT (CBC)	.0
	(RBCS) COUNT AND INDICES	10		10.0 17.0
HAEMOGLOBIN (H)	В)	12	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT OCUSING, ELECTRICAL IMPEDENCE	4.33	Millions	
PACKED CELL VOLU		37.5 ^L	%	40.0 - 54.0
MEAN CORPUSCUL		86.7	fL	80.0 - 100.0
MEAN CORPUSCUL	AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	27.7	pg	27.0 - 34.0
	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	32	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	15.1	%	11.00 - 16.00
	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	49	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		20.02	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INE by CALCULATED		30.22	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE				
TOTAL LEUCOCYTE	COUNT (TLC) by sf cube & microscopy	7170	/cmm	4000 - 11000
	LOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
NUCLEATED RED B	LOOD CELLS (nRBCS) % utomated hematology analyzer	NIL	%	< 10 %





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Test Name		Value	Unit	Biological Reference interval
DIFFERENTIAL LE	UCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY	52	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY	36	%	20 - 40
EOSINOPHILS by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY	4	%	1 - 6
MONOCYTES by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY	8	%	2 - 12
BASOPHILS by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKO	CYTES (WBC) COUNT			
ABSOLUTE NEUTR	OPHIL COUNT y by sf cube & microscopy	3728	/cmm	2000 - 7500
ABSOLUTE LYMPH by FLOW CYTOMETR	OCYTE COUNT y by sf cube & microscopy	2581	/cmm	800 - 4900
ABSOLUTE EOSINO	OPHIL COUNT Y by sf cube & microscopy	287	/cmm	40 - 440
ABSOLUTE MONOC	CYTE COUNT Y by sf cube & microscopy	574	/cmm	80 - 880
<u>PLATELETS AND (</u>	OTHER PLATELET PREDICTIVE	<u>E MARKERS.</u>		
PLATELET COUNT by hydro dynamic f	(PLT) FOCUSING, ELECTRICAL IMPEDENCE	226000	/cmm	150000 - 450000
PLATELETCRIT (PC	CT) FOCUSING, ELECTRICAL IMPEDENCE	0.24	%	0.10 - 0.36
MEAN PLATELET V		11	fL	6.50 - 12.0
PLATELET LARGE	CELL COUNT (P-LCC)	71000	/cmm	30000 - 90000
PLATELET LARGE	CELL RATIO (P-LCR) FOCUSING, ELECTRICAL IMPEDENCE	31.3	%	11.0 - 45.0
PLATELET DISTRI	BUTION WIDTH (PDW) FOCUSING, ELECTRICAL IMPEDENCE	16	%	15.0 - 17.0
-	ICTED ON EDTA WHOLE BLOOD			





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ARCODE NO.	: 01521527		COLLECTION DATE	: 27/Nov/2024 10:22AM
LIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 27/Nov/2024 10:56AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AI	MBALA CANTT		
'est Name		Value	Unit	Biological Reference interval
Imune disease, but An ESR can be affe C-reactive protein This test may also Stemic lupus eryth DNDITION WITH LO low ESR can be see olycythaemia), sigr sickle cells in sickl DTE: ESR and C - reactiv Generally, ESR doe CRP is not affected If the ESR is elevat Women tend to ha Drugs such as dext	does not tell the health practition cted by other conditions besides in be used to monitor disease activity ematosus WESR n with conditions that inhibit the r hificantly high white blood cell cou e cell anaemia) also lower the ESF e protein (C-RP) are both markers of ses not change as rapidly as does CR by as many other factors as is ESR, ed, it is typically a result of two typ ye a higher ESR, and menstruation	er exactly wher flammation. For y and response hormal sedimer int (leucocytosi R. of inflammation P, either at the making it a be bes of proteins, and pregnancy	te the inflammation is in the pr this reason, the ESR is ty to therapy in both of the a ntation of red blood cells, s s) , and some protein abno n. e start of inflammation or as tter marker of inflammation globulins or fibrinogen. can cause temporary eleva	picallý used in conjunctión with other test sucl bove diseases as well as some others, such as uch as a high red blood cell count rmalities. Some changes in red cell shape (suc s it resolves. 1 .





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	MD (Vinay Chopra Pathology & Microbiology) man & Consultant Patholog		(Pathology)	
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CLIENT CODE.	: KOS DIAGNOSTIC	LAB	REPORTING DATE	: 27/Nov/2024 12:24PM	
CLIENT ADDRESS	: 6349/1, NICHOLS	ON ROAD, AMBALA CANT	Т		
Test Name	_	Value	Unit	Biological Reference interval	
		CLINICAL CHEMI	STRY/BIOCHEMIST	'RY	
		GLUCOS	E FASTING (F)		

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

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 text (after consumption of 75 gms of glucose) is recommended for all such patients.

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 y & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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LIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
		LIPID PROF	ILE : BASIC	
HOLESTEROL TO	TAL · SFRUM	108.39	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		100.00	ing/ ull	BORDERLINE HIGH: 200.0 -
				239.0
				HIGH CHOLESTEROL: > OR = 240.0
RIGLYCERIDES: S		73.91	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSP	HATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 - 199.0
				HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
IDL CHOLESTERO	L (DIRECT): SERUM	46.96	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0
by CLECTIVE INITIAL				60.0
				HIGH HDL: $> OR = 60.0$
DL CHOLESTEROI		46.65	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.
<i>»</i> , <i>«</i> , <i>»</i>				BORDERLINE HIGH: 130.0 -
				159.0
				HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLEST	TEROL: SERUM	61.43	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPE	CTROPHOTOMETRY		Ū	ABOVE OPTIMAL: 130.0 - 159.
				BORDERLINE HIGH: 160.0 - 189.0
				HIGH: 190.0 - 219.0
LDL CHOLESTER	N · SEDIM	14.78	mg/dL	VERY HIGH: > OR = 220.0 0.00 - 45.00
by CALCULATED, SPE	CTROPHOTOMETRY	14.70		0.00 - 43.00
OTAL LIPIDS: SER		290.69 ^L	mg/dL	350.00 - 700.00
HOLESTEROL/HD	L RATIO: SERUM	2.31	RATIO	LOW RISK: 3.30 - 4.40
by CALCULATED, SPE	CTROPHOTOMETRY			AVERAGE RISK: 4.50 - 7.0
				MODERATE RISK: 7.10 - 11.0

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		sultant Pathologist	CEO & Consultant	Pathologist
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S		0.99	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	1.57 ^L	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
			EST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI	: SERUM pectrophotometry	0.59	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	Г (CONJUGATED): SERUM spectrophotometry	0.18	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE by CALCULATED, SPE	CCT (UNCONJUGATED): SERUM	0.41	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	[/RIDOXAL PHOSPHATE	22.9	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	[/RIDOXAL PHOSPHATE	24.9	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE		0.92	RATIO	0.00 - 46.00
ALKALINE PHOSPI by PARA NITROPHEN PROPANOL	HATASE: SERUM I'YL PHOSPHATASE BY AMINO METHYL	106.83	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRON	L TRANSFERASE (GGT): SERUM PHTOMETRY	18.64	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.8	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		4.14	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE		2.66	gm/dL	2.30 - 3.50
A : G RATIO: SERUI	M ectrophotometry	1.56	RATIO	1.00 - 2.00

by CALCULATED, SPECTROPHOTOMETRY

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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INTERPRETATION





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Test Name	Valu	le 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



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 27/Nov/2024 01:40PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
	KIDNE	EY FUNCTION 7	TEST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAN	IATE DEHYDROGENASE (GLDH)	33.59	mg/dL	10.00 - 50.00
CREATININE: SERI		1.11	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY		15.7	mg/dL	7.0 - 25.0
BLOOD UREA NITH RATIO: SERUM by CALCULATED, SPE	ROGEN (BUN)/CREATININE	14.14	RATIO	10.0 - 20.0
UREA/CREATININ by CALCULATED, SPE	E RATIO: SERUM	30.26	RATIO	
URIC ACID: SERUM		3.69	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE		10.5	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE by phosphomolybe		3.18	mg/dL	2.30 - 4.70
<u>ELECTROLYTES</u>				
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	142.3	mmol/L	135.0 - 150.0
POTASSIUM: SERU by ISE (ION SELECTIV	M	4.02	mmol/L	3.50 - 5.00
CHLORIDE: SERUN by ISE (ION SELECTIV	1	106.73	mmol/L	90.0 - 110.0
ESTIMATED GLOM (eGFR): SERUM by calculated	ERULAR FILTERATION RATE	64.7		
<u>INTERPRETATION:</u> To differentiate betw	veen 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.

2. Catabolic states with increased tissue breakdown.

3. GI haemorrhage.



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT					: 27/Nov/2024 01:40PM		
Test Name			Value	Un	nit	Biolog	gical Refere	ence interv
INCREASED RĂTIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<	(BUN rises disp superimposed o 0:1) WITH DECR	TED CREATININE LEV coportionately more n renal disease.		ne) (e.g. obstructive	e uropathy)).		
NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 8. Phenacimide thera 2. Rhabdomyolysis (r 8. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1	0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Ind starvation. 2. creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop rent sis (acetoacetate creased BUN/crea apy (interferes v ULAR FILTERATION Nor	TED CREATININE LEV roportionately more n renal disease. EASED BUN : is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creatin reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu V RATE: DESCRIPTION mal kidney function	than creatini but of extrac blood). due to tubu e to creatinir se in creatini urement).	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90	a. thodologie: ASSOC	s,resulting in no IATED FINDINGS proteinuria	S	vhen dehydr
NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 8. Phenacimide thera 9. Rhabdomyolysis (r 8. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido 5. Nould produce an in 2. Cephalosporin ther 5. STIMATED GLOMERL OKD STAGE	0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Ind starvation. 2. creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/creased apy (interferes v ULAR FILTERATION Nor Nor	TED CREATININE LEV roportionately more n renal disease. EASED BUN : is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creatin reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu vith creatinine measu NATE: DESCRIPTION mal kidney function dney damage with	than creatini but of extrac blood). due to tubu e to creatinir se in creatini urement).	ellular fluid). lar secretion of urea ne). ne with certain met	a. thodologie: ASSOC	s,resulting in no IATED FINDINGS proteinuria nce of Protein ,	<u>s</u>	vhen dehydr
NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI CKD STAGE G1 G2	0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Ind starvation. 2. creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/crea apy (interferes v ULAR FILTERATION Nor Nor	TED CREATININE LEV roportionately more n renal disease. EASED BUN : is creatinine diffuses is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creatin reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu vith creatinine measu NATE: DESCRIPTION mal kidney function dney damage with prmal or high GFR	than creatini but of extrac blood). due to tubu e to creatinir se in creatini urement).	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90 >90	a. thodologie: ASSOC	s,resulting in no IATED FINDINGS proteinuria	<u>s</u>	vhen dehydr
NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1	0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECRI osis. Ind starvation. 2: creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea apy (interferes v UAR FILTERATION Nor Nor	TED CREATININE LEV roportionately more n renal disease. EASED BUN : is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creatin reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu vith creatinine measu NATE: DESCRIPTION mal kidney function dney damage with	than creatini but of extrac blood). due to tubu e to creatini e in creatini rement). GFR (n	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90	a. thodologie: ASSOC	s,resulting in no IATED FINDINGS proteinuria nce of Protein ,	<u>s</u>	vhen dehydr
INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERU CKD STAGE G1 G2 G3a	0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECRI osis. Ind starvation. E. creased urea syr urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea apy (interferes v UAR FILTERATION Nor Nor Nor Nor Nor Nor	TED CREATININE LEV roportionately more n renal disease. EASED BUN : thesis. creatinine diffuses is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creatin reatinine). hal failure. causes false increase eatinine ratio). vith creatinine measu vith creatinine measu vith creatinine measu vith creatinine measu vith creatinine measu MATE: DESCRIPTION mal kidney function dney damage with ormal or high GFR id decrease in GFR	than creatini but of extrac blood). due to tubu e to creatini e in creatini rement). GFR (n	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90 >90 60 -89	a. thodologie: ASSOC	s,resulting in no IATED FINDINGS proteinuria nce of Protein ,	<u>s</u>	vhen dehydr





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

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.0349/1, MURDLSON KOAD, J	AWDALA UANT I	
$\cdot 6240/1$ NICHOI SON POAD		
: KOS DIAGNOSTIC LAB	REPORTING DATE	: 27/Nov/2024 01:40PM
:01521527	COLLECTION DATE	: 27/Nov/2024 10:22AM
:	REGISTRATION DATE	: 27/Nov/2024 10:15 AM
: SURJESH	REG. NO./LAB NO.	:012411270025
: 86 YRS/MALE	PATIENT ID	: 1683556
: Mr. GURMEJ SINGH		
		D (Pathology) Int Pathologist
		m Chopra
	MD (Pathology & Chairman & Con : Mr. GURMEJ SINGH : 86 YRS/MALE : SURJESH : : 01521527 : KOS DIAGNOSTIC LAB	MD (Pathology & Microbiology) Chairman & Consultant Pathologist CEO & Consultant : Mr. GURMEJ SINGH : 86 YRS/MALE PATIENT ID : SURJESH REG. NO./LAB NO. : REGISTRATION DATE : 01521527 COLLECTION 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)

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	Dr. Vinay Chopra MD (Pathology & Microt Chairman & Consultant				
AGE/ GENDER: 86 YRS/NCOLLECTED BY: SURJESHREFERRED BY:BARCODE NO.: 0152152CLIENT CODE.: KOS DIA		COLLECTI REPORTI	LAB NO. ATION DATE ON DATE	: 1683556 : 012411270025 : 27/Nov/2024 10:15 AM : 27/Nov/2024 10:22AM : 27/Nov/2024 11:23AM	
Test Name	T	/alue	Unit	Biological Reference interval	
PHYSICAL EXAMINATION		NICAL PATHO E & MICROSCOF		ATION	
QUANTITY RECIEVED by DIP STICK/REFLECTANCE SPEC COLOUR by DIP STICK/REFLECTANCE SPEC TRANSPARANCY by DIP STICK/REFLECTANCE SPEC SPECIFIC GRAVITY by DIP STICK/REFLECTANCE SPEC	TROPHOTOMETRY TROPHOTOMETRY TROPHOTOMETRY	10 AMBER YELLOW CLEAR 1.01	ml	PALE YELLOW CLEAR 1.002 - 1.030	
CHEMICAL EXAMINATION REACTION by DIP STICK/REFLECTANCE SPEC PROTEIN by DIP STICK/REFLECTANCE SPEC SUGAR by DIP STICK/REFLECTANCE SPEC PH by DIP STICK/REFLECTANCE SPEC BILIRUBIN by DIP STICK/REFLECTANCE SPEC UROBILINOGEN by DIP STICK/REFLECTANCE SPEC KETONE BODIES by DIP STICK/REFLECTANCE SPEC BLOOD by DIP STICK/REFLECTANCE SPEC BLOOD by DIP STICK/REFLECTANCE SPEC BLOOD by DIP STICK/REFLECTANCE SPEC ASCORBIC ACID by DIP STICK/REFLECTANCE SPEC	TROPHOTOMETRY	NEUTRAL Negative Negative Negative Normal Negative Negative NEGATIVE (-ve)	EU/dL	NEGATIVE (-ve) NEGATIVE (-ve) 5.0 - 7.5 NEGATIVE (-ve) NEGATIVE (-ve) 0.2 - 1.0 NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve)	





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



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

NAME	: Mr. GURMEJ SINGH			
AGE/ GENDER	: 86 YRS/MALE]	PATIENT ID	: 1683556
COLLECTED BY	: SURJESH]	REG. NO./LAB NO.	: 012411270025
REFERRED BY	:]	REGISTRATION DATE	: 27/Nov/2024 10:15 AM
BARCODE NO.	: 01521527		COLLECTION DATE	: 27/Nov/2024 10:22AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB]	REPORTING DATE	: 27/Nov/2024 11:23AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
,	CENTRIFUGED URINARY SEDIMENT			
PUSCEUS		2-3	/HPF	0 - 5

PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-2	/HPF	ABSENT
CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
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





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