



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)	
NAME	: Mr. SAMPURAN SINGH BAINS				
AGE/ GENDER	: 69 YRS/MALE		PATIENT ID	: 1743531	
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012502030005	
REFERRED BY	:		REGISTRATION DATE	:03/Feb/202508:21 AM	
BARCODE NO.	: 01524847		COLLECTION DATE	:03/Feb/202508:31AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 03/Feb/2025 09:24AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB.	ALA CANTI			
Test Name		Value	Unit	Biological Reference	interval
	SWAST	HYA WF	LLNESS PANEL: 1.	0	
			OOD COUNT (CBC)	·	
RED BLOOD CELLS	(RBCS) COUNT AND INDICES				
HAEMOGLOBIN (H		14	gm/dL	12.0 - 17.0	
by CALORIMETRIC RED BLOOD CELL (4 77 1	Millions	/	
by HYDRO DYNAMIC F	RBC) COUNT OCUSING, ELECTRICAL IMPEDENCE	4.71	MIIIIONS	/cmm 3.50 - 5.00	
PACKED CELL VOLU	JME (PCV) UTOMATED HEMATOLOGY ANALYZER	44.5	%	40.0 - 54.0	
MEAN CORPUSCUL		94.5	fL	80.0 - 100.0	
MEAN CORPUSCUL	AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	29.8	pg	27.0 - 34.0	
MEAN CORPUSCUL	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	31.5 ^L	g/dL	32.0 - 36.0	
RED CELL DISTRIB	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	13.5	%	11.00 - 16.00	
RED CELL DISTRIB	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	47.4	fL	35.0 - 56.0	
MENTZERS INDEX		20.06	RATIO	BETA THALASSEMIA	TRAIT: <
by CALCULATED				13.0 IRON DEFICIENCY AN	NEMIA:
				>13.0	
GREEN & KING IND by CALCULATED	DEX	27.16	RATIO	BETA THALASSEMIA 65.0	TRAIT:<=
				IRON DEFICIENCY AN	NEMIA: >
WHITE BLOOD CE	LLS (WBCS)			65.0	
TOTAL LEUCOCYTE		6250	/cmm	4000 - 11000	
by FLOW CYTOMETRY	BY SF CUBE & MICROSCOPY			0.00 00.00	
by AUTOMATED 6 PAF	SLOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00	
	LOOD CELLS (nRBCS) % UTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %	
.,					
			A		





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KOS Diagnostic Lab (A Unit of KOS Healthcare)

MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Dr. Vinay Chopra



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

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	44 ^L	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	47 ^H	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2750	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2938	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	250	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by flow cytometry by sf cube & microscopy	312	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE	<u>MARKERS.</u>		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	124000 ^L	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.15	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	12	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	50000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	40.4	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence	16.6	%	15.0 - 17.0
ADVICE	KINDLY CORRE	LATE CLINICALLY	

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Test Name Value Unit **Biological Reference interval**

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

: 6349/1, NICHOLSON ROAD, AMBALA CANTT

RECHECKED.

CLIENT ADDRESS



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	:03/Feb/2025 10:18AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT	
Test Name		Value Unit	Biological Reference interval
	ERYTHROO	CYTE SEDIMENTATION RATI	E (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), sigras sickle cells in sickl NOTE: 1. ESR and C - reactive 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	does not tell the health practitioner cted by other conditions besides inf be used to monitor disease activity a ematosus W ESR n with conditions that inhibit the no ificantly high white blood cell coun e cell anaemia) also lower the ESR. e protein (C-RP) are both markers of s not change as rapidly as does CRP by as many other factors as is ESR, n ed, it is typically a result of two type ye a higher ESR. and menstruation a	r exactly where the inflammation is in lammation. For this reason, the ESR is and response to therapy in both of th prmal sedimentation of red blood cell- t (leucocytosis), and some protein at finflammation. , either at the start of inflammation o naking it a better marker of inflamma es of proteins, globulins or fibrinogen. Ind pregnancy can cause temporary el	s typicallý used in conjunction with other test such le above diseases as well as some others, such as s, such as a high red blood cell count onormalities. Some changes in red cell shape (such er as it resolves. tion.





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CLIENT ADDRESS	: 6349/1, NICHOLSON RO	AD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLI	NICAL CHEMIS	TRY/BIOCHEMIST	'RY
		GLUCOSE	FASTING (F)	
		dictool		

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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 test (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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Test Name		Value	Unit	Biological Reference interval
		LIPID PRO	FILE : BASIC	
CHOLESTEROL TOTA by CHOLESTEROL OXID		165.69	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SEF by GLYCEROL PHOSPHA	RUM ATE OXIDASE (ENZYMATIC)	167.23 ^H	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (by SELECTIVE INHIBITION		57.31	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: by CALCULATED, SPECT		74.93	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 CHOLESTE by calculated, spect		108.38	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL by CALCULATED, SPECT		33.45	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERU by CALCULATED, SPECT	Μ	498.61	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL by CALCULATED, SPECT	RATIO: SERUM	2.89	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0





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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S		1.31	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	2.92 ^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.

3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available

to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues. 4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL

5. Additional testing for Apolipoprotein B, hsCRP,Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement





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

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Test Name	Value	Unit	Biological Reference interval
LIVER	FUNCTION T	EST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.43	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.12	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.31	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	20.4	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	33.8	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by calculated, spectrophotometry	0.6	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	76.87	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	23.26	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.54	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.02	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.52	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.6	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
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)
HEPATOCELLULAR CARCINOMIA & CHRONIC HEPATTIS	> 1.3 (Slightly Increased)



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DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)

2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

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



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	KIDNE	Y FUNCTION	TEST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAM	ATE DEHYDROGENASE (GLDH)	25.15	mg/dL	10.00 - 50.00
CREATININE: SERU		1.02	mg/dL	0.40 - 1.40
BLOOD UREA NITR by CALCULATED, SPE	OGEN (BUN): SERUM	11.75	mg/dL	7.0 - 25.0
BLOOD UREA NITR RATIO: SERUM	COGEN (BUN)/CREATININE	11.52	RATIO	10.0 - 20.0
by CALCULATED, SPE	CTROPHOTOMETRY			
UREA/CREATININI by CALCULATED, SPE		24.66	RATIO	
URIC ACID: SERUM		5.77	mg/dL	3.60 - 7.70
by URICASE - OXIDAS CALCIUM: SERUM		9.37	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE PHOSPHOROUS: SE by PHOSPHOMOLYBD		3.51	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	E ELECTRODE)	137.6	mmol/L	135.0 - 150.0
POTASSIUM: SERUN by ISE (ION SELECTIV		4	mmol/L	3.50 - 5.00
CHLORIDE: SERUM		103.2	mmol/L	90.0 - 110.0
	IERULAR FILTERATION RATE			
ESTIMATED GLOM (eGFR): SERUM by CALCULATED INTERPRETATION:	ERULAR FILTERATION RATE	79.6		

<u>INTERPRETATION:</u> 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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REFERRED BY				REGISTRATION D		03/Feb/2025			
BARCODE NO.	: 01524847			COLLECTION DAT		03/Feb/2025			
CLIENT CODE.	: KOS DIAGNO	STIC I AB		REPORTING DATI		03/Feb/2025			
CLIENT ADDRESS		HOLSON ROAD, AMB	ALA CANTT			03/190/2023	10.J0AW		
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Fest Name			Value	Un	it	Biolog	gical Ref	erence int	terval
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necro	ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis.	stomy) creatinine productior cocorticoids) TED CREATININE LEV roportionately more n renal disease.) :LS:	ion, GI bleeding, thy ine) (e.g. obstructive					
2. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. NCREASED RATIO (>2 4. Postrenal azotemia 5. Prerenal azotemia 6. Acute tubular necro 6. Low protein diet ar 6. Severe liver disease 6. Other causes of de 6. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome c 6. Pregnancy. 7. Phenacimide thera 7. Rhabdomyolysis (ro 8. Muscular patients 7. NAPPROPIATE RATIO 7. Diabetic ketoacido 7. Diabetic ketoacido 7. Cephalosporin ther 7. STIMATED GLOMERL 7. CKD STAGE	(e.g. ureter cold ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. 2: creased urea syn urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/cre apy (interferes v LAR FILTERATIO	stomy) creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creating reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu V RATE: DESCRIPTION	e in creatini ement).	ine) (e.g. obstructive cellular fluid). lar secretion of urea ne). ine with certain met	e uropathy) I. hodologies ASSOC	s,resulting in no	ormal rati	o when de	
Urine reabsorption Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necro Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (ro Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an ine STIMATED GLOMERL CKD STAGE	(e.g. ureter colo ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. 2: creased urea syn urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ren sis (acetoacetate creased BUN/crea apy (interferes of LAR FILTERATIO	stomy) creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creating reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu V RATE: DESCRIPTION mal kidney function	e in creatini ement).	ine) (e.g. obstructive cellular fluid). lar secretion of urea ne). ine with certain met nL/min/1.73m2) >90	e uropathy) I. hodologies <u>ASSOC</u> No	s,resulting in no IATED FINDING	ormal rati	o when de	
Urine reabsorption Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necro Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (ro Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an ine STIMATED GLOMERL CKD STAGE	(e.g. ureter colo ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. 2: creased urea syn urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ren sis (acetoacetate creased BUN/crea apy (interferes v LAR FILTERATIO	stomy) creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creating reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu V RATE: DESCRIPTION	e in creatini ement).	ine) (e.g. obstructive cellular fluid). lar secretion of urea ne). ine with certain met	e uropathy) I. hodologies ASSOC No Prese	s,resulting in no	ormal rati	o when de	
Urine reabsorption Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necro Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Rhabdomyolysis (re Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an ine CERD STAGE G1 G2	(e.g. ureter cold ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. creased urea syn urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop reason creased BUN/creason LAR FILTERATION Nor Nor Nor Nor	stomy) creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creating reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu. NATE: DESCRIPTION mal kidney function dney damage with ormal or high GFR id decrease in GFR	e in creatini ement).	ine) (e.g. obstructive cellular fluid). lar secretion of urea ne). ine with certain met nL/min/1.73m2) >90 >90 60 -89	e uropathy) I. hodologies ASSOC No Prese	s,resulting in no IATED FINDING proteinuria nce of Protein ,	ormal rati	o when de	
7. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. NCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (<1 1. Acute tubular necro 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (ro 3. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an inc 2. Cephalosporin ther ESTIMATED GLOMERU G1 G2	(e.g. ureter cold ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. creased urea syn urea rather than monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ren- sis (acetoacetate creased BUN/crea sis (acetoacetate creased BUN/crea by (interferes of LAR FILTERATIO Nor Kin Model Model	stomy) creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) ASED CREATININE: conversion of creating reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu V RATE: DESCRIPTION mal kidney function dney damage with prmal or high GFR	e in creatini ement).	ine) (e.g. obstructive cellular fluid). lar secretion of urea ne). ine with certain met nL/min/1.73m2) >90 >90	e uropathy) I. hodologies ASSOC No Prese	s,resulting in no IATED FINDING proteinuria nce of Protein ,	ormal rati	o when de	





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

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







	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant	obiology) ME	n Chopra D (Pathology) It Pathologist
NAME	: Mr. SAMPURAN SINGH BAINS		
AGE/ GENDER	: 69 YRS/MALE	PATIENT ID	: 1743531
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012502030005
REFERRED BY	:	REGISTRATION DATE	: 03/Feb/2025 08:21 AM
BARCODE NO.	: 01524847	COLLECTION DATE	: 03/Feb/2025 08:31AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 03/Feb/2025 10:56AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	LA 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



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

MBBS, MD (PATHOLOGY)

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Dr. Vinay Chopra



Dr. Yugam Chopra

	MD (Pathology & I Chairman & Const		MD (Pathology) CEO & Consultant Pathologist		
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BARCODE NO.	: 01524847	COL	LECTION DATE	: 03/Feb/2025 08:31AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REP	ORTING DATE	:03/Feb/202509:42AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
		CLINICAL PA	THOLOGY		
	URINE ROU	UTINE & MICRO	SCOPIC EXAMINA	ATION	
PHYSICAL EXAMIN					
QUANTITY RECIEV		10	ml		
COLOUR	TANCE SPECTROPHOTOMETRY	PALE YELLOW	N	PALE YELLOW	
TRANSPARANCY	TANCE SPECINOPHOTOWERN	CLEAR		CLEAR	
SPECIFIC GRAVITY		1.02		1.002 - 1.030	
by DIP STICK/REFLEC CHEMICAL EXAMI	TANCE SPECTROPHOTOMETRY				
REACTION		ACIDIC			
-	TANCE SPECTROPHOTOMETRY				
PROTEIN by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
SUGAR by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
рH	TANCE SPECTROPHOTOMETRY	5.5		5.0 - 7.5	
BILIRUBIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
NITRITE	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)	
ASCORBIC ACID	TANCE SPECTROPHOTOMETRY TANCE SPECTROPHOTOMETRY AMINATION	NEGATIVE (-v	ve)	NEGATIVE (-ve)	
RED BLOOD CELLS		NEGATIVE (-v	/HPF	0 - 3	





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



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

NAME	: Mr. SAMPURAN SINGH BAINS	5		
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
by MICROSCOPY ON O	CENTRIFUGED URINARY SEDIMENT			
PUS CELLS		2-3	/HPF	0 - 5
EPITHELIAL CELLS	CENTRIFUGED URINARY SEDIMENT S CENTRIFUGED URINARY SEDIMENT	1-2	/HPF	ABSENT
CRYSTALS		NEGATIVE (-ve)		NEGATIVE (-ve)

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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