

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mr. SURJIT SINGH			
AGE/ GENDER	: 53 YRS/MALE		PATIENT ID	: 1078644
COLLECTED BY	:		REG. NO./LAB NO.	: 012410270012
REFERRED BY	:		REGISTRATION DATE	: 27/Oct/2024 07:53 AM
BARCODE NO.	:01519624		COLLECTION DATE	: 27/Oct/2024 07:55AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 27/Oct/2024 09:02AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
	SWAST	HYA WEI	LINESS PANEL: 1.0	D
	COMP	PLETE BLO	OOD COUNT (CBC)	
RED BLOOD CELLS	<u>S (RBCS) COUNT AND INDICES</u>			
HAEMOGLOBIN (H	B)	13.5	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL ((RBC) COUNT	4.4	Millions	/cmm 3.50 - 5.00
	OCUSING, ELECTRICAL IMPEDENCE	49.9	%	
	UNIE (PCV) AUTOMATED HEMATOLOGY ANALYZER	42.2	70	40.0 - 54.0
	AR VOLUME (MCV) AUTOMATED HEMATOLOGY ANALYZER	96	fL	80.0 - 100.0
MEAN CORPUSCUL	AR HAEMOGLOBIN (MCH)	30.8	pg	27.0 - 34.0
MEAN CORPUSCUL	AUTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC)	32.1	g/dL	32.0 - 36.0
	AUTOMATED HEMATOLOGY ANALYZER	12.9	%	11.00 - 16.00
	AUTOMATED HEMATOLOGY ANALYZER			11.00 - 10.00
	SUTION WIDTH (RDW-SD) AUTOMATED HEMATOLOGY ANALYZER	46.2	fL	35.0 - 56.0
MENTZERS INDEX		21.82	RATIO	BETA THALASSEMIA TRAIT: <
by CALCULATED				13.0 IRON DEFICIENCY ANEMIA:
				>13.0
GREEN & KING INI by calculated	DEX	28.25	RATIO	BETA THALASSEMIA TRAIT:<= 65.0
.,				IRON DEFICIENCY ANEMIA: >
WHITE BLOOD CE	TIS (WRCS)			65.0
IOTAL LEUCOCYTI		4940	/cmm	4000 - 11000
by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY		/ chini	
	BLOOD CELLS (nRBCS) rt hematology analyzer	NIL		0.00 - 20.00
NUCLEATED RED H	BLOOD CELLS (nRBCS) %	NIL	%	< 10 %
by CALCOLATED BY A	AUTOMATED HEMATOLOGY ANALYZER			





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

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







Dr. Yugam Chopra

MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. SURJIT SINGH AGE/ GENDER : 53 YRS/MALE **PATIENT ID** :1078644 **COLLECTED BY** :012410270012 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 27/Oct/2024 07:53 AM **BARCODE NO.** :01519624 **COLLECTION DATE** : 27/Oct/2024 07:55AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 27/Oct/2024 09:02AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC)** NEUTROPHILS 60 % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 24% 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 5 % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 11 % 2 - 12by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE LEUKOCYTES (WBC) COUNT** ABSOLUTE NEUTROPHIL COUNT 2964 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 1186 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 247/cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 543 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 324000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.33 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 10 fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 30000 - 90000 PLATELET LARGE CELL COUNT (P-LCC) 84000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE % PLATELET LARGE CELL RATIO (P-LCR) 2611.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 15.0 - 17.0 15.8% by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

Dr. Vinay Chopra

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Test Name	Value	Unit	Biological Reference interval





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







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LIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 27/Oct/2024 09:28AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
est Name		Value	Unit	Biological Reference interval
VTERPRETATION: . ESR is a non-speciti nmune disease, but	does not tell the health pract	sult often indicates the itioner exactly where the	e inflammation is in the	e body or what is causing it.
TERPRETATION: ESR is a non-speci nmune disease, but An ESR can be affe s C-reactive protein This test may also ystemic lupus eryth ONDITION WITH LO Iow ESR can be see polycythaemia), sign	fic test because an elevated re c does not tell the health pract ected by other conditions besic be used to monitor disease ac ematosus W ESR en with conditions that inhibit	sult often indicates the itioner exactly where the des inflammation. For th ctivity and response to the the normal sedimentation count (leucocytosis), a	e inflammation is in the is reason, the ESR is ty nerapy in both of the a on of red blood cells, s	ion associated with infection, cancer and auto-





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Test Name		Value	Unit	Biological Reference interval
	CI	INICAL CHEMIS	TRY/BIOCHEMIST	'RY
		GLUCOSE	FASTING (F)	

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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Fest Name		Value	Unit	Biological Reference interval
		LIPID PROFILI	E : BASIC	
CHOLESTEROL TO	TAL: SERUM	227.89 ^H	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		~~ i .00	0	BORDERLINE HIGH: 200.0 -
				239.0 HIGH CHOLESTEROL: > OR =
				240.0
RIGLYCERIDES: S		200.96 ^H	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSP	PHATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 - 199.0
				HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
IDL CHOLESTERO	L (DIRECT): SERUM	53.38	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0
., 01110111				60.0
				HIGH HDL: $> OR = 60.0$
DL CHOLESTEROI by CALCULATED, SPE		134.32 ^H	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.
<i>xy ci</i> .2 <i>cc2ii</i> .2 <i>b, ci 2</i>				BORDERLINE HIGH: 130.0 -
				159.0
				HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLEST	FEROL: SERUM	174.51 ^H	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPE	CTROPHOTOMETRY		-	ABOVE OPTIMAL: 130.0 - 159.
				BORDERLINE HIGH: 160.0 - 189.0
				HIGH: 190.0 - 219.0
		40.10		VERY HIGH: $> OR = 220.0$
LDL CHOLESTER(by CALCULATED, SPE		40.19	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER		656.74	mg/dL	350.00 - 700.00
by CALCULATED, SPE CHOLESTEROL/HD		4.27	RATIO	LOW RISK: 3.30 - 4.40
by CALCULATED, SPE		1.61	INATIO	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 by CALCULATED, SPE		2.52	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	3.76	RATIO	3.00 - 5.00

INTERPRETATION:

1. Measurements in the same patient can show physiological analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.

2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.

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

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





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Test Name		Value	Unit	Biological Reference interval
	LIVER	FUNCTIO	N TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SH	: SERUM PECTROPHOTOMETRY	0.83	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.13	mg/dL	0.00 - 0.40
-	CT (UNCONJUGATED): SERUM	0.7	mg/dL	0.10 - 1.00
SGOT/AST: SERUM		21.6	U/L	7.00 - 45.00
SGPT/ALT: SERUM	[/RIDOXAL PHOSPHATE	22.3	U/L	0.00 - 49.00
AST/ALT RATIO: S	ERUM	0.97	RATIO	0.00 - 46.00
ALKALINE PHOSPH		80.56	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTROF	L TRANSFERASE (GGT): SERUM PHTOMETRY	50.34	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO	SERUM	6.83	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		4.28	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	1	2.55	gm/dL	2.30 - 3.50
A : G RATIO: SERUN	M	1.68	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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	Dr. Vinay Chop	ra I Dr. Yugai	m Chopra

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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MBBS, MD (PATHOLOGY)







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Test Name		Value	Unit	Biological Reference interval	
	KIDN	EY FUNCTION	TEST (COMPLETE)		
UREA: SERUM	IATE DEHYDROGENASE (GLDH)	39.36	mg/dL	10.00 - 50.00	
CREATININE: SER		1.18	mg/dL	0.40 - 1.40	
by ENZYMATIC, SPEC	CTROPHOTOMETERY ROGEN (BUN): SERUM	18.39	mg/dL	7.0 - 25.0	
by CALCULATED, SPECTROPHOTOMETRY					
BLOOD UREA NITH RATIO: SERUM	ROGEN (BUN)/CREATININE	15.58	RATIO	10.0 - 20.0	
by CALCULATED, SPE					
UREA/CREATININ	E RATIO: SERUM	33.36	RATIO		
URIC ACID: SERUM	1	4.51	mg/dL	3.60 - 7.70	
by URICASE - OXIDAS CALCIUM: SERUM	SE PEROXIDASE	9.91	mg/dL	8.50 - 10.60	
by ARSENAZO III, SPE	ECTROPHOTOMETRY		ling/ uL	8.30 - 10.00	
PHOSPHOROUS: SE	ERUM DATE, SPECTROPHOTOMETRY	3.24	mg/dL	2.30 - 4.70	
ELECTROLYTES					
SODIUM: SERUM		140.2	mmol/L	135.0 - 150.0	
by ISE (ION SELECTIV POTASSIUM: SERU		4.3	mmol/L	3.50 - 5.00	
by ISE (ION SELECTIV	/E ELECTRODE)		IIIII01/L		
CHLORIDE: SERUN by ISE (ION SELECTIV		105.15	mmol/L	90.0 - 110.0	
	IERULAR FILTERATION RATE				
	IERULAR FILTERATION RATE	73.8			

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 Name		Value	Un	it	Biologie	cal Refere	nce interv
8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia	(e.g. ureter colostomy) ass (subnormal creatinin tetracycline, glucocortico 0:1) WITH ELEVATED CRE/ (BUN rises disproportior superimposed on renal d 0:1) WITH DECREASED BL	oids) ATININE LEVELS: Nately more than creatir isease.	nine) (e.g. obstructive	e uropathy).			
8. Reduced muscle m 9. Certain drugs (e.g. 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 GLOMERI CKD STAGE	ass (subnormal creatinin- tetracycline, glucocortico 0:1) WITH ELEVATED CREA (BUN rises disproportior superimposed on renal d 0:1) WITH DECREASED BL osis. Id starvation. 2: creased urea synthesis. urea rather than creatini monemias (urea is virtua f inappropiate antidiuret 0:1) WITH INCREASED CR oy (accelerates conversic eleases muscle creatinine who develop renal failure sis (acetoacetate causes creased BUN/creatinine r apy (interferes with creat LAR FILTERATION RATE: 	and s) ATININE LEVELS: hately more than creating isease. IN : In e diffuses out of extra ly absent in blood). ic harmone) due to tube EATININE: n of creatine to creating e). e. false increase in creating e). e). e). e). e). e). e). e).	icellular fluid). ular secretion of urea ine). nine with certain met <u>mL/min/1.73m2)</u> >90	hodologies,re ASSOCIAT	ED FINDINGS oteinuria	mal ratio w	hen dehydr
B. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Nuscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERL CKD STAGE	ass (subnormal creatinin- tetracycline, glucocortico 0:1) WITH ELEVATED CREA (BUN rises disproportior superimposed on renal d 0:1) WITH DECREASED BL osis. Id starvation. 2. creased urea synthesis. urea rather than creatini monemias (urea is virtua f inappropiate antidiuret 0:1) WITH INCREASED CR oy (accelerates conversic eleases muscle creatinine who develop renal failure sis (acetoacetate causes creased BUN/creatinine r apy (interferes with creat LAR FILTERATION RATE: 	and s) ATININE LEVELS: hately more than creating isease. IN : In e diffuses out of extra ly absent in blood). ic harmone) due to tube EATININE: n of creatine to creating e). e. false increase in creating e). e). e). e). e). e). e). e).	icellular fluid). ular secretion of urea ine). nine with certain met mL/min/1.73m2)	hodologies,re ASSOCIAT No pr Presence	ED FINDINGS oteinuria of Protein ,		hen dehydr
B. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rabdomyolysis (r Nuscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERU CKD STAGE G1 G2	ass (subnormal creatinin- tetracycline, glucocortico 0:1) WITH ELEVATED CREA (BUN rises disproportior superimposed on renal d 0:1) WITH DECREASED BL osis. Id starvation. 2. creased urea synthesis. urea rather than creatini monemias (urea is virtua f inappropiate antidiuret 0:1) WITH INCREASED CR oy (accelerates conversic eleases muscle creatinine who develop renal failure sis (acetoacetate causes creased BUN/creatinine r apy (interferes with creat LAR FILTERATION RATE: 	bids) ATININE LEVELS: bately more than creating isease. IN : IN : In e diffuses out of extra Ily absent in blood). ic harmone) due to tube EATININE: n of creatine to creating e).	incellular fluid). ular secretion of urea ine). hine with certain met <u>mL/min/1.73m2) >90 >90</u>	hodologies,re ASSOCIAT No pr Presence	ED FINDINGS oteinuria		hen dehydr
B. Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Inherited hyperam SIADH (syndrome of Nuscular patients INAPPROPIATE RATIO Liabetic ketoacido should produce an in Cephalosporin ther ESTIMATED GLOMERL G1 G2 G3a	ass (subnormal creatinin- tetracycline, glucocortico 0:1) WITH ELEVATED CREA (BUN rises disproportior superimposed on renal d 0:1) WITH DECREASED BL osis. Id starvation. 2: creased urea synthesis. urea rather than creatini monemias (urea is virtua f inappropiate antidiuret 0:1) WITH INCREASED CR oy (accelerates conversic eleases muscle creatinine who develop renal failuret sis (acetoacetate causes creased BUN/creatinine r apy (interferes with creat LAR FILTERATION RATE: DESCRI Normal kidne Kidney dam normal or Mild decreat	bids) ATININE LEVELS: bately more than creating isease. IN : In e diffuses out of extra Ily absent in blood). ic harmone) due to tube EATININE: n of creatine to creating e). e). false increase in creating e).	incellular fluid). ular secretion of urea ine). hine with certain met <u>mL/min/1.73m2) >90 >90 60 -89</u>	hodologies,re ASSOCIAT No pr Presence	ED FINDINGS oteinuria of Protein ,		hen dehydr
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (Rhabdomyolysis (r Muscular patients INAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther ESTIMATED GLOMERI G1 G2 	ass (subnormal creatinin- tetracycline, glucocortico 0:1) WITH ELEVATED CREA (BUN rises disproportior superimposed on renal d 0:1) WITH DECREASED BL osis. Id starvation. 2. creased urea synthesis. urea rather than creatini monemias (urea is virtua f inappropiate antidiuret 0:1) WITH INCREASED CR oy (accelerates conversic eleases muscle creatinine who develop renal failure sis (acetoacetate causes creased BUN/creatinine r apy (interferes with creat LAR FILTERATION RATE: 	and sides) ATININE LEVELS: hately more than creating isease. IN : In e diffuses out of extra Ily absent in blood). ic harmone) due to tube EATININE: n of creatine to creating e. false increase in GFR ise in GFR ise in GFR rease in GFR	incellular fluid). ular secretion of urea ine). hine with certain met <u>mL/min/1.73m2) >90 >90</u>	hodologies,re ASSOCIAT No pr Presence	ED FINDINGS oteinuria of Protein ,		hen dehydr





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









	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant	biology) MI	m Chopra D (Pathology) nt Pathologist
NAME	: Mr. SURJIT SINGH		
AGE/ GENDER	: 53 YRS/MALE	PATIENT ID	: 1078644
COLLECTED BY	:	REG. NO./LAB NO.	: 012410270012
REFERRED BY	:	REGISTRATION DATE	: 27/Oct/2024 07:53 AM
BARCODE NO.	: 01519624	COLLECTION DATE	: 27/Oct/2024 07:55AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 27/Oct/2024 10:27AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBAI	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)







	Dr. Vinay Cho MD (Pathology & Chairman & Cons	Microbiology)	Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist		
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AGE/ GENDER	: 53 YRS/MALE	PATIENT I	D	: 1078644	
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BARCODE NO.	:01519624	COLLECTI	ON DATE	: 27/Oct/2024 07:55AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTIN	IG DATE	: 27/Oct/2024 09:32AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
		CLINICAL PATHO	LOGY		
	URINE RO	UTINE & MICROSCOP		ATION	
PHYSICAL EXAMIN	ATION				
QUANTITY RECIEVE		10	ml		
COLOUR	TANCE SPECTROPHOTOMETRY	AMBER YELLOW		PALE YELLOW	
by DIP STICK/REFLECT	TANCE SPECTROPHOTOMETRY				
TRANSPARANCY by DIP STICK/REFLECT	TANCE SPECTROPHOTOMETRY	CLEAR		CLEAR	
SPECIFIC GRAVITY		<=1.005		1.002 - 1.030	
by DIP STICK/REFLECT CHEMICAL EXAMIN	TANCE SPECTROPHOTOMETRY				
REACTION	MIION	ACIDIC			
by DIP STICK/REFLECT	TANCE SPECTROPHOTOMETRY				
PROTEIN by DIP STICK/REFLECT	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
SUGAR		Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECT pH	TANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5	
	TANCE SPECTROPHOTOMETRY	<-3.0		5.0 - 7.5	
BILIRUBIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
NITRITE		Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECT UROBILINOGEN	TANCE SPECTROPHOTOMETRY.	Normal	EU/dL	0.2 - 1.0	
	TANCE SPECTROPHOTOMETRY	Normai	EU/ UL	0.2 - 1.0	
KETONE BODIES	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
BLOOD		Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECT ASCORBIC ACID	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)	
	TANCE SPECTROPHOTOMETRY	NEGATIVE (-Ve)		MEGATIVE (-ve)	
MICROSCOPIC EXA					
RED BLOOD CELLS	(RBCs)	NEGATIVE (-ve)	/HPF	0 - 3	

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

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

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

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REFERRED BY			REGISTRATION DATE		
BARCODE NO.			COLLECTION DATE		
CLIENT CODE.			REPORTING DATE		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AI	MBALA CANT	Т		
Test Name		Value	Unit	Biological Reference interval	
by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT				
PUS CELLS by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	0 - 5	

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT				
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	0-1	/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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V DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)

