

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



KOS Diagnostic Lab (A Unit of KOS Healthcare)

	Dr. Vinay Cho MD (Pathology & Chairman & Consi	Microbiology)	Dr. Yugam MD (I CEO & Consultant F	Pathology)
NAME	: Mr. BHUPINDER SINGH	0		
AGE/ GENDER	: 29 YRS/MALE	1	PATIENT ID	: 1642043
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012410130025
REFERRED BY	:		REGISTRATION DATE	: 13/Oct/2024 10:37 AM
BARCODE NO.	: 01518800		COLLECTION DATE	: 13/Oct/2024 10:44AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	1	REPORTING DATE	: 13/Oct/2024 10:56AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	SW	/ASTHYA WE	LLNESS PANEL: G	
	C	OMPLETE BLO	OD COUNT (CBC)	
RED BLOOD CELLS (R	BCS) COUNT AND INDICES			
HAEMOGLOBIN (HB)		13.1	gm/dL	12.0 - 17.0
by CALORIMETRIC			Millions/cr	nm 3.50 - 5.00
RED BLOOD CELL (RB by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE	5.18 ^H		3.50 - 5.00
PACKED CELL VOLUN	E (PCV) UTOMATED HEMATOLOGY ANALYZE	40.7	%	40.0 - 54.0
MEAN CORPUSCULA	R VOLUME (MCV)	78.5 ^L	fL	80.0 - 100.0
	utomated hematology analyze R HAEMOGLOBIN (MCH)	25.2 ^L	pg	27.0 - 34.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZE	R		
	R HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZE	32.1 R	g/dL	32.0 - 36.0
RED CELL DISTRIBUT	ON WIDTH (RDW-CV)	14	%	11.00 - 16.00
	JTOMATED HEMATOLOGY ANALYZE ON WIDTH (RDW-SD)	R 41.2	fL	35.0 - 56.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZE			
MENTZERS INDEX		15.15	RATIO	BETA THALASSEMIA TRAIT: < 13.0
GREEN & KING INDE	K	21.14	RATIO	IRON DEFICIENCY ANEMIA: >13.0 BETA THALASSEMIA TRAIT:<= 65.0
by CALCULATED				IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS				
TOTAL LEUCOCYTE CO	DUNT (TLC) by sf cube & microscopy	10540	/cmm	4000 - 11000
NUCLEATED RED BLC	OD CELLS (nRBCS)	NIL		0.00 - 20.00
by AUTOMATED 6 PAR NUCLEATED RED BLC	T HEMATOLOGY ANALYZER	NIL	%	< 10 %
	UD GELLS (TROGS) % UTOMATED HEMATOLOGY ANALYZE		/0	< 10 /0
DIFFERENTIAL LEUCO	<u>CYTE COUNT (DLC)</u>			





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Test Name	Value	Unit	Biological Reference interval
LYMPHOCYTES	19 ^L	%	20 - 40
by FLOW CYTOMETRY BY SF CUBE & MIC EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MIC	1	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MIC	5	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MIC		%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUN	<u>IT</u>		
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MIN	7905 ^H	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MIC	2003	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MIC	105	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MIC	527 CROSCOPY	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MIC		/cmm	0 - 110
PLATELETS AND OTHER PLATELET PR	EDICTIVE MARKERS.		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRI	260000 CAL IMPEDENCE	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRI	0.31 CAL IMPEDENCE	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRI	12 CAL IMPEDENCE	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTR		/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRI	39.3 CAL IMPEDENCE	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PD)	W) 16.8	%	15.0 - 17.0





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Test Name		Value	Unit	Biological Reference interval
	GL	YCOSYLATED HAEMOGL	OBIN (HBA1C)	
GLYCOSYLATED HAEMO WHOLE BLOOD		14.8 ^H	%	4.0 - 6.4
ESTIMATED AVERAGE F	MANCE LIQUID CHROMATOGRAPHY) PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY)	378.06 ^H	mg/dL	60.00 - 140.00
	AS PER AMERICAN DIAB	ETES ASSOCIATION (ADA):		
	FERENCE GROUP	GLYCOSYLATED HE	Moglogib (Hbaic) ii	n %
	etic Adults >= 18 years		<5.7	
	Risk (Prediabetes)		7 - 6.4	
Diag	gnosing Diabetes		>= 6.5	
			> 19 Years	
Theses Lin		Goals of Therapy:	< 7.0	
inerapeutic	goals for glycemic control	Actions Suggested:	>8.0	
		Age < 19 Years		
		Goal of therapy:	<7.5	

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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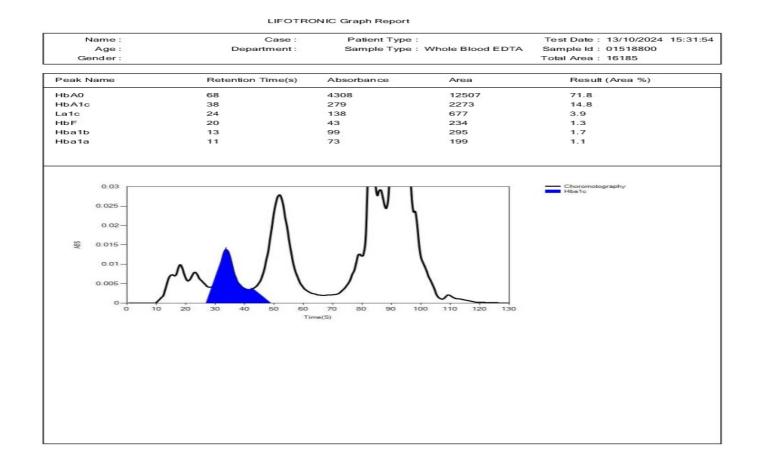
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Test Name		Value Unit	Biological Reference interval





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANT	Г	
Test Name		Value	Unit	Biological Reference interval
by RED CELL AGGRE INTERPRETATION: 1. ESR is a non-specif immune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythe	MENTATION RATE (ESR) <i>GATION BY CAPILLARY PHOTOME</i> ic test because an elevated resu does not tell the health practiti cted by other conditions beside be used to monitor disease acti ematosus	33 ^H Ilt often indicates oner exactly whe s inflammation. F	re the inflammation is in the for this reason, the ESR is ty	
(polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactiv	n with conditions that inhibit th	count (leucocytos ESR. rs of inflammatio	is) , and some protein abno n.	uch as a high red blood cell count rmalities. Some changes in red cell shape (such

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 2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation.
 3. CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.
 4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
 5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
 6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspiring cortisone, and quipino may decrease it. aspirin, cortisone, and quinine may decrease it



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT Value	Unit	Biological Reference interval
CLIENT ADDRESS		Value	/BIOCHEMISTR	

intolerant or prediabetic. A fasting and post-prandial blood

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.
 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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SO 9001 : 2008 CERTIFIED LAB			EXCELLENCE IN HEALTHCARE &	DIAGNOSTICS
	Dr. Vinay Chopra MD (Pathology & Microbi Chairman & Consultant P		Dr. Yugam MD (CEO & Consultant	Pathology)
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Test Name	Va	lue	Unit	Biological Reference interval
		PID PROFIL	F : BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP		30.25 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE		3.82	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERU by SELECTIVE INHIBITION	JM 67	1.54	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOM		51.95 ^H	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 CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOM		58.71 ^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189. HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOME		6.76	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOME	54	14.32	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUN by CALCULATED, SPECTROPHOTOME	1 3.	74	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOME		47	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
		gho	fra	

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Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD		1.36 ^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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			N TEST (COMPLETE)	
BILIRUBIN TOTAL: SI by DIAZOTIZATION, SF	ERUM <i>pectrophotometry</i>	0.45	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	CONJUGATED): SERUM	0.21	mg/dL	0.00 - 0.40
	(UNCONJUGATED): SERUM	0.24	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	RIDOXAL PHOSPHATE	21.34	U/L	7.00 - 45.00
SGPT/ALT: SERUM	RIDOXAL PHOSPHATE	25.72	U/L	0.00 - 49.00
AST/ALT RATIO: SER	UM	0.83	RATIO	0.00 - 46.00
ALKALINE PHOSPHA		130	U/L	40.0 - 150.0
	TRANSFERASE (GGT): SERUM	26	U/L	0.00 - 55.0
TOTAL PROTEINS: SE	RUM	7.62	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		5.13	gm/dL	3.50 - 5.50
GLOBULIN: SERUM		2.49	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPE		2.06 ^H	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)



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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).

PROGNOSTIC SIGNIFICANCE:

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



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Test Name		Value	Unit	Biological Reference interval	
	кі	DNEY FUNCTIO	N TEST (COMPLETE)		
UREA: SERUM		24.66	mg/dL	10.00 - 50.00	
-	NATE DEHYDROGENASE (GLDH)				
CREATININE: SERUN by ENZYMATIC, SPEC		0.68	mg/dL	0.40 - 1.40	
	DGEN (BUN): SERUM	11.52	mg/dL	7.0 - 25.0	
by CALCULATED, SPECTROPHOTOMETRY					
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM		16.94	RATIO	10.0 - 20.0	
by CALCULATED, SPE	ECTROPHOTOMETRY				
UREA/CREATININE F		36.26	RATIO		
by CALCULATED, SPECTROPHOTOMETRY URIC ACID: SERUM		3.65	mg/dL	3.60 - 7.70	
by URICASE - OXIDAS	SE PEROXIDASE	5.05	ing/uL	3.00 - 7.70	
CALCIUM: SERUM		9.86	mg/dL	8.50 - 10.60	
by ARSENAZO III, SPECTROPHOTOMETRY PHOSPHOROUS: SERUM		3.4	mg/dL	2.30 - 4.70	
by PHOSPHOMOLYBE	DATE, SPECTROPHOTOMETRY	0.1	ilig/ dL	2.00 1.70	
<u>ELECTROLYTES</u>					
SODIUM: SERUM		142.6	mmol/L	135.0 - 150.0	
by ISE (ION SELECTIVE ELECTRODE) POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)		4.32	mmol/L	3.50 - 5.00	
			minol/ E		
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)		106.95	mmol/L	90.0 - 110.0	
	RULAR FILTERATION RATE				
	RULAR FILTERATION RATE	129			
(eGFR): SERUM		127			
by CALCULATED					

INTERPRETATION:

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.



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



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





	Dr. Vinay Chop MD (Pathology & Mi Chairman & Consult	crobiology)	Dr. Yugam Chopra MD (Pathology) & Consultant Pathologist	
NAME	: Mr. BHUPINDER SINGH			
AGE/ GENDER	: 29 YRS/MALE	PATIENT ID	: 1642043	
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	:0124101300)25
REFERRED BY		REGISTRATION DA		
BARCODE NO.	: 01518800	COLLECTION DATI		
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	2 : 13/Oct/2024	12:24PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT		
Test Name		Value Uni	it Biolog	jical Reference interval
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia 	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine productio tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LE a (BUN rises disproportionately more	/ELS:	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 GLOMERL OKD STAGE	(e.g. ureter colostomy) ass (subnormal creatinine productio tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATININE LE a (BUN rises disproportionately more superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. urea rather than creatinine diffuses monemias (urea is virtually absent i of inappropiate antidiuretic harmone (0:1) WITH INCREASED CREATININE: py (accelerates conversion of creati eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false increatic creased BUN/creatinine ratio). rapy (interferes with creatinine meas JLAR FILTERATION RATE:	VELS: than creatinine) (e.g. obstructive out of extracellular fluid). h blood). due to tubular secretion of urea. he to creatinine). ase in creatinine with certain methesurement). GFR (mL/min/1.73m2)	hodologies,resulting in n	
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 GLOMERL	(e.g. ureter colostomy) ass (subnormal creatinine productio tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LE a (BUN rises disproportionately more superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. furea rather than creatinine diffuses monemias (urea is virtually absent i of inappropiate antidiuretic harmone 10:1) WITH INCREASED CREATININE: py (accelerates conversion of creati eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false increatic creased BUN/creatinine ratio). apy (interferes with creatinine meas JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with	VELS: than creatinine) (e.g. obstructive out of extracellular fluid). h blood). due to tubular secretion of urea. he to creatinine). ase in creatinine with certain methesurement). GFR (mL/min/1.73m2)	hodologies,resulting in n	<u>s</u>
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	(e.g. ureter colostomy) ass (subnormal creatinine production tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATININE LET a (BUN rises disproportionately more superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. and starvation. e. creased urea synthesis. furea rather than creatinine diffuses monemias (urea is virtually absent in of inappropiate antidiuretic harmone (0:1) WITH INCREASED CREATININE: py (accelerates conversion of creatine eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false increat creased BUN/creatinine ratio). apy (interferes with creatinine meas JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR	VELS: a than creatinine) (e.g. obstructive a out of extracellular fluid). n blood). a) due to tubular secretion of urea. b to creatinine). ase in creatinine with certain methers surement). Surement). >90 >90	hodologies,resulting in n ASSOCIATED FINDING No proteinuria	S
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal 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 GLOMERL G1 G2 	(e.g. ureter colostomy) ass (subnormal creatinine production tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATININE LET a (BUN rises disproportionately more superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. and starvation. e. creased urea synthesis. furea rather than creatinine diffuses monemias (urea is virtually absent in of inappropiate antidiuretic harmone (0:1) WITH INCREASED CREATININE: py (accelerates conversion of creatine eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false increat creased BUN/creatinine ratio). apy (interferes with creatinine meas JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR Mild decrease in GFR	VELS: a than creatinine) (e.g. obstructive a out of extracellular fluid). n blood). a) due to tubular secretion of urea. b to creatinine). asse in creatinine with certain mether surement). Surement). 90 60 - 89	hodologies,resulting in n ASSOCIATED FINDING No proteinuria Presence of Protein	S
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 GLOMERU CKD STAGE G1 G2	(e.g. ureter colostomy) ass (subnormal creatinine production tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATININE LET a (BUN rises disproportionately more superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. and starvation. e. creased urea synthesis. furea rather than creatinine diffuses monemias (urea is virtually absent in of inappropiate antidiuretic harmone (0:1) WITH INCREASED CREATININE: py (accelerates conversion of creatine eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false increat creased BUN/creatinine ratio). apy (interferes with creatinine meas JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR	VELS: a than creatinine) (e.g. obstructive a out of extracellular fluid). n blood). a) due to tubular secretion of urea. b) due to tubular secretion of urea. ase in creatinine). asse in creatinine with certain mether surement). 90 60 - 89 R 30-59	hodologies,resulting in n ASSOCIATED FINDING No proteinuria Presence of Protein	S



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

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









	Dr. Vinay Chop MD (Pathology & Mid Chairman & Consulta	crobiology)	ugam Chopra MD (Pathology) sultant Pathologist
NAME	: Mr. BHUPINDER SINGH		
AGE/ GENDER	: 29 YRS/MALE	PATIENT ID	: 1642043
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	:012410130025
REFERRED BY	:	REGISTRATION DA	TE : 13/Oct/2024 10:37 AM
BARCODE NO.	:01518800	COLLECTION DATE	E : 13/Oct/2024 10:44AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 13/Oct/2024 12:24PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMI	BALA CANTT	
Test Name		Value Uni	t 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

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	Dr. Vinay Ch MD (Pathology & Chairman & Cons		Dr. Yugam MD CEO & Consultant	(Pathology)	
NAME	: Mr. BHUPINDER SINGH				
AGE/ GENDER	: 29 YRS/MALE	PA	FIENT ID	: 1642043	
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 13/Oct/2024 11:41AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A				
	, , ,				
Test Name		Value	Unit	Biological Reference interval	
		CLINICAL PA	THOLOGY		
		OUTINE & MICRO	SCOPIC EXAMINAT	TION	
PHYSICAL EXAMINA					
		10	ml		
QUANTITY RECIEVED by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		10	ml		
COLOUR		AMBER YELLOW		PALE YELLOW	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY					
TRANSPARANCY		HAZY		CLEAR	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY SPECIFIC GRAVITY		1.01		1.002 - 1.030	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY					
CHEMICAL EXAMINA	ATION				
REACTION		ACIDIC			
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	1+		NEGATIVE (-ve)	
	CTANCE SPECTROPHOTOMETRY	1+		NEGATIVE (-ve)	
SUGAR		2+		NEGATIVE (-ve)	
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5	
	TANCE SPECTROPHOTOMETRY	<=3.0		3.0 - 7.3	
BILIRUBIN		Negative		NEGATIVE (-ve)	
	TANCE SPECTROPHOTOMETRY	Dealthus			
NITRITE by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY.	Positive		NEGATIVE (-ve)	
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0	
	TANCE SPECTROPHOTOMETRY				
KETONE BODIES	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
BLOOD		TRACE		NEGATIVE (-ve)	
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY				
ASCORBIC ACID		NEGATIVE (-ve	2)	NEGATIVE (-ve)	
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY				

MICROSCOPIC EXAMINATION



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



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

NAME	: Mr. BHUPINDER SINGH			
AGE/ GENDER	: 29 YRS/MALE	PATIENT	ID	: 1642043
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CLIENT CODE.	: KOS DIAGNOSTIC LAB			: 13/Oct/2024 11:41AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
RED BLOOD CELLS (F	RBCs) CENTRIFUGED URINARY SEDIMENT	3-4	/HPF	0 - 3
PUS CELLS	CENTRIFUGED URINARY SEDIMENT	8-10	/HPF	0 - 5
EPITHELIAL CELLS	CENTRIFUGED URINARY SEDIMENT	1-2	/HPF	ABSENT
CRYSTALS by MICROSCOPY ON (CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS by MICROSCOPY ON O	CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA by MICROSCOPY ON (CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS by MICROSCOPY ON O	CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAC	GINALIS (PROTOZOA)	ABSENT		ABSENT

TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

End Of Report





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