

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultan	obiology)		Pathology)
JAME : M	r. RAVINDER SAINI			
GE/ GENDER : 46	YRS/MALE]	PATIENT ID	: 1659822
COLLECTED BY :]	REG. NO./LAB NO.	: 012411040002
EFERRED BY :]	REGISTRATION DATE	:04/Nov/202406:20AM
	520007		COLLECTION DATE	: 04/Nov/2024 06:34AM
)S DIAGNOSTIC LAB 49/1, NICHOLSON ROAD, AMB/		REPORTING DATE	: 04/Nov/2024 07:52AM
Fest Name		Value	Unit	Biological Reference interval
	SWASTI	HYA WEI	LNESS PANEL: 1.0	
	COMP	LETE BLO	OOD COUNT (CBC)	
	<u>CS) COUNT AND INDICES</u>			
HAEMOGLOBIN (HB)		12.9	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC)		5.46 ^H	Millions/	cmm 3.50 - 5.00
by HYDRO DYNAMIC FOCUS	ING, ELECTRICAL IMPEDENCE (PCV)	41.4	%	40.0 - 54.0
MEAN CORPUSCULAR VO		75.8 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR H		23.6 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR H	ATED HEMATOLOGY ANALYZER EMOGLOBIN CONC. (MCHC) ATED HEMATOLOGY ANALYZER	31.2 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTIO		13	%	11.00 - 16.00
RED CELL DISTRIBUTIO		37.7	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		13.88	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED		18.03	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (FOTAL LEUCOCYTE COU		11000	/cmm	4000 - 11000
by FLOW CYTOMETRY BY S	F CUBE & MICROSCOPY	11280 ^H	/ Chini	
NUCLEATED RED BLOOD		NIL		0.00 - 20.00
NUCLEATED RED BLOO		NIL	%	< 10 %

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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







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

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	61	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	28	%	20 - 40
EOSINOPHILS	4	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY		/0	1 0
MONOCYTES	7	%	2 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	6881	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0150	,	000 4000
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3158	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT	451 ^H	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	451	/ cililii	011-01
ABSOLUTE MONOCYTE COUNT	790	/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	<u>E MARKERS.</u>		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	592000 ^H	/cmm	150000 - 450000
PLATELETCRIT (PCT)	0.47 ^H	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.47	/0	0.10 0.00
MEAN PLATELET VOLUME (MPV)	8	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL COUNT (P-LCC)	70000	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	11.5	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW)	15.5	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	10.0	/0	10.0 - 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



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'est Name		Value	Unit	Biological Reference interval
by RED CELL AGGRE	DIMENTATION RATE (ESR) gation by capillary photometr	77 ^H	IMENTATION RATE (mm/1st	hr 0 - 20
by RED CELL AGGRE TERPRETATION: ESR is a non-speci imune disease, but An ESR can be affe	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETR fic test because an elevated resul does not tell the health practitio ected by other conditions besides	77H t often indicates ner exactly whe	mm/1st s the presence of inflammat re the inflammation is in th	hr 0 - 20 ion associated with infection, cancer and auto
by RED CELL AGGRE TERPRETATION: ESR is a non-speci mune disease, bui An ESR can be affe C-reactive proteir This test may also stemic lupus eryth NDITION WITH LO	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETR fic test because an elevated result t does not tell the health practitio ected by other conditions besides be used to monitor disease activitiematosus W ESR	77H t often indicates ner exactly whe inflammation. F ity and response	mm/1st s the presence of inflammat re the inflammation is in th for this reason, the ESR is ty e to therapy in both of the a	hr 0 - 20 ion associated with infection, cancer and auto e body or what is causing it. pically used in conjunction with other test such bove diseases as well as some others, such as
by RED CELL AGGRE TERPRETATION: ESR is a non-speci mune disease, bui An ESR can be affe C-reactive proteir This test may also stemic lupus eryth DNDITION WITH LO low ESR can be see olycythaemia), sig sickle cells in sick	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETR fic test because an elevated result does not tell the health practitio ected by other conditions besides be used to monitor disease activitient ematosus W ESR en with conditions that inhibit the	77H toften indicates ner exactly whe inflammation. F ity and response e normal sedime bunt (leucocytos	mm/1st s the presence of inflammat re the inflammation is in th for this reason, the ESR is ty e to therapy in both of the a ntation of red blood cells, s	hr 0 - 20 ion associated with infection, cancer and auto e body or what is causing it. pically used in conjunction with other test such
by RED CELL AGGRE TERPRETATION: ESR is a non-speci- nmune disease, but An ESR can be affe C-reactive proteir This test may also stemic lupus eryth DNDITION WITH LO low ESR can be see olycythaemia), sig sickle cells in sick OTE: ESR and C - reactiv Generally, ESR do CRP is not affected If the ESR is eleva	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETR fic test because an elevated result t does not tell the health practitio ected by other conditions besides be used to monitor disease activity ematosus W ESR en with conditions that inhibit the nificantly high white blood cell co le cell anaemia) also lower the Est re protein (C-RP) are both markers es not charge as rapidly as does C I by as many other factors as is ESI ted, it is typically a result of two t	77H toften indicates ner exactly whe inflammation. F ity and response e normal sedime bunt (leucocytos SR. s of inflammatio CRP, either at the R , making it a be ypes of proteins	mm/1st s the presence of inflammat re the inflammation is in th for this reason, the ESR is ty e to therapy in both of the a ntation of red blood cells, s is) , and some protein abno n. e start of inflammation or a etter marker of inflammatio , globulins or fibrinogen.	hr 0 - 20 ion associated with infection, cancer and auto e body or what is causing it. pically used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count rmalities. Some changes in red cell shape (suc s it resolves. n.
by RED CELL AGGRE TERPRETATION: ESR is a non-speci mune disease, bui An ESR can be affe C-reactive proteir This test may also stemic lupus eryth DNDITION WITH LO ow ESR can be seed olycythaemia), sig sickle cells in sick DTE: ESR and C - reactive Generally, ESR door CRP is not affected If the ESR is elevait Women tend to ha Drugs such as dex	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETR fic test because an elevated result does not tell the health practitio ected by other conditions besides be used to monitor disease activitient we used to monitor disease activitient of the conditions that inhibit the nificantly high white blood cell co le cell anaemia) also lower the Est we protein (C-RP) are both markers as not change as rapidly as does C by as many other factors as is ESI ted, it is typically a result of two to ave a higher ESR, and menstruatio	77H toften indicates ner exactly whe inflammation. F ity and response ount and response sof inflammatio CRP, either at the R , making it a be ypes of proteins an and pregnanc	mm/1st s the presence of inflammat re the inflammation is in th for this reason, the ESR is ty e to therapy in both of the a ntation of red blood cells, s is) , and some protein abno n. e start of inflammation or a etter marker of inflammation s, globulins or fibrinogen. y can cause temporary eleva	hr 0 - 20 ion associated with infection, cancer and auto e body or what is causing it. pically used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count rmalities. Some changes in red cell shape (suc s it resolves. n.





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		& Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLINI	ICAL CHEMISTRY	BIOCHEMIST	RY
		GLUCOSE FAST	ING (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 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
			FILE : BASIC	
HOLESTEROL TOT		116.52		OPTIMAL: < 200.0
by CHOLESTEROL OX		110.32	mg/dL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
				240.0
'RIGLYCERIDES: SI by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	130.29	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
IDL CHOLESTEROI by SELECTIVE INHIBITI	L (DIRECT): SERUM ON	25.85 ^L	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0
DL CHOLESTEROL by CALCULATED, SPE		64.61	mg/dL	HIGH HDL: > OR = 60.0 OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0
ION HDL CHOLEST by calculated, spec		90.67	mg/dL	VERY HIGH: > OR = 190.0 OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0
				VERY HIGH: $> OR = 220.0$
LDL CHOLESTERC		26.06	mg/dL	0.00 - 45.00
OTAL LIPIDS: SER	UM ctrophotometry	363.33	mg/dL	350.00 - 700.00
HOLESTEROL/HD		4.51 ^H	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 by CALCULATED, SPE		2.5	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	5.04 ^H	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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gm/dL

gm/dL

gm/dL

RATIO

6.20 - 8.00

3.50 - 5.50

2.30 - 3.50

1.00 - 2.00

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Test Name		Value	Unit	Biological Reference interval
	LIVER	FUNCTION T	TEST (COMPLETE)	
BILIRUBIN TOTAL: by DIAZOTIZATION, SF	SERUM PECTROPHOTOMETRY	0.52	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.13	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE by CALCULATED, SPE	CT (UNCONJUGATED): SERUM	0.39	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	16.1	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	34.1	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE		0.47	RATIO	0.00 - 46.00
ALKALINE PHOSPI by para nitrophen propanol	IATASE: SERUM YL PHOSPHATASE BY AMINO METHYL	102.14	U/L	40.0 - 130.0
GAMMA GLUTAMY	L TRANSFERASE (GGT): SERUM	150.18 ^H	U/L	0.00 - 55.0

by BIURET, SPECTROPHOTOMETRY ALBUMIN: SERUM 3.77 by BROMOCRESOL GREEN **GLOBULIN: SERUM** 3.01 by CALCULATED, SPECTROPHOTOMETRY A : G RATIO: SERUM 1.25 by CALCULATED, SPECTROPHOTOMETRY

INTERPRETATION

by SZASZ, SPECTROPHTOMETRY TOTAL PROTEINS: SERUM

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:

> 2
> 2 (Highly Suggestive)
1.4 - 2.0
> 1.5
> 1.3 (Slightly Increased)

6.78





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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



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







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Test Name		Value	Unit	Biological Reference interval			
	KIDNI	EX FUNCTION	TEST (COMPLETE)				
UREA: SERUM		24.46	mg/dL	10.00 - 50.00			
by UREASE - GLUTAN	NATE DEHYDROGENASE (GLDH)						
CREATININE: SERU		1.03	mg/dL	0.40 - 1.40			
BLOOD UREA NITE	ROGEN (BUN): SERUM	11.43	mg/dL	7.0 - 25.0			
BLOOD UREA NITH	ROGEN (BUN)/CREATININE	11.1	RATIO	10.0 - 20.0			
RATIO: SERUM	ECTROPHOTOMETRY						
UREA/CREATININ	by CALCULATED, SPECTROPHOTOMETRY UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY		RATIO				
URIC ACID: SERUM	URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE		mg/dL	3.60 - 7.70			
CALCIUM: SERUM			mg/dL	8.50 - 10.60			
PHOSPHOROUS: SE by phosphomolybe	ERUM DATE, SPECTROPHOTOMETRY	3.37	mg/dL	2.30 - 4.70			
ELECTROLYTES							
SODIUM: SERUM			mmol/L	135.0 - 150.0			
POTASSIUM: SERU	by ISE (ION SELECTIVE ELECTRODE) POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE) CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)		mmol/L	3.50 - 5.00			
CHLORIDE: SERUM			mmol/L	90.0 - 110.0			
	MERULAR FILTERATION RATE						
ESTIMATED GLOM (eGFR): SERUM by calculated INTERDRETATION:	IERULAR FILTERATION RATE	90.7					

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.

3. GI haemorrhage.



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

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

 KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana

 KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana

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 care@koshealthcare.com



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





	1	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		Dr. Yugam Chopra MD (Pathology) st CEO & Consultant Pathologist			
IAME	: Mr. RAVIND	ER SAINI					
AGE/ GENDER	: 46 YRS/MALI	Ξ	PATIENT	T ID	: 1659822		
COLLECTED BY	:		REG. NO.	/LAB NO.	:012411040002	;	
REFERRED BY			REGISTR	ATION DATE	:04/Nov/202406:	:20 AM	
BARCODE NO.	:01520007			TION DATE	: 04/Nov/2024 06:		
CLIENT CODE.	: KOS DIAGNO	STIC Ι ΔΒ		ING DATE			
CLIENT ADDRESS		HOLSON ROAD, AMB					
Test Name			Value	Unit	Biologica	al Reference interval	
1. Postrenal azotemia	ı (BUN rises dispr		LS: han creatinine) (e.g. c	bstructive urop	athy).		
 Postrenal azotemia Prerenal azotemia Prerenal azotemia Prerenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necro Low protein diet ar Severe liver disease Other causes of der Repeated dialysis (Inherited hyperaming SIADH (syndrome on Pregnancy. DECREASED RATIO (<1 Phenacimide therand Rhabdomyolysis (ref Muscular patients of Muscular patients of Diabetic ketoacidos should produce an info Cephalosporin ther ESTIMATED GLOMERU CKD STAGE 	I (BUN rises dispr superimposed o IO:1) WITH DECRE osis. Ind starvation. 2. creased urea syr urea rather than monemias (urea of inappropiate a IO:1) WITH INCRE py (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/cre apy (interferes v JLAR FILTERATION	roportionately more t n renal disease. EASED BUN : in creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creatine treatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu N RATE: DESCRIPTION	han creatinine) (e.g. c nut of extracellular flu blood). due to tubular secreti e to creatinine). e in creatinine with co rement). GFR (mL/min/1.	id). on of urea. ertain methodol	ogies,resulting in norm SSOCIATED FINDINGS	nal ratio when dehydrat	
Postrenal azotemia Prerenal azotemia Cecreased RATIO (<1 Acute tubular necre Low protein diet an Severe liver disease Other causes of dec Repeated dialysis (Inherited hyperami SIADH (syndrome o Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (re Rhabdomyolysis (re NAPPROPIATE RATIO Diabetic ketoacido hould produce an inc Cephalosporin ther STIMATED GLOMERU CKD STAGE	(BUN rises dispr superimposed o IO:1) WITH DECRE osis. ad starvation. creased urea syr urea rather than monemias (urea of inappropiate a IO:1) WITH INCRE py (accelerates c eleases muscle c who develop rer : sis (acetoacetate creased BUN/cre apy (interferes w ULAR FILTERATION Norn	roportionately more t n renal disease. EASED BUN : in creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creatine treatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measu N RATE: DESCRIPTION mal kidney function	han creatinine) (e.g. c out of extracellular flu blood). due to tubular secreti e to creatinine). e in creatinine with co rement). GFR (mL/min/1. >90	id). on of urea. ertain methodol 73m2) A t	ogies,resulting in norm SSOCIATED FINDINGS No proteinuria	nal ratio when dehydrat	
Postrenal azotemia Prerenal azotemia Cecreased RATIO (<1 Acute tubular necro Low protein diet ar Severe liver disease Other causes of der Severe liver disease Nepeated dialysis (Severe liver disease Naperoprise RATIO (<1 Nenacimide thera Severe RATIO concerning Naperopiate RATIO Diabetic ketoacido Should produce an ind Cephalosporin ther STIMATED GLOMERU CKD STAGE G1 G2	(BUN rises dispr superimposed o IO:1) WITH DECRE osis. ad starvation. e. creased urea syr urea rather than monemias (urea of inappropiate a IO:1) WITH INCRE py (accelerates c eleases muscle c who develop rer : sis (acetoacetate creased BUN/cre apy (interferes w UAR FILTERATION Norm Kic no	roportionately more t n renal disease. EASED BUN : in creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creatine creatinine). hal failure. e causes false increase extinine ratio). vith creatinine measu N RATE: DESCRIPTION mal kidney function dney damage with pormal or high GFR	han creatinine) (e.g. c ut of extracellular flu blood). due to tubular secreti e to creatinine). e in creatinine with co rement). GFR (mL/min/1. >90 >90	id). on of urea. ertain methodol 73m2) At	ogies,resulting in norm SSOCIATED FINDINGS	nal ratio when dehydrat	
Postrenal azotemia Prerenal azotemia CECREASED RATIO (<1 Acute tubular necro Low protein diet ar Severe liver disease Other causes of der Severe liver disease Other causes of der SIADH (syndrome o Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (re NAPPROPIATE RATIO Diabetic ketoacido hould produce an ind Cephalosporin ther STIMATED GLOMERU G1 G2	(BUN rises dispr superimposed o IO:1) WITH DECRE osis. ad starvation. e. creased urea syr urea rather than monemias (urea of inappropiate a IO:1) WITH INCRE py (accelerates c eleases muscle c who develop rer : sis (acetoacetate creased BUN/cre apy (interferes w UAR FILTERATION Norm Kin Norm Kin Norm	roportionately more t n renal disease. EASED BUN : in creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creatine creatinine). hal failure. e causes false increase extinine ratio). vith creatinine measur N RATE: DESCRIPTION mal kidney function dney damage with formal or high GFR Id decrease in GFR	han creatinine) (e.g. c ut of extracellular flu blood). due to tubular secreti e to creatinine). e in creatinine with co rement). GFR (mL/min/1. >90 >90 60 -89	id). on of urea. ertain methodol 73m2) At	ogies,resulting in norm SSOCIATED FINDINGS No proteinuria Presence of Protein ,	hal ratio when dehydrat	
Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necro Low protein diet ar Severe liver disease Other causes of der Repeated dialysis (Inherited hyperami SIADH (syndrome o Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (re MAPPROPIATE RATIO Diabetic ketoacido should produce an ind CED STAGE G1 G2	(BUN rises dispr superimposed o IO:1) WITH DECRE osis. ad starvation. creased urea syr urea rather than monemias (urea of inappropiate a IO:1) WITH INCRE py (accelerates c eleases muscle c who develop rer : sis (acetoacetate creased BUN/cre apy (interferes w UAR FILTERATION Norm Kic Norm Kic Norde	roportionately more t n renal disease. EASED BUN : in creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creatine creatinine). hal failure. e causes false increase extinine ratio). vith creatinine measu N RATE: DESCRIPTION mal kidney function dney damage with pormal or high GFR	han creatinine) (e.g. c ut of extracellular flu blood). due to tubular secreti e to creatinine). e in creatinine with co rement). GFR (mL/min/1. >90 >90	id). on of urea. ertain methodol 73m2) At	ogies,resulting in norm SSOCIATED FINDINGS No proteinuria Presence of Protein ,	nal ratio when dehydrat	





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

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









CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMI	BALA CANTT	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 04/Nov/2024 07:15AM
BARCODE NO.	: 01520007	COLLECTION DATE	:04/Nov/202406:34AM
REFERRED BY	:	REGISTRATION DATE	: 04/Nov/2024 06:20 AM
COLLECTED BY	:	REG. NO./LAB NO.	:012411040002
AGE/ GENDER	: 46 YRS/MALE	PATIENT ID	: 1659822
NAME	: Mr. RAVINDER SAINI		
	Dr. Vinay Chopi MD (Pathology & Mic Chairman & Consulta	crobiology) MI	m Chopra D (Pathology) nt Pathologist

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 Ch MD (Pathology & Chairman & Con	k Microbiology)	Dr. Yugam MD EO & Consultant	(Pathology)
NAME	: Mr. RAVINDER SAINI			
AGE/ GENDER	: 46 YRS/MALE	PATIENT	ID	: 1659822
COLLECTED BY	:	REG. NO./	LAB NO.	: 012411040002
REFERRED BY	:	REGISTR	ATION DATE	: 04/Nov/2024 06:20 AM
BARCODE NO.	: 01520007		ION DATE	: 04/Nov/2024 06:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTI	NG DATE	: 04/Nov/2024 09:28AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PATHO	LOGY	
	URINE RO	OUTINE & MICROSCO	PIC EXAMINA	ATION
PHYSICAL EXAMIN	NATION			
QUANTITY RECIEV	ED TANCE SPECTROPHOTOMETRY	10	ml	
COLOUR		PALE YELLOW		PALE YELLOW
TRANSPARANCY	TANCE SPECTROPHOTOMETRY	CLEAR		CLEAR
SPECIFIC GRAVITY	TANCE SPECTROPHOTOMETRY	1.02		1.002 - 1.030
CHEMICAL EXAMI	NATION			
REACTION		ACIDIC		
PROTEIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR	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	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
ASCORBIC ACID	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
RED BLOOD CELLS		NEGATIVE (-ve)	/HPF	0 - 3



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







EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

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

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

NAME	: Mr. RAVINDER SAINI			
AGE/ GENDER	: 46 YRS/MALE		PATIENT ID	: 1659822
COLLECTED BY	:		REG. NO./LAB NO.	: 012411040002
REFERRED BY	:		REGISTRATION DATE	: 04/Nov/2024 06:20 AM
BARCODE NO.	: 01520007		COLLECTION DATE	:04/Nov/202406:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	:04/Nov/202409:28AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANT	T	
Test Name		Value	Unit	Biological Reference interval
by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT			
PUS CELLS		2-4	/HPF	0 - 5

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
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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