

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



	Dr. Vinay Chop MD (Pathology & Mi Chairman & Consult	crobiology)		(Pathology)
NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Mr. VICKY YADAV : 48 YRS/MALE : : : 01515822 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AM	BALA CANTT	PATIENT ID REG. NO./LAB NO. REGISTRATION DATE COLLECTION DATE REPORTING DATE	: 1593222 : 012408270052 : 27/Aug/2024 02:42 PM : 27/Aug/2024 02:46PM : 27/Aug/2024 03:26PM
Test Name		Value	Unit	Biological Reference interval
		HAEM	ATOLOGY	
	CO		DOD COUNT (CBC)	
RED BLOOD CELLS (R	RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB)		12.1	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (RE		4.21	Millions/c	mm 3.50 - 5.00
by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE	37.3 ^L	%	40.0 - 54.0
	AUTOMATED HEMATOLOGY ANALYZER	88.5	fL	80.0 - 100.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
by CALCULATED BY A	R HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	28.7	pg	27.0 - 34.0
	R HEMOGLOBIN CONC. (MCHC)	32.4	g/dL	32.0 - 36.0
	ION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	13	%	11.00 - 16.00
RED CELL DISTRIBUT	ION WIDTH (RDW-SD)	43	fL	35.0 - 56.0
by CALCULATED BY A MENTZERS INDEX by CALCULATED	UTOMATED HEMATOLOGY ANALYZER	21.02	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDE	Х	27.29	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS	<u>S (WBCS)</u>			
	OUNT (TLC) / by sf cube & microscopy	7510	/cmm	4000 - 11000
NUCLEATED RED BLC	DOD CELLS (nRBCS)	NIL		0.00 - 20.00
NUCLEATED RED BLC	RT HEMATOLOGY ANALYZER DOD CELLS (NRBCS) % UTOMATED HEMATOLOGY ANALYZER DCYTE COUNT (DLC)	NIL	%	< 10 %
NEUTROPHILS	Y BY SF CUBE & MICROSCOPY	53	%	50 - 70



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LYMPHOCYTES		37	%	20 - 40
EOSINOPHILS	Y BY SF CUBE & MICROSCOPY	5	%	1 - 6
-	Y BY SF CUBE & MICROSCOPY			
MONOCYTES	Y BY SF CUBE & MICROSCOPY	5	%	2 - 12
BASOPHILS		0	%	0 - 1
	Y BY SF CUBE & MICROSCOPY			
ABSOLUTE LEUKOCY ABSOLUTE NEUTROF		2020	/cmm	2000 - 7500
	Y BY SF CUBE & MICROSCOPY	3980	/cmm	2000 - 7500
ABSOLUTE LYMPHO		2779	/cmm	800 - 4900
ABSOLUTE EOSINOP	Y BY SF CUBE & MICROSCOPY HIL COUNT	376	/cmm	40 - 440
by FLOW CYTOMETRY	Y BY SF CUBE & MICROSCOPY		/ drift	10 110
	TE COUNT Y by sf cube & microscopy	376	/cmm	80 - 880
	HER PLATELET PREDICTIVE MARK	ERS.		
PLATELET COUNT (PI	LT) FOCUSING, ELECTRICAL IMPEDENCE	234000	/cmm	150000 - 450000
PLATELETCRIT (PCT)	OCUSING, ELECTRICAL IMPEDENCE	0.25	%	0.10 - 0.36
MEAN PLATELET VOI by hydro dynamic f	LUME (MPV) FOCUSING, ELECTRICAL IMPEDENCE	11	fL	6.50 - 12.0
PLATELET LARGE CEL		71000	/cmm	30000 - 90000
PLATELET LARGE CEL		30.5	%	11.0 - 45.0
-	TION WIDTH (PDW) FOCUSING, ELECTRICAL IMPEDENCE ICTED ON EDTA WHOLE BLOOD	16.1	%	15.0 - 17.0



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1	SO 9001 : 2008 CERTI	FIED LAB			EXCELLENCE IN HEALTHCARE	& DIAGNOSTICS	
			Dr. Vinay Chopra MD (Pathology & Microbio Chairman & Consultant Pat			(Pathology)	
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	Test Name		Val	ue	Unit	Biological Reference interval	
	ABO GROUP by SLIDE AGGLUTINATI By SLIDE AGGLUTINATI		B	DRITIVE	AND RH FACTOR TYP	ING	
	KOS Molecular Lab: IInd F	loor, Parry Hot	d, Ambala Cantt -133 001, Harya el, Staff Road, Opp. GPO, Ambal ⊵koshealthcare.com │ www.kos	la Cantt ·		Page 3 of 17	





		hopra & Microbiology) nsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)	
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	R	EPORTING DATE	: 27/Aug/2024 03:58PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
	PR	OTHROMBIN TIM	E STUDIES (PT/INR)		
PT TEST (PATIENT) by PHOTO OPTICAL C		OTHROMBIN TIM 12.6	E STUDIES (PT/INR) SECS	11.5 - 14.5	
by PHOTO OPTICAL C	CLOT DETECTION				
by PHOTO OPTICAL C PT (CONTROL) by PHOTO OPTICAL C	CLOT DETECTION	12.6	SECS		
РТ (CONTROL) by PHOTO OPTICAL C ISI by PHOTO OPTICAL C	CLOT DETECTION CLOT DETECTION CLOT DETECTION DRMALISED RATIO (INR)	12.6 12	SECS		

INTERPRETATION:-

1.INR is the parameter of choice in monitoring adequacy of oral anti-coagulant therapy. Appropriate therapeutic range varies with the disease and treatment intensity.

2. Prolonged INR suggests potential bleeding disorder /bleeding complications

3. Results should be clinically correlated.

4. Test conducted on Citrated Plasma

RECOMMENDED THERAPEUTIC RANGE FOR	ORAL ANTI-CO	AGULANT THE	RAPY (INR)
INDICATION		INTERNATIO	NAL NORMALIZED RATIC (INR)
Treatment of venous thrombosis			
Treatment of pulmonary embolism			
Prevention of systemic embolism in tissue heart valves			
Valvular heart disease	Low Intensity		2.0 - 3.0
Acute myocardial infarction			
Atrial fibrillation			
Bileaflet mechanical valve in aortic position			
Recurrent embolism			
Mechanical heart valve	High Intensity		2.5 - 3.5
Antiphospholipid antibodies ⁺			
COMMENTS:			





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Test Name		Value Un	it Biological Reference interval

The prothrombin time (PT) and its derived measures of prothrombin ratio (PR) and international normalized ratio (INR) are measures of the efficacy of the extrinsic pathway of coagulation. PT test reflects the adequacy of factors I (fibrinogen), II (prothrombin), V, VII, and X. It is used in conjunction with the activated partial thromboplastin time (aPTT) which measures the intrinsic pathway. The common causes of prolonged prothrombin time are :

1.Oral Anticoagulant therapy.

2.Liver disease.

3.Vit K. deficiency.

4.Disseminated intra vascular coagulation.

5.Factor 5, 7, 10 or Prothrombin dificiency



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Test Name		Value	Unit	Biological Reference interval
	ACTIVA	TED PARTIAL TH	ROMBOPLASTIN TIME	(APTT)
APTT (PATIENT VALL by photo optical c	JE)	32.1	SECS	28.6 - 38.2

KOS Diagnostic Lab

(A Unit of KOS Healthcare)

INTERPRETATION:-

The activated partial thromboplastin time (aPTT or APTT) is a performance indicator measuring the efficacy of both the **intrinsic** (now referred to as the contact activation pathway) and the common coagulation pathways. Apart from detecting abnormalities in blood clotting, it is also used to monitor the treatment effects with heparin, a major anticoagulant. It is used in conjunction with the prothrombin time (PT) which measures the extrinsic pathway.

COMMON CAUSES OF PROLONGED APTT :-

1. Disseminated intravascular coagulation.

2. Liver disease.

3. Massive transfusion with stored blood.

4. Heparin administration or contamination.

5. A circulating Anticogulant.

6. Deficiency of a coagulation Factor other than factor 7.



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPOR	TING DATE	: 28/Aug/2024 09:19AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMISTRY/E	BIOCHEMISTRY	
		GLUCOSE FASTI	NG (F)	
GLUCOSE FASTING (by GLUCOSE OXIDAS	F): PLASMA se - peroxidase (god-pod)	115.94 ^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0



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Test Name		Value	Unit	Biological Reference interval
		GLUCOSE RAN	DOM (R)	
-	(R): PLASMA E - PEROXIDASE (GOD-POD)	189.24 ^H	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > 0R = 200.0
INTERPRETATION				

KOS Diagnostic Lab (A Unit of KOS Healthcare)

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

A random plasma glucose level below 140 mg/dl is considered normal.
 A random glucose level between 140 - 200 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prnadial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
 A random glucose level of above 200 mg/dl is highly suggestive of diabetic state. A repeat post-prnadial 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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
<u></u>	KID	NEY FUNCTION	TEST (COMPLETE)	
UREA: SERUM		26.55	mg/dL	10.00 - 50.00
	ATE DEHYDROGENASE (GLDH)			
CREATININE: SERUN by ENZYMATIC, SPEC		1.13	mg/dL	0.40 - 1.40
BLOOD UREA NITRO	GEN (BUN): SERUM	12.41	mg/dL	7.0 - 25.0
BLOOD UREA NITRO RATIO: SERUM	GEN (BUN)/CREATININE	10.98	RATIO	10.0 - 20.0
by CALCULATED, SPE UREA/CREATININE R by CALCULATED, SPE	ATIO: SERUM	23.5	RATIO	
URIC ACID: SERUM by URICASE - OXIDAS	E PEROXIDASE	6.48	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.02	mg/dL	8.50 - 10.60
-	UM wate, spectrophotometry	3.62	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	E ELECTRODE)	141.2	mmol/L	135.0 - 150.0
POTASSIUM: SERUM		4.13	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIV		105.9	mmol/L	90.0 - 110.0
ESTIMATED GLOME	RULAR FILTERATION RATE	80.2		

(eGFR): SERUM 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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8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia	superimposed on renal disease.		uropathy).	
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 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 Muscular patients of Muscular patients of Diabetic ketoacido: Should produce an ing Cephalosporin ther 	ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. e. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse f inappropiate antidiuretic harm 0:1) WITH INCREASED CREATININ py (accelerates conversion of cree eleases muscle creatinine). who develop renal failure.	LEVELS: nore than creatinine) (e.g. obstructive uses out of extracellular fluid). nt in blood). one) due to tubular secretion of urea. HE: eatine to creatinine).		al ratio when dehydratic
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal 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 hyperami SIADH (syndrome o Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rabdomyolysis (re Muscular patients of Muscular patients of Should produce an ind Cephalosporin there 	ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse f inappropiate antidiuretic harm 0:1) WITH INCREASED CREATININ py (accelerates conversion of creatinine). who develop renal failure. sis (acetoacetate causes false ind creased BUN/creatinine ratio). apy (interferes with creatinine m UAR FILTERATION RATE:	LEVELS: nore than creatinine) (e.g. obstructive uses out of extracellular fluid). nt in blood). one) due to tubular secretion of urea. IE: eatine to creatinine). crease in creatinine). crease in creatinine with certain metheasurement). GFR (mL/min/1.73m2)	nodologies,resulting in norm	al ratio when dehydratic
 Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal 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 hyperamin SIADH (syndrome o Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rabdomyolysis (ref Muscular patients of Muscular patients of Cephalosporin ther Cephalosporin ther ESTIMATED GLOMERU CKD STAGE 	ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse f inappropiate antidiuretic harm 0:1) WITH INCREASED CREATININ py (accelerates conversion of creatinine). who develop renal failure. creased BUN/creatinine ratio). apy (interferes with creatinine m LAR FILTERATION RATE: DESCRIPTION Normal kidney funct Kidney damage wit	LEVELS: nore than creatinine) (e.g. obstructive uses out of extracellular fluid). nt in blood). one) due to tubular secretion of urea. IE: eatine to creatinine). crease in creatinine with certain metheasurement). GFR (mL/min/1.73m2) ion >90 th >90	nodologies,resulting in norm ASSOCIATED FINDINGS No proteinuria Presence of Protein ,	al ratio when dehydratic
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal 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 hyperami SIADH (syndrome o Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (ref Muscular patients of Muscular patients of Cephalosporin ther ESTIMATED GLOMERU CKD STAGE G1 G2 	ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse f inappropiate antidiuretic harm 0:1) WITH INCREASED CREATININ py (accelerates conversion of creatinine). who develop renal failure. creased BUN/creatinine ratio). apy (interferes with creatinine m LAR FILTERATION RATE: DESCRIPTION Normal kidney funct Kidney damage wit normal or high GF	LEVELS: nore than creatinine) (e.g. obstructive uses out of extracellular fluid). nt in blood). one) due to tubular secretion of urea. IE: eatine to creatinine). crease in creatinine with certain metheasurement). GFR (mL/min/1.73m2) ion >90 ch >90 R >90	nodologies,resulting in norm ASSOCIATED FINDINGS No proteinuria	al ratio when dehydratic
B. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necro Low protein diet ar Severe liver disease Other causes of dec Repeated dialysis (SIADH (syndrome o Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (re Robert RATIO (<1 Nuscular patients NAPPROPIATE RATIO Cephalosporin ther STIMATED GLOMERU G1 G2	ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse f inappropiate antidiuretic harm 0:1) WITH INCREASED CREATININ py (accelerates conversion of creatinine). who develop renal failure. : sis (acetoacetate causes false ind creased BUN/creatinine ratio). apy (interferes with creatinine m LAR FILTERATION RATE: DESCRIPTION Normal kidney funct Kidney damage wit normal or high GF Mild decrease in Gi	LEVELS: nore than creatinine) (e.g. obstructive asses out of extracellular fluid). nt in blood). one) due to tubular secretion of urea. IE: eatine to creatinine). crease in creatinine with certain metheasurement). ion >90 ch >90 R 60 - 89	nodologies,resulting in norm ASSOCIATED FINDINGS No proteinuria Presence of Protein ,	al ratio when dehydratic
8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necro 2. Low protein diet ar 3. Severe liver disease 4. Other causes of deu 5. Repeated dialysis (6. Inherited hyperami 7. SIADH (syndrome o 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (re 3. Muscular patients o INAPPROPIATE RATIO 1. Diabetic ketoacidos should produce an ind 2. Cephalosporin ther ESTIMATED GLOMERU CKD STAGE G1 G2	ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse f inappropiate antidiuretic harm 0:1) WITH INCREASED CREATININ py (accelerates conversion of creatinine). who develop renal failure. creased BUN/creatinine ratio). apy (interferes with creatinine m LAR FILTERATION RATE: DESCRIPTION Normal kidney funct Kidney damage wit normal or high GF	LEVELS: nore than creatinine) (e.g. obstructive uses out of extracellular fluid). nt in blood). one) due to tubular secretion of urea. IE: eatine to creatinine). crease in creatinine with certain mether neasurement). Ion >90 In >90 R 60 -89 GFR 30-59	nodologies,resulting in norm ASSOCIATED FINDINGS No proteinuria Presence of Protein ,	al ratio when dehydratic



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

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









	Dr. Vinay Chop MD (Pathology & Mi Chairman & Consult	crobiology) M	m Chopra D (Pathology) nt Pathologist
NAME	: Mr. VICKY YADAV		
AGE/ GENDER	: 48 YRS/MALE	PATIENT ID	: 1593222
COLLECTED BY	:	REG. NO./LAB NO.	: 012408270052
REFERRED BY	:	REGISTRATION DATE	: 27/Aug/2024 02:42 PM
BARCODE NO.	: 01515822	COLLECTION DATE	: 27/Aug/2024 02:46PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 27/Aug/2024 04:56PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA 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





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
IMMUNOPATHOLOGY/SEROLOGY HEPATITIS C VIRUS (HCV) ANTIBODY: TOTAL				
	DY (HCV) TOTAL: SERUM ESCENT MICROPARTICLE IMMUNOA	0.09 ASSAY)	S/CO	NEGATIVE: < 1.00 POSITIVE: > 1.00
HEPATITIS C ANTIBO	DY (HCV) TOTAL	NON - RE	ACTIVE	
RESULT by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)				
INTERPRETATION:-				
RE	SULT (INDEX)			
	<1.00 >=1.00 REACTIV		NON - REACTIVE/NOT - DETECTED CTIVE/ASYMPTOMATIC/INFECTIVE STATE/CARRIER STATE.	
Hepatitis C (HCV) is a	n RNA virus of Favivirus group	transmitted via b	lood transfusions, transplai	ntation, injection drug abusers, accidental
Hepatitis C (HCV) is an RNA virus of Favivirus group transmitted via blood transfusions, transplantation, injection drug abusers, accidental needle punctures in healthcare workers, dialysis patients and rarely from mother to infant. 10 % of new cases show sexual transmission. As compared to HAV & HBV, chronic infection with HCV occurs in 85 % of infected individuals. In high risk population, the predictive value of Anti HCV for HCV infection is > 99% whereas in low risk populations it is only 25 %. USES: 1. Indicator of past or present infection, but does not differentiate between Acute/ Chronic/Resolved Infection.				

2. Routine screening of low and high prevelance population including blood donors.

NOTE:

1. False positive results are seen in Auto-immune disease, Rheumatoid Factor, HYpergammaglobulinemia, Paraproteinemia, Passive antibody transfer, Anti-idiotypes and Anti-superoxide dismutase.

2. False negative results are seen in early Acute infection, Immunosuppression and Immuno-incompetence.

3. HCV-RNĂ PCR recommended in all reactive results to differentiate between past and present infection.





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		Chopra y & Microbiology) onsultant Pathologist	Dr. Yugan MD CEO & Consultant	(Pathology)
NAME	: Mr. VICKY YADAV			
AGE/ GENDER	: 48 YRS/MALE	PATI	ENT ID	: 1593222
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	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAI		RTING DATE	: 27/Aug/2024 04:56PM
CLIENT CODE. CLIENT ADDRESS Test Name			Unit	Biological Reference interval
CLIENT ADDRESS Test Name	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT Value	Unit	
CLIENT ADDRESS Test Name ANT HIV 1/2 AND P24 AI	: 6349/1, NICHOLSON ROAD	D, AMBALA CANTT Value CIENCY VIRUS (HIV) DU 0.07	Unit	Biological Reference interval
CLIENT ADDRESS Test Name ANT HIV 1/2 AND P24 AI by CMIA (CHEMILUMI HIV 1/2 AND P24 AI	: 6349/1, NICHOLSON ROAD	D, AMBALA CANTT Value CIENCY VIRUS (HIV) DU 0.07 DASSAY) NON - REACTIVE	Unit JO ULTRA WITH S/CO	Biological Reference interval (P-24 ANTIGEN DETECTION) NEGATIVE: < 1.00
CLIENT ADDRESS Test Name ANT HIV 1/2 AND P24 AI by CMIA (CHEMILUMII HIV 1/2 AND P24 AI by CMIA (CHEMILUMII INTERPRETATION:-	: 6349/1, NICHOLSON ROAD	D, AMBALA CANTT Value CIENCY VIRUS (HIV) DU 0.07 DASSAY) NON - REACTIVE	Unit JO ULTRA WITH S/CO	Biological Reference interval (P-24 ANTIGEN DETECTION) NEGATIVE: < 1.00
CLIENT ADDRESS Test Name ANT HIV 1/2 AND P24 AI by CMIA (CHEMILUMII HIV 1/2 AND P24 AI by CMIA (CHEMILUMII INTERPRETATION:- RESU	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT Value CIENCY VIRUS (HIV) DU 0.07 DASSAY) NON - REACTIVE DASSAY)	Unit JO ULTRA WITH S/CO	Biological Reference interval (P-24 ANTIGEN DETECTION) NEGATIVE: < 1.00

exposed to HIV 1/2 infection or the sample has been tested during the "window phase" i.e. before the development of detectable levels of antibodies. Hence a Non Reactive result does not exclude the possibility of exposure or infection with HIV 1/2. **RECOMMENDATIONS:**

Results to be clinically correlated
 Rarely falsenegativity/positivity may occur.

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





		hopra & Microbiology) nsultant Pathologist		(Pathology)
NAME	: Mr. VICKY YADAV			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
Test Name	НЕРАТ		Unit ANTIGEN (HBsAg) UL	
L HEPATITIS B SURFA SERUM	CE ANTIGEN (HBsAg):	ITIS B SURFACE		
HEPATITIS B SURFA SERUM <i>by CMIA (CHEMILUMII</i> HEPATITIS B SURFA RESULT		ITIS B SURFACE 0.22 ASSAY) NON REAC	ANTIGEN (HBsAg) UL S/CO	TRA NEGATIVE: < 1.0
HEPATITIS B SURFA SERUM <i>by CMIA (CHEMILUMII</i> HEPATITIS B SURFA RESULT <i>by CMIA (CHEMILUMII</i>	CE ANTIGEN (HBsAg): <i>NESCENT MICROPARTICLE IMMUNO</i> CE ANTIGEN (HBSAg)	ITIS B SURFACE 0.22 ASSAY) NON REAC	ANTIGEN (HBsAg) UL S/CO	TRA NEGATIVE: < 1.0
HEPATITIS B SURFA SERUM <i>by CMIA (CHEMILUMII</i> HEPATITIS B SURFA RESULT <i>by CMIA (CHEMILUMII</i> <u>INTERPRETATION:</u> RESU	CE ANTIGEN (HBsAg): <i>NESCENT MICROPARTICLE IMMUNO</i> CE ANTIGEN (HBSAg)	ITIS B SURFACE 0.22 ASSAY) NON REAC	ANTIGEN (HBsAg) UL S/CO	TRA NEGATIVE: < 1.0

Hepatitis B Virus (HBV) is a member of the Hepadna virus family causing infection of the liver with extremely variable clinical features. Hepatitis B is transmitted primarily by body fluids especially serum and also spread effectively sexually and from mother to baby. In most individuals HBV hepatitis is self limiting, but 1-2 % normal adolescent and adults develop Chronic Hepatitis. Frequency of chronic HBV infection is 5-10% in immunocompromised patients and 80 % neonates. The initial serological marker of acute infection is HBsAg which typically appears 2-3 months after infection and disappears 12-20 weeks after onset of symtoms. Persistence of HBsAg for more than 6 months indicates carrier state or Chronic Liver disease.





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BARCODE NO.	: 01515822	COLLECTION DATE	: 27/Aug/2024 02:46PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 27/Aug/2024 03:57PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		
Test Name		Value Unit	Biological Reference interval
		VDRL	
		NON REACTIVE	NON REACTIVE
		NON REACTIVE	NON REACTIVE
by IMMUNOCHROMAT	FOGRAPHY	NON REACTIVE	NON REACTIVE
<i>by IMMUNOCHROMAT</i> INTERPRETATION: 1.Does not become p	positive until 7 - 10 days after ap		NON REACTIVE
<i>by IMMUNOCHROMAT</i> <u>INTERPRETATION:</u> 1.Does not become p 2. High titer (>1:16) - a	positive until 7 - 10 days after ap active disease.	pearance of chancre.	NON REACTIVE
by IMMUNOCHROMAT INTERPRETATION: 1.Does not become p 2.High titer (>1:16) - a 3.Low titer (<1:8) - bit 4.Treatment of prima	positive until 7 - 10 days after ap active disease. iological falsepositive test in 90% ary syphillis causes progressive c	pearance ofchancre. cases or due to late or late latent syphillis. lecline tonegative VDRL within 2 years.	NON REACTIVE
by IMMUNOCHROMAT INTERPRETATION: 1.Does not become p 2.High titer (>1:16) - a 3.Low titer (<1:8) - bit 4.Treatment of prima 5.Rising titer (4X) ind	positive until 7 - 10 days after ap active disease. iological falsepositive test in 90% ary syphillis causes progressive c licates relapse,reinfection, or trea	pearance ofchancre. cases or due to late or late latent syphillis. lecline tonegative VDRL within 2 years. atment failure and need for retreatment.	NON REACTIVE
by IMMUNOCHROMAT INTERPRETATION: 1.Does not become p 2.High titer (>1:16) - a 3.Low titer (<1:8) - bit 4.Treatment of prima 5.Rising titer (4X) ind 6.May benonreactive	positive until 7 - 10 days after ap active disease. iological falsepositive test in 90% ary syphillis causes progressive o licates relapse,reinfection, or trea e in early primary, late latent, an	pearance ofchancre. cases or due to late or late latent syphillis. lecline tonegative VDRL within 2 years.	
by IMMUNOCHROMAT INTERPRETATION: 1.Does not become p 2.High titer (>1:16) - a 3.Low titer (<1:8) - bi 4.Treatment of prima 5.Rising titer (4X) ind 6.May benonreactive 7.Reactive and weak	positive until 7 - 10 days after ap active disease. iological falsepositive test in 90% ary syphillis causes progressive c licates relapse,reinfection, or trea e in early primary, late latent, an ly reactive tests should always be	pearance ofchancre. cases or due to late or late latent syphillis. lecline tonegative VDRL within 2 years. atment failure and need for retreatment. Ind late syphillis (approx. 25% ofcases). e confirmedwith FTA-ABS (fluorescent trepon	
INTERPRETATION: 1.Does not become p 2.High titer (>1:16) - i 3.Low titer (<1:8) - bi 4.Treatment of prima 5.Rising titer (4X) ind 6.May benonreactive 7.Reactive and weak SHORTTERM FALSE PC 1.Acute viral illnesse:	positive until 7 - 10 days after app active disease. iological falsepositive test in 90% ary syphillis causes progressive of licates relapse,reinfection, or trea e in early primary, late latent, an ly reactive tests should always be OSITIVE TEST RESULTS (<6 MONTHes (e.g., hepatitis, measles, infect	pearance ofchancre. <i>cases or due to late or late latent syphillis.</i> lecline tonegative VDRL within 2 years. atment failure and need for retreatment. Id late syphillis (approx. 25% ofcases). <i>e confirmedwith FTA-ABS (fluorescent trepon</i> HS DURATION) MAY OCCURIN:	
by IMMUNOCHROMAT INTERPRETATION: 1.Does not become p 2.High titer (>1:16) - i 3.Low titer (>1:8) - bi 4.Treatment of prima 5.Rising titer (4X) ind 6.May benonreactive 7.Reactive and weak SHORTTERM FALSE PC 1.Acute viral illnesses	positive until 7 - 10 days after app active disease. iological falsepositive test in 90% ary syphillis causes progressive of licates relapse,reinfection, or trea e in early primary, late latent, an dy reactive tests should always be OSITIVE TEST RESULTS (<6 MONTHes (e.g., hepatitis, measles, infect hlamydia; Malaria infection.	pearance ofchancre. <i>cases or due to late or late latent syphillis.</i> lecline tonegative VDRL within 2 years. atment failure and need for retreatment. Id late syphillis (approx. 25% ofcases). <i>e confirmedwith FTA-ABS (fluorescent trepon</i> HS DURATION) MAY OCCURIN:	

LONGTERM FALSE POSITIVE TEST RESULTS (>6 MONTHS DURATION) MAY OCCUR IN:

- 1. Serious underlying disease e.g., collagen vascular diseases, leprosy, malignancy.
- 2.Intravenous drug users.
- 3. Rheumatoid arthritis, thyroiditis, AIDS, Sjogren's syndrome.
- 4.<10 % of patients older thanage 70 years.
- 5.Patients taking some anti-hypertensive drugs.





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





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NAME	: Mr. VICKY YADAV			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		Value	onit	
		CLINICAL PAT	HOLOGY	
	URINE R	OUTINE & MICROS	SCOPIC EXAMINAT	ION
PHYSICAL EXAMINA	TION			
QUANTITY RECIEVE		10	ml	
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
COLOUR	TANCE SPECTROPHOTOMETRY	PALE YELLOW		PALE YELLOW
TRANSPARANCY	TANCE SPECTROPHOTOMETRY	CLEAR		CLEAR
-	TANCE SPECTROPHOTOMETRY			
SPECIFIC GRAVITY	TANCE SPECTROPHOTOMETRY	>=1.030		1.002 - 1.030
CHEMICAL EXAMINA				
REACTION		ACIDIC		
-	TANCE SPECTROPHOTOMETRY			
PROTEIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR		Negative		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY			50.75
pH by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5
BILIRUBIN		Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC NITRITE	TANCE SPECTROPHOTOMETRY	Negativo		
	TANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY	Negative		
BLOOD		Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
ASCORBIC ACID by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		NEGATIVE (-VE)		

MICROSCOPIC EXAMINATION



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

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

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

NAME	: Mr. VICKY YADAV			
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Test Name		Value	Unit	Biological Reference interval
RED BLOOD CELLS (F	RBCs) CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3
PUS CELLS	CENTRIFUGED URINARY SEDIMENT	3-4	/HPF	0 - 5
EPITHELIAL CELLS		1-3	/HPF	ABSENT

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
CRYSTALS	NEGATIVE (-ve)	NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
CASTS	NEGATIVE (-ve)	NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
BACTERIA	NEGATIVE (-ve)	NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
OTHERS	NEGATIVE (-ve)	NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	· ,	
TRICHOMONAS VAGINALIS (PROTOZOA)	ABSENT	ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		

*** End Of Report ***





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