



	Dr. Vinay Chopr MD (Pathology & Mice Chairman & Consultar	robiology)		(Pathology)
NAME	: Master. AADVIK			
AGE/ GENDER	: 7 YRS/MALE		PATIENT ID	: 1694041
COLLECTED BY	:		REG. NO./LAB NO.	: 012412080028
REFERRED BY	:		REGISTRATION DATE	: 08/Dec/2024 12:57 PM
BARCODE NO.	: 01522166		COLLECTION DATE	: 08/Dec/2024 12:58PM
CLIENT CODE. CLIENT ADDRESS	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMB		REPORTING DATE	: 08/Dec/2024 01:31PM
CLIENT ADDRESS	. 05457 I, MCHOLSON ROAD, AND			
Test Name		Value	Unit	Biological Reference interval
	SWAST	HVA WF	LLNESS PANEL: 1.0	
			OOD COUNT (CBC)	
RED BLOOD CELLS	S (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H		12.7	gm/dL	12.0 - 16.0
by CALORIMETRIC		F 1F	Ű	
RED BLOOD CELL (by HYDRO DYNAMIC F	(RBC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	5.15	Millions/	ícmm 3.50 - 5.50
PACKED CELL VOL	UME (PCV) automated hematology analyzer	40.1	%	35.0 - 49.0
MEAN CORPUSCUL	AR VOLUME (MCV)	77.8 ^L	fL	80.0 - 100.0
	AUTOMATED HEMATOLOGY ANALYZER LAR HAEMOGLOBIN (MCH)	24.7 ^L	pg	27.0 - 34.0
	AUTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC)		g/dL	32.0 - 36.0
	AUTOMATED HEMATOLOGY ANALYZER	31.8 ^L	Ŭ	32.0 - 30.0
	SUTION WIDTH (RDW-CV) AUTOMATED HEMATOLOGY ANALYZER	13.7	%	11.00 - 16.00
RED CELL DISTRIB	UTION WIDTH (RDW-SD)	40.1	fL	35.0 - 56.0
MENTZERS INDEX	AUTOMATED HEMATOLOGY ANALYZER	15.11	RATIO	BETA THALASSEMIA TRAIT: <
by CALCULATED				13.0
				IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INI	DEX	20.73	RATIO	BETA THALASSEMIA TRAIT:<=
by CALCULATED				65.0 IRON DEFICIENCY ANEMIA: >
				65.0
WHITE BLOOD CE				
TOTAL LEUCOCYTI	E COUNT (TLC) y by sf cube & microscopy	15800 ^H	/cmm	5000 - 15000
NUCLEATED RED H	BLOOD CELLS (nRBCS)	NIL		0.00 - 20.00
,	RT HEMATOLOGY ANALYZER BLOOD CELLS (nRBCS) %	NIL	%	< 10 %
	AUTOMATED HEMATOLOGY ANALYZER			. 10 /0





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist CEO & Consu DVIK E PATIENT ID REG. NO./LAB NO. REGISTRATION DAT

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

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	58	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	32	%	20 - 45
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	7	%	3 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by flow cytometry by Sf cube & microscopy	9164 ^H	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by flow cytometry by sf cube & microscopy	5056 ^H	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	474 ^H	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1106 ^H	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by flow cytometry by sf cube & microscopy	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	319000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.34	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	11	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	95000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	29.7	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.3	%	15.0 - 17.0



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Test Name	Value	e Unit	Biological Reference interval



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LIENT ADDRESS	: 6349/1, NICHOLSON]	ROAD, AMBALA CANTI	ſ	
lest Name		Value	Unit	Biological Reference interval
s C-reactive protein b. This test may also ystemic lupus erythe CONDITION WITH LOW	be used to monitor diseas ematosus N ESR	se activity and response	e to therapy in both of the a	picallý used in conjunctiŏn with other test such bove diseases as well as some others, such as
IOW LSD can be coo	i with conditions that him	indit the normal seume		uch as a high rod blood coll count
polycythaemia), sigr is sickle cells in sickl IOTE:	ificantly high white blooc e cell anaemia) also lowe e protein (C-RP) are both i	er the ESR.	is) , and some protein abno	uch as a high red blood cell count rmalities. Some changes in red cell shape (such





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	MD (Path	ay Chopra ology & Microbiology) & Consultant Pathologist	Dr. Yugam C MD (Par CEO & Consultant Pat	thology)
NAME	: Master. AADVIK			
AGE/ GENDER	: 7 YRS/MALE	PATI	ENT ID :	1694041
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPC	DRTING DATE :	08/Dec/2024 01:49PM
CLIENT ADDRESS	: 6349/1, NICHOLSON 1	ROAD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	Cl	LINICAL CHEMISTRY	/BIOCHEMISTRY	Y
		GLUCOSE FAS	TING (F)	
GLUCOSE FASTING	G (F): PLASMA SE - PEROXIDASE (GOD-POD)	96.04	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

IN ACCRDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES: 1. A fasting plasma glucose level below 100 mg/dl is considered normal. 2. 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. 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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		Chopra y & Microbiology) consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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Test Name		Value	Unit	Biological Reference interval
		I IPIN PRA	FILE : BASIC	
CHOLESTEROL TO		145.74	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL O		145.74	nig/ uL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSE	ERUM PHATE OXIDASE (ENZYMATIC)	118.81	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
	L (DIRECT): SERUM	44.03	mg/dL	VERY HIGH: > OR = 500.0 LOW HDL: < 30.0
by SELECTIVE INHIBIT	70N			BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		77.95	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 CHOLES' by CALCULATED, SPE		101.71	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTER(23.76	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEF by CALCULATED, SPE	RUM	410.29	mg/dL	350.00 - 700.00
CHOLESTEROL/HI by CALCULATED, SPE		3.31	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0



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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S		1.77	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H	IDL RATIO: SERUM	2.7 ^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.

 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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EXCELLENCE IN HEALTHCARE & DIAGNOSTICS
Dr. Yugam Chopra

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Test Name	Value	Unit	Biological Reference interva

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LIVER	FUNCTION TEST (CO	MPLETE)	
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.2	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.06	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.14	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	23.8	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	22	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.08	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	174.24	U/L	50.00 - 370.00
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	15.47	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.32	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.38	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.94	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by calculated, spectrophotometry	1.49	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:

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





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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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Test Name		Value	Unit	Biological Reference interval
	KIDN	EY FUNCTION	NTEST (COMPLETE))
UREA: SERUM by UREASE - GLUTAN	IATE DEHYDROGENASE (GLDH)	15.51	mg/dL	10.00 - 50.00
CREATININE: SERU		0.71	mg/dL	0.40 - 1.40
BLOOD UREA NITE by CALCULATED, SPE	COGEN (BUN): SERUM	7.25	mg/dL	7.0 - 25.0
BLOOD UREA NITE RATIO: SERUM by CALCULATED, SPE	COGEN (BUN)/CREATININE	10.21	RATIO	10.0 - 20.0
UREA/CREATININ	E RATIO: SERUM	21.85	RATIO	
URIC ACID: SERUM	[2.13 ^L	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE		10.07	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE	ERUM	4.66	mg/dL	2.30 - 4.70

by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY
ELECTROLYTES
SODIUM: SERUM 141 mmol/L
by ISE (ION SELECTIVE ELECTRODE)
POTASSIUM: SERUM 4.55 mmol/L
by ISE (ION SELECTIVE ELECTRODE)
CHLORIDE: SERUM 105.75 mmol/L
by ISE (ION SELECTIVE ELECTRODE)
ESTIMATED GLOMERULAR FILTERATION RATE

146

ESTIMATED GLOMERULAR FILTERATION RATE (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.

3. GI haemorrhage.



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135.0 - 150.0

3.50 - 5.00

90.0 - 110.0





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Test Name		Va	alue	Unit	Biolog	gical Refer	ence interv
8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia	superimposed on ren	nine production) ticoids) REATININE LEVELS: tionately more than al disease.	n creatinine) (e.g. obst	ructive uropa	thy).		
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr Low protein diet ar Severe liver diseas 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 their 	ass (subnormal creati tetracycline, glucocor 0:1) WITH ELEVATED ((BUN rises dispropor superimposed on ren 0:1) WITH DECREASED osis. ad starvation. by creased urea synthesi urea rather than crea monemias (urea is vir of inappropiate antidiu 0:1) WITH INCREASED py (accelerates conve eleases muscle creati who develop renal fail sis (acetoacetate caus creased BUN/creatini apy (interferes with c	nine production) ticoids) CREATININE LEVELS: tionately more than al disease. DBUN : S. tinine diffuses out tually absent in blc uretic harmone) duc CREATININE: rsion of creatine to nine). lure. ses false increase ir ne ratio). reatinine measuren	n creatinine) (e.g. obst of extracellular fluid). od). e to tubular secretion creatinine). n creatinine with certa	of urea.		ormal ratio v	when dehydr
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr Low protein diet ar Severe liver diseas 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 their 	ass (subnormal creati tetracycline, glucocor 0:1) WITH ELEVATED ((BUN rises dispropor superimposed on ren 0:1) WITH DECREASED osis. ad starvation. by creased urea synthesi urea rather than crea monemias (urea is vir of inappropiate antidiu 0:1) WITH INCREASED py (accelerates conve eleases muscle creati who develop renal fail sis (acetoacetate caus creased BUN/creatini apy (interferes with c ULAR FILTERATION RAT	nine production) ticoids) CREATININE LEVELS: tionately more than al disease. DBUN : S. tinine diffuses out tually absent in blc uretic harmone) duc CREATININE: rsion of creatine to nine). lure. ses false increase ir ne ratio). reatinine measuren	n creatinine) (e.g. obst of extracellular fluid). od). e to tubular secretion creatinine). n creatinine with certa	of urea. in methodolo			when 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 diseas Other causes of de Repeated dialysis (SIADH (syndrome of Pregnancy. DECREASED RATIO (Rhabdomyolysis (r Muscular patients Muscular patients Mappropiate RATIO Diabetic ketoacido Should produce an in Cephalosporin their ESTIMATED GLOMERI CKD STAGE 	ass (subnormal creati tetracycline, glucocor 0:1) WITH ELEVATED ((BUN rises dispropor superimposed on ren 0:1) WITH DECREASED osis. Ind starvation. 2: creased urea synthesi urea rather than crea monemias (urea is vir of inappropiate antidiu 0:1) WITH INCREASED py (accelerates conve eleases muscle creati who develop renal fai : sis (acetoacetate caus creased BUN/creatini apy (interferes with c ULAR FILTERATION RAT DES Normal k	nine production) ticoids) CREATININE LEVELS: tionately more than al disease. DBUN : s. tinine diffuses out tually absent in blc uretic harmone) duc CREATININE: rsion of creatine to nine). lure. ses false increase in ne ratio). reatinine measuren <u>E:</u> CRIPTION idney function	n creatinine) (e.g. obst of extracellular fluid). od). e to tubular secretion creatinine). n creatinine with certa nent). <u>GFR (mL/min/1.73n</u> >90	of urea. in methodolo	gies,resulting in no SOCIATED FINDING No proteinuria	S	when dehydr
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 an 3. Severe liver diseas 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 OKD STAGE	ass (subnormal creati tetracycline, glucocor 0:1) WITH ELEVATED ((BUN rises dispropor superimposed on ren 0:1) WITH DECREASED osis. Ind starvation. 2. creased urea synthesi urea rather than crea monemias (urea is vir of inappropiate antidiu 0:1) WITH INCREASED py (accelerates conve eleases muscle creati who develop renal fai : sis (acetoacetate caus creased BUN/creatini apy (interferes with c ULAR FILTERATION RAT DES Normal k	nine production) ticoids) CREATININE LEVELS: tionately more than al disease. DBUN : S. tinine diffuses out tually absent in blc uretic harmone) duc CREATININE: rsion of creatine to nine). lure. Ses false increase in ne ratio). reatinine measuren <u>E:</u> CRIPTION idney function damage with	n creatinine) (e.g. obst of extracellular fluid). od). e to tubular secretion creatinine). n creatinine with certa nent). GFR (mL/min/1.73n	of urea. in methodolo	gies,resulting in no SOCIATED FINDING No proteinuria esence of Protein ,	<u>s</u>	when dehydr
A. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Severe liver diseas Other causes of de Severe liver diseas Other causes of de Severe liver diseas Nother causes of de Severe liver diseas A Other causes of de Severe liver diseas A Other causes of de Severe liver diseas Nother causes of de Severe liver diseas Other causes of de Severe liver diseas Nother causes of de Severe liver diseas Severe liver Severe liver dise Severe liver diseas Severe liver	ass (subnormal creati tetracycline, glucocor 0:1) WITH ELEVATED ((BUN rises dispropor superimposed on ren 0:1) WITH DECREASED osis. ad starvation. 2: creased urea synthesi urea rather than crea monemias (urea is vir of inappropiate antidiu 0:1) WITH INCREASED py (accelerates conve eleases muscle creati who develop renal fai : sis (acetoacetate caus creased BUN/creatini apy (interferes with c ULAR FILTERATION RAT DES Normal k Kidney of normal	nine production) ticoids) CREATININE LEVELS: tionately more than al disease. DBUN : S. tinine diffuses out tually absent in blc uretic harmone) duc CREATININE: rsion of creatine to nine). lure. Ses false increase in ne ratio). reatinine measuren E: CRIPTION idney function damage with or high GFR	n creatinine) (e.g. obst of extracellular fluid). od). e to tubular secretion creatinine). n creatinine with certa nent). GFR (mL/min/1.73m >90 >90	of urea. in methodolo	gies,resulting in no SOCIATED FINDING No proteinuria	<u>s</u>	when dehydr
A. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Nuscular patients Muscular patients MapPROPIATE RATIO Loiabetic ketoacido should produce an in Cephalosporin there STIMATED GLOMERI CKD STAGE G1 G2 G3a	ass (subnormal creati tetracycline, glucocor 0:1) WITH ELEVATED ((BUN rises dispropor superimposed on ren 0:1) WITH DECREASED osis. Ind starvation. 2: creased urea synthesi urea rather than crea monemias (urea is vir of inappropiate antidiu 0:1) WITH INCREASED py (accelerates conve eleases muscle creati who develop renal fai : sis (acetoacetate caus creased BUN/creatini apy (interferes with c UAR FILTERATION RAT DES Normal k Kidney of normal	nine production) ticoids) CREATININE LEVELS: tionately more than al disease. DBUN : S. tinine diffuses out tually absent in blc uretic harmone) duc CREATININE: rsion of creatine to nine). lure. Ses false increase in ne ratio). reatinine measuren E: CRIPTION idney function damage with or high GFR crease in GFR	n creatinine) (e.g. obst of extracellular fluid). od). e to tubular secretion creatinine). n creatinine with certa nent). GFR (mL/min/1.73m >90 >90 60 -89	of urea. in methodolo	gies,resulting in no SOCIATED FINDING No proteinuria esence of Protein ,	<u>s</u>	when dehydr
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 an 3. Severe liver diseas 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 G1 G2	ass (subnormal creati tetracycline, glucocor 0:1) WITH ELEVATED ((BUN rises dispropor superimposed on ren 0:1) WITH DECREASED osis. Ind starvation. 2: creased urea synthesi urea rather than crea monemias (urea is vir of inappropiate antidiu 0:1) WITH INCREASED py (accelerates conve eleases muscle creati who develop renal fai : sis (acetoacetate caus creased BUN/creatini apy (interferes with c UAR FILTERATION RAT DES Normal k Kidney of normal Mild dec Moderate	nine production) ticoids) CREATININE LEVELS: tionately more than al disease. DBUN : S. tinine diffuses out tually absent in blc uretic harmone) duc CREATININE: rsion of creatine to nine). lure. Ses false increase in ne ratio). reatinine measuren E: CRIPTION idney function damage with or high GFR	n creatinine) (e.g. obst of extracellular fluid). od). e to tubular secretion creatinine). n creatinine with certa nent). GFR (mL/min/1.73m >90 >90	of urea. in methodolo	gies,resulting in no SOCIATED FINDING No proteinuria esence of Protein ,	<u>s</u>	when dehydr





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	Dr. Vinay Chopra MD (Pathology & Microbiolog, Chairman & Consultant Pathol		(Pathology)
NAME	: Master. AADVIK		
AGE/ GENDER	: 7 YRS/MALE	PATIENT ID	: 1694041
COLLECTED BY	:	REG. NO./LAB NO.	: 012412080028
REFERRED BY	:	REGISTRATION DATE	: 08/Dec/2024 12:57 PM
BARCODE NO.	: 01522166	COLLECTION DATE	:08/Dec/2024 12:58PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	:08/Dec/202401:57PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CAN	NTT	
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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	MD (Pathology & I Chairman & Const		MD (CEO & Consultant	Pathology) Pathologist
IAME	: Master. AADVIK			
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BARCODE NO.	: 01522166	COLLE	CTION DATE	:08/Dec/2024 12:58PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPOF	TING DATE	:08/Dec/202401:49PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
	IMM	UNOPATHOLOG	Y/SEROLOGY	
	(C-REACTIVE PROT	EIN (CRP)	
	EIN (CRP) QUANTITATIVE:	1.05	mg/L	0.0 - 6.0
SERUM by NEPHLOMETRY	LIN (ORF) QUANTITATIVE.			
SERUM by NEPHLOMETRY INTERPRETATION: 1. C-reactive protein	(CRP) is one of the most sensitive	acute-phase reactants f	or inflammation.	inflommation surgery or populatio
SERUM by NEPHLOMETRY INTERPRETATION: 1. C-reactive protein	(CRP) is one of the most sensitive	acute-phase reactants f ire) after severe trauma	or inflammation. , bacterial infection	, inflammation, surgery, or neoplastic

5. Elevated values are consistent with an acute inflammatory process. **NOTE:**

KOS Diagnostic Lab (A Unit of KOS Healthcare)

Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
 Oral contraceptives may increase CRP levels.





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		/ & Microbiology)		(Pathology)
	Chairman & Co	onsultant Pathologist	CEO & Consultant	Pathologist
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BARCODE NO.	:01522166		LECTION DATE	: 08/Dec/2024 12:58PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		PORTING DATE	: 08/Dec/2024 03:03PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANT I		
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PA	THOLOGY	
	UDINE D	OUTINE & MICRO		ATION
PHYSICAL EXAMIN			SCOPIC EXAMINA	ATION
QUANTITY RECIEV		10	ml	
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
COLOUR by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	PALE YELLO	W	PALE YELLOW
TRANSPARANCY	TANCE SPECTROPHOTOMETRY	CLEAR		CLEAR
SPECIFIC GRAVITY		1.02		1.002 - 1.030
CHEMICAL EXAMI	TANCE SPECTROPHOTOMETRY			
REACTION		ACIDIC		
PROTEIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	6		5.0 - 7.5
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
BILIRUBIN by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
NITRITE by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN	TANCE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0
KETONE BODIES		Negative		NEGATIVE (-ve)
BLOOD		Negative		NEGATIVE (-ve)
ASCORBIC ACID	TANCE SPECTROPHOTOMETRY	NEGATIVE (-•	ve)	NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
MICROSCOPIC EXA		NECATIVE (.	ve) /HPF	0.2
RED BLOOD CELLS	(RDUS)	NEGATIVE (-	ve) / HPF	0 - 3



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.







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

: Master. AADVIK		
: 7 YRS/MALE	PATIENT ID	: 1694041
:	REG. NO./LAB NO.	: 012412080028
:	REGISTRATION DATE	: 08/Dec/2024 12:57 PM
: 01522166	COLLECTION DATE	:08/Dec/2024 12:58PM
: KOS DIAGNOSTIC LAB	REPORTING DATE	:08/Dec/202403:03PM
: 6349/1, NICHOLSON ROAD, AMBALA CANT	Г	
Value	Unit	Biological Reference interval
	: 7 YRS/MALE : : : 01522166 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMBALA CANT	: 7 YRS/MALEPATIENT ID:REG. NO./LAB NO.:REGISTRATION DATE: 01522166COLLECTION DATE: KOS DIAGNOSTIC LABREPORTING DATE: 6349/1, NICHOLSON ROAD, AMBALA CANTT

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-3	/HPF	0 - 5
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





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		hopra & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)	
NAME	: Master. AADVIK				
AGE/ GENDER	: 7 YRS/MALE	PATI	ENT ID	: 1694041	
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BARCODE NO.	:01522166	COLL	ECTION DATE	:08/Dec/2024 12:58PM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	DRTING DATE	:08/Dec/202401:56PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT			
Test Name		Value	Unit	Biological Refe	rence interval
Test Name	N-TERMINAL PR	SPECIAL INVEST	FIGATIONS		erence interval
N-TERMINA PRO E (NT-PRO BNP) by Elfa (ENZYME LIN	N-TERMINAL PR TYPE NATRIURETIC PEPTI iked flourescent assay)	SPECIAL INVEST O B TYPE NATRIUR	FIGATIONS		rence interval
N-TERMINA PRO E (NT-PRO BNP) by Elfa (ENZYME LIN	TYPE NATRIURETIC PEPTI	SPECIAL INVEST O B TYPE NATRIUR	FIGATIONS RETIC PEPTIDE pg/mL	(NT-PRO BNP)	rence interval
N-TERMINA PRO E (NT-PRO BNP) by Elfa (ENZYME LIN	TYPE NATRIURETIC PEPTI iked flourescent assay) AGE AND CONDITION	SPECIAL INVEST O B TYPE NATRIUR DE 16.7	FIGATIONS RETIC PEPTIDE pg/mL	(NT-PRO BNP)	rence interval
N-TERMINA PRO B (NT-PRO BNP) <i>by elfa (enzyme lin</i> INTERPRETATION: AGE (TYPE NATRIURETIC PEPTI iked flourescent Assay) AGE AND CONDITION IN Years)	SPECIAL INVEST O B TYPE NATRIUR DE 16.7 RELATED CUT OFF VALUES IACUTE HEART FAILURE UNITS (pg/mL)	FIGATIONS RETIC PEPTIDE pg/mL	(NT-PRO BNP) < 300	rence interval
(NT-PRO BNP) by ELFA (ENZYME LIN INTERPRETATION: AGE (TYPE NATRIURETIC PEPTI iked flourescent assay) AGE AND CONDITION	SPECIAL INVEST O B TYPE NATRIUR DE 16.7 RELATED CUT OFF VALUES	FIGATIONS RETIC PEPTIDE pg/mL S FOR NT-PRO BNP	(NT-PRO BNP) < 300	rence interval

>75 pg/mL 450 NEGATIVE PREDICTIVE VALUE CUT OFF FOR NT-PRO BNP: < 300 pg/ml (HEART FAILUE UNLIKELY)

IN CHRONIC HEART FAILURE

pg/mL

The N-terminal of the prohormone brain natriuretic peptide (NT-proBNP), is a 76 amino acid terminal inactive protein that is cleaved from proBNP to release brain natriuretic peptide.

The main physiological function of NP is homeostasis and protection of among others the cardiovascular (CV) system from the effects of volume overload. They play an important role in regulating blood pressure (BP) and body fluid volume by their natriuretic and diuretic actions, arterial dilatation, and inhibition of the renin angiotensin system.

Concentrations of NP increase in patients with congestive heart failure (CHF) and other CV diseases owing to pressure and volume overload, whereas levels below cutoff are a strong negative predictor for CHF.

Both BNP and NT-proBNP levels in the blood are used for screening, diagnosis of acute congestive heart failure (CHF) and may be useful to establish prognosis in heart failure, as both markers are typically higher in patients with worse outcome. The plasma concentrations of both BNP and NT-proBNP are also typically increased in patients with asymptomatic or symptomatic left ventricular dysfunction and is associated with coronary artery disease and myocardial ischemia

It can be used, along with other cardiac biomarkers test, to detect heart stress and damage and/or along with lung function tests to distinguish between causes of shortness of breath. Heart failure can be confused with other conditions, and it may co-exist with them. BNP and NT-proBNP



< 75

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Test Name		/alue Unit	Biological Reference interval

levels can help doctors differentiate between heart failure and other problems, such as lung disease. An accurate diagnosis is important because the treatments are often different and must be started as soon as possible.

A BNP or NT-proBNP test may be ordered when a person has signs and symptoms that could be due to heart failure. These may include: 1.Difficulty breathing, shortness of breath

2.Fatigue

3.Swelling in the feet, ankles, legs, abdomen

NOTE:

1.Lack of NT-ProBNP elevation has been reported if Congestive Heart Failure (CHF) is very acute (first hour) or if there is Ventricular inflow obstruction

2.As per a number of studies, threshold for NT-ProBNP is 125 pg/mL

3.BNP and NT-proBNP levels decrease in most people who are taking drug therapies for heart failure, such as angiotensin-converting enzyme (ACE) inhibitors, beta blockers and diuretics.

4.Levels of both BNP and NT-proBNP tend to increase with age.

5.Levels of NT-proBNP and BNP may be increased in persons with kidney disease due to reduced clearance.

6. While both BNP and NT-proBNP will rise with left ventricle dysfunction and either can be measured for diagnosis or monitoring therapy, they are not interchangeable and the results cannot be directly compared.

7.Results to be clinically correlated.

CLINICAL USE:

1.As an aid in the diagnosis of suspected cases of CHF

2. Detection of mild forms of cardiac dysfunction

3.To assess severity of heart failure in already diagnosed cases of CHF

4.For risk stratification of patients with Acute Coronary Syndrome & CHF For monitoring therapy in patients with Left Ventricular dysfunction

*** End Of Report ***





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