

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	<b>Dr. Vinay Chopr</b> MD (Pathology & Micr Chairman & Consultar	robiology)		(Pathology)	
AME	: Mr. PARAMJEET SINGH				
GE/ GENDER	: 60 YRS/MALE		PATIENT ID	: 1684538	
OLLECTED BY	: SURJESH		REG. NO./LAB NO.	:012411280014	
EFERRED BY	:		<b>REGISTRATION DATE</b>	: 28/Nov/2024 09:33 AM	
ARCODE NO.	: 01521587		COLLECTION DATE	: 28/Nov/2024 09:45AM	
LIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 28/Nov/2024 10:18AM	
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CANTI	2		
Cest Name		Value	Unit	Biological Referen	nce interval
	SWAST	THYA W	ELLNESS PANEL: G	i	
	COME	PLETE BI	OOD COUNT (CBC)		
ED BLOOD CELLS	(RBCS) COUNT AND INDICES				
IAEMOGLOBIN (H		13.2	gm/dL	12.0 - 17.0	
ED BLOOD CELL (	RBC) COUNT	4.91	Millions	/cmm 3.50 - 5.00	
ACKED CELL VOLU		42.2	%	40.0 - 54.0	
IEAN CORPUSCUL	AR VOLUME (MCV) UTOMATED HEMATOLOGY ANALYZER	85.9	fL	80.0 - 100.0	
	AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	26.9 <sup>L</sup>	pg	27.0 - 34.0	
MEAN CORPUSCUL	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	31.3 <sup>L</sup>	g/dL	32.0 - 36.0	
	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	14.9	%	11.00 - 16.00	
ED CELL DISTRIB	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	48.2	fL	35.0 - 56.0	
IENTZERS INDEX		17.49	RATIO	BETA THALASSE 13.0 IRON DEFICIENC >13.0	
REEN & KING INE by CALCULATED		26.08	RATIO	BETA THALASSE 65.0 IRON DEFICIENC 65.0	
VHITE BLOOD CE	LLS (WBCS)				
OTAL LEUCOCYTE	BY SF CUBE & MICROSCOPY	6950	/cmm	4000 - 11000	
	SLOOD CELLS (nRBCS)	NIL		0.00 - 20.00	
UCLEATED RED B	RT HEMATOLOGY ANALYZER				

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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

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

NAME	: Mr. PARAMJEET SINGH		
AGE/ GENDER	: 60 YRS/MALE	PATIENT ID	: 1684538
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Test Name	Value	Unit	<b>Biological Reference interval</b>

Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Test Name	Value	Unit	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	59	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	34	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	2 - 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	4101	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2363	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	70	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	417	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRANULOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0.0 - 999.0
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	178000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.27	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	15 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	109000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	61.5 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence	16	%	15.0 - 17.0



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NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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CLIENT CODE. CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM		ONTING DATE	. 28/ NOV/ 2024 02.331 M
CLIENT ADDRESS	. 0349/ I, NICHOLSON KOAD, AN	IDALA CANTI		
Test Name		Value	Unit	Biological Reference interva
	OT NOOM			7)
WHOLE BLOOD	EMOGLOBIN (HbA1c):	SYLATED HAEM 6.4	OGLOBIN (HBA1( %	<b>C)</b> 4.0 - 6.4
WHOLE BLOOD by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI				
WHOLE BLOOD by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI	EMOGLOBIN (HbA1c): rmance liquid chromatography) GE PLASMA GLUCOSE	6.4 136.98	% mg/dL	4.0 - 6.4
WHOLE BLOOD by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION:	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	6.4 136.98 IABETES ASSOCIATIO	% mg/dL N (ADA): SYLATED HEMOGLOGIB	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFON ESTIMATED AVERA by HPLC (HIGH PERFON INTERPRETATION:	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years	6.4 136.98 IABETES ASSOCIATIO	% mg/dL N (ADA): SYLATED HEMOGLOGIB <5.7	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFON ESTIMATED AVERA by HPLC (HIGH PERFON INTERPRETATION: NOT dia Non dia A	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	6.4 136.98 IABETES ASSOCIATIO	% mg/dL N (ADA): SYLATED HEMOGLOGIB <5.7 5.7 - 6.4	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFON ESTIMATED AVERA by HPLC (HIGH PERFON INTERPRETATION: NOT dia Non dia A	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years	6.4 136.98 IABETES ASSOCIATIO	% mg/dL N (ADA): SYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION: Non dia A D	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes) iagnosing Diabetes	6.4 136.98 ABETES ASSOCIATION GLYCOS Goals of Th	% mg/dL N (ADA): <u>SYLATED HEMOGLOGIB</u> <5.7 5.7 – 6.4 >= 6.5 Age > 19 Years nerapy:	4.0 - 6.4 60.00 - 140.00 (HBAIC) in % < 7.0
WHOLE BLOOD by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION: Non dia A D	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	6.4 136.98	% mg/dL N (ADA): <u>SYLATED HEMOGLOGIB</u> <5.7 5.7 – 6.4 >= 6.5 Age > 19 Years nerapy:	4.0 - 6.4 60.00 - 140.00 (HBAIC) in %

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## COMMENTS:

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients. 2.Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

3. Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropriate.

4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications 5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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LIENT CODE.	: KOS DIAGNOSTIC LAB	R	EPORTING DATE	: 28/Nov/2024 10:39AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Fest Name		Value	Unit	<b>Biological Reference interval</b>
polycythaemia), sign	n with conditions that inhibit the r	unt (leucocytosis) , R.	tion of red blood cells, su and some protein abnor	uch as a high red blood cell count rmalities. Some changes in red cell shape (suc



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CLIENT CODE.	: KOS DIAGNOST	FIC LAB	R	EPORTING DATE	: 28/Nov/2024 03:54PM
CLIENT ADDRESS	: 6349/1, NICHO	DLSON ROAD,	AMBALA CANTT		
Test Name			Value	Unit	<b>Biological Reference interval</b>
		CLINI	CAL CHEMIST	RY/BIOCHEMIST	'RY
			<b>GLUCOSE F</b>	ASTING (F)	
GLUCOSE FASTIN	G (F): PLASMA Se - peroxidase (go	)D-POD)	123.23 <sup>H</sup>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0

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. 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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Test Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PROF	ILE : BASIC	
CHOLESTEROL TO	TAL · SERUM	166.42	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL O		100.42	ing/ uL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSE	ERUM HATE OXIDASE (ENZYMATIC)	142.91	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
HDL CHOLESTERO	L (DIRECT): SERUM	39.83	mg/dL	VERY HIGH: > OR = 500.0 LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0
LDL CHOLESTEROI by CALCULATED, SPE		98.01	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 VEDV MCL - OD - 100.0
NON HDL CHOLEST by CALCULATED, SPE		126.59	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
VLDL CHOLESTER(		28.58	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER by CALCULATED, SPE	RUM	475.75	mg/dL	350.00 - 700.00
CHOLESTEROL/HE by CALCULATED, SPE	DL RATIO: SERUM	4.18	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		2.46	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	IDL RATIO: SERUM	3.59	RATIO	3.00 - 5.00

## **INTERPRETATION:**

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

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

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

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





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			TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI	: SERUM PECTROPHOTOMETRY	0.88	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.21	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE by CALCULATED, SPE	CT (UNCONJUGATED): SERUM	0.67	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	[ /RIDOXAL PHOSPHATE	15.5	U/L	7.00 - 45.00
SGPT/ALT: SERUM		18.2	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE		0.85	RATIO	0.00 - 46.00
ALKALINE PHOSPI by para nitrophen propanol	HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL	87.12	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTROF	L TRANSFERASE (GGT): SERUM PHTOMETRY	18.76	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.22	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		4.37	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	1	1.85 <sup>L</sup>	gm/dL	2.30 - 3.50
A : G RATIO: SERUI	M	2.36 <sup>H</sup>	RATIO	1.00 - 2.00

by CALCULATED, SPECTROPHOTOMETRY

## INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

# **INCREASED:**

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)



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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





	<b>Dr. Vinay Chopra</b> MD (Pathology & Micro Chairman & Consultan	obiology) MD	n Chopra 9 (Pathology) t Pathologist
NAME	: Mr. PARAMJEET SINGH		
AGE/ GENDER	: 60 YRS/MALE	PATIENT ID	: 1684538
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012411280014
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 28/Nov/2024 09:33 AM
BARCODE NO.	: 01521587	COLLECTION DATE	: 28/Nov/2024 09:45AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 28/Nov/2024 12:09PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA CANTT	
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).

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



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SU 9001 : 2008 CERT		·	EXCELLENCE IN HEALTHCARE & I	
	<b>Dr. Vinay Cho</b> j MD (Pathology & M Chairman & Consu	licrobiology)	Dr. Yugam ( MD (P CEO & Consultant P	Pathology)
NAME	: Mr. PARAMJEET SINGH			
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Test Name		Value	Unit	Biological Reference interval
	KIDNE	Y FUNCTION TE	ST (COMPLETE)	
UREA: SERUM	MATE DEHYDROGENASE (GLDH)	25.87	mg/dL	10.00 - 50.00
CREATININE: SER		1.09	mg/dL	0.40 - 1.40
-		10.00		7.0 . 95.0
	ROGEN (BUN): SERUM	12.09	mg/dL	7.0 - 25.0
	ROGEN (BUN)/CREATININE	11.09	RATIO	10.0 - 20.0
RATIO: SERUM	ECTROPHOTOMETRY			
UREA/CREATININ	E RATIO: SERUM	23.73	RATIO	
by CALCULATED, SPI URIC ACID: SERUM	ECTROPHOTOMETRY 1	7.13	mg/dL	3.60 - 7.70
by URICASE - OXIDAS				
CALCIUM: SERUM	ECTROPHOTOMETRY	10.05	mg/dL	8.50 - 10.60
PHOSPHOROUS: SI	ERUM	2.83	mg/dL	2.30 - 4.70
by PHOSPHOMOLYBI	DATE, SPECTROPHOTOMETRY			
SODIUM: SERUM		142.3	mmol/L	135.0 - 150.0
by ISE (ION SELECTI)		142.5	IIIII01/ L	135.0 - 150.0
POTASSIUM: SERU		4.05	mmol/L	3.50 - 5.00
CHLORIDE: SERUN		106.73	mmol/L	90.0 - 110.0
by ISE (ION SELECTIN				
	MERULAR FILTERATION RATE	~~~		
ESTIMATED GLOM (eGFR): SERUM	IERULAR FILTERATION RATE	77.7		
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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	<b>Dr. Vinay Ch</b> MD (Pathology & Chairman & Con	Microbiology)	r. Yugam Chopra MD (Pathology) Consultant Pathologist	
AME	: Mr. PARAMJEET SINGH			
GE/ GENDER	: 60 YRS/MALE	PATIENT ID	: 1684538	
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EFERRED BY		REGISTRATION		024 09:33 AM
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LIENT ADDRESS	: 6349/1, NICHOLSON ROAD,		. 20/ NOV/ 20	24 12.09FM
lest Name		Value	Unit Bio	ological Reference interval
. Prerenal azotemia	superimposed on renal disease.	nore than creatinine) (e.g. obstruc	tive uropathy).	
Prerenal azotemia <b>DECREASED RATIO</b> (<         Acute tubular necr         Low protein diet al         Severe liver diseas         Other causes of de         Repeated dialysis         Inherited hyperam         SIADH (syndrome of         Pregnancy. <b>DECREASED RATIO</b> (<         Phenacimide thera         Rhabdomyolysis (r         Muscular patients <b>NAPPROPIATE RATIO</b> Diabetic ketoacido         hould produce an in         Cephalosporin thei <b>STIMATED GLOMERN</b>	a (BUN rises disproportionately m superimposed on renal disease. <b>10:1) WITH DECREASED BUN :</b> rosis. and starvation. e. ccreased urea synthesis. (urea rather than creatinine diffu imonemias (urea is virtually absect of inappropiate antidiuretic harm <b>10:1) WITH INCREASED CREATININ</b> apy (accelerates conversion of create releases muscle creatinine). who develop renal failure. bisis (acetoacetate causes false in increased BUN/creatinine ratio). rapy (interferes with creatinine m <b>JLAR FILTERATION RATE:</b>	nore than creatinine) (e.g. obstruc uses out of extracellular fluid). ent in blood). none) due to tubular secretion of u <b>VE:</b> eatine to creatinine). crease in creatinine with certain r neasurement).	rea.	
<ul> <li>Prerenal azotemia</li> <li>ECREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet ai</li> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of the syndrome of the</li></ul>	a (BUN rises disproportionately m superimposed on renal disease. <b>10:1) WITH DECREASED BUN :</b> rosis. and starvation. e. ccreased urea synthesis. (urea rather than creatinine diffu imonemias (urea is virtually absect of inappropiate antidiuretic harm <b>10:1) WITH INCREASED CREATININ</b> apy (accelerates conversion of create releases muscle creatinine). who develop renal failure. bisis (acetoacetate causes false in increased BUN/creatinine ratio). rapy (interferes with creatinine m <b>JLAR FILTERATION RATE:</b>	nore than creatinine) (e.g. obstruc uses out of extracellular fluid). ent in blood). none) due to tubular secretion of u <b>VE:</b> eatine to creatinine). crease in creatinine with certain r neasurement). GFR ( mL/min/1.73m2 )	rea. nethodologies,resulting ir	NGS
. Prerenal azotemia ECREASED RATIO (< . Acute tubular necr . Low protein diet al . Severe liver diseas . Other causes of de . Repeated dialysis . Inherited hyperam . SIADH (syndrome of . Pregnancy. ECREASED RATIO (< . Phenacimide thera . Rhabdomyolysis (r . Muscular patients VAPPROPIATE RATIO . Diabetic ketoacido hould produce an in . Cephalosporin the STIMATED GLOMERI CKD STAGE	a (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. and starvation. e. creased urea synthesis. (urea rather than creatinine diffu imonemias (urea is virtually absect of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATININ apy (accelerates conversion of create releases muscle creatinine). who develop renal failure. bis (acetoacetate causes false in increased BUN/creatinine ratio). rapy (interferes with creatinine m JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with	nore than creatinine) (e.g. obstruc uses out of extracellular fluid). ent in blood). none) due to tubular secretion of u <b>VE:</b> eatine to creatinine). crease in creatinine with certain r neasurement). GFR (mL/min/1.73m2) tion >90 th >90	rea. nethodologies,resulting ir ASSOCIATED FINDI	NGS a in ,
. Prerenal azotemia ECREASED RATIO (< . Acute tubular necr . Low protein diet al . Severe liver diseas . Other causes of de . Repeated dialysis . Inherited hyperam . SIADH (syndrome of . Pregnancy. ECREASED RATIO (< . Phenacimide thera . Rhabdomyolysis (r . Muscular patients DAPPROPIATE RATIO . Diabetic ketoacido nould produce an in . Cephalosporin the STIMATED GLOMERI CKD STAGE G1	a (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. and starvation. e. creased urea synthesis. (urea rather than creatinine diffu imonemias (urea is virtually abse of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATININ apy (accelerates conversion of create releases muscle creatinine). who develop renal failure. bis (acetoacetate causes false in increased BUN/creatinine ratio). rapy (interferes with creatinine m JLAR FILTERATION RATE: DESCRIPTION Normal kidney function	nore than creatinine) (e.g. obstruc uses out of extracellular fluid). ent in blood). none) due to tubular secretion of u <b>VE:</b> eatine to creatinine). crease in creatinine with certain r neasurement). GFR (mL/min/1.73m2) tho >90 th >90 FR	rea. nethodologies,resulting ir ASSOCIATED FINDI No proteinuria Presence of Prote	NGS a in ,
Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients JAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin the STIMATED GLOMERI G1 G2 G3a G3a G3b	a (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. and starvation. e. creased urea synthesis. (urea rather than creatinine diffu monemias (urea is virtually abse of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATININ apy (accelerates conversion of cre- releases muscle creatinine). who develop renal failure. bis (acetoacetate causes false in increased BUN/creatinine ratio). rapy (interferes with creatinine m JLAR FILTERATION RATE: DESCRIPTION Normal kidney func- Kidney damage wi normal or high GF Mild decrease in G Moderate decrease in	nore than creatinine) (e.g. obstruc uses out of extracellular fluid). ent in blood). none) due to tubular secretion of u <b>VE:</b> eatine to creatinine). crease in creatinine). <u><b>VE:</b></u> eatine to creatinine with certain r neasurement). <u><b>GFR (mL/min/1.73m2 )</b></u> tion >90 th >90 FR 60 -89 n GFR 30-59	rea. nethodologies,resulting ir ASSOCIATED FINDI No proteinuria Presence of Prote	NGS a in ,
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AGE/ GENDER	: 60 YRS/MALE	PATIENT ID	: 1684538
NAME	: Mr. PARAMJEET SINGH		
	Chairman & Cons		
	Dr. Vinay Cho MD (Pathology &		am Chopra 1D (Pathology)

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

End Of Report \*\*\*





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