

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopi MD (Pathology & Mic Chairman & Consulta	robiology)	Dr. Yugam C MD (Pa CEO & Consultant Pa	thology)
NAME	: Mr. OM PARKASH GAMBHIR			
AGE/ GENDER	: 62 YRS/MALE	PAT	IENT ID	: 1673436
COLLECTED BY	: SURJESH	REG.	NO./LAB NO.	: 012411160024
REFERRED BY	:	REG	ISTRATION DATE	: 16/Nov/2024 10:23 AM
BARCODE NO.	:01520900	COLI	LECTION DATE	: 16/Nov/2024 10:30AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		ORTING DATE	: 16/Nov/2024 10:57AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AME	BALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	SWAS	THYA WELLI	NESS PANEL: G	
	СОМ	PLETE BLOOD	COUNT (CBC)	
RED BLOOD CELLS	(RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H)	B)	12	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (1	RBC) COUNT	4.84	Millions/cm	nm 3.50 - 5.00
by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE			
PACKED CELL VOLU	JME (PCV) UTOMATED HEMATOLOGY ANALYZER	38.9 ^L	%	40.0 - 54.0
MEAN CORPUSCUL	AR VOLUME (MCV)	80.3	fL	80.0 - 100.0
	UTOMATED HEMATOLOGY ANALYZER AR HAEMOGLOBIN (MCH)	24.6 ^L	pg	27.0 - 34.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
MEAN CORPUSCUL. by CALCULATED BY A	AR HEMOGLOBIN CONC. (MCHC) utomated hematology analyzer	30.7 ^L	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV)	16.3 ^H	%	11.00 - 16.00
	UTOMATED HEMATOLOGY ANALYZER UTION WIDTH (RDW-SD)	49.3	fL	35.0 - 56.0
,	UTOMATED HEMATOLOGY ANALYZER		D. LITTO	
MENTZERS INDEX by CALCULATED		16.59	RATIO	BETA THALASSEMIA TRAIT: < 13.0
				IRON DEFICIENCY ANEMIA:
CDEEN & VINCIND	LEN.	90.99	DATIO	>13.0 BETA THALASSEMIA TRAIT:<=
GREEN & KING IND by calculated	JEX	26.83	RATIO	65.0
				IRON DEFICIENCY ANEMIA: >
WHITE BLOOD CE	IS (WRCS)			65.0
TOTAL LEUCOCYTE		8040	/cmm	4000 - 11000
by FLOW CYTOMETRY	BY SF CUBE & MICROSCOPY		, chini	
	LOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
		NIT	%	< 10 %
NUCLEATED RED B	LOOD CELLS (NKBCS) % UTOMATED HEMATOLOGY ANALYZER	NIL	70	< 10 /0





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KOS Diagnostic Lab (A Unit of KOS Healthcare)

MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Dr. Vinay Chopra



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

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	56	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	22	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	14 ^H	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	8	%	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	4502	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1769	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1126 ^H	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by flow cytometry by sf cube & microscopy	643	/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	165000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.22	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	14 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	85000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	51.5 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.4	%	15.0 - 17.0



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Test Name	Value	Unit	Biological Reference interval



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CLIENT CODE.	: KOS DIAGNOSTIC LAB		EPORTING DATE	: 16/Nov/2024 02:37PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AN		EI ORTING DATE	. 10/100/ 2024 02.571 M
CLIENT ADDRESS	. 0349/1, NICHOLSON ROAD, AN	IDALA CANTI		
Test Name		Value	Unit	Biological Reference interval
WHOLE BLOOD	EMOGLOBIN (HbA1c):	7.1 ^H	%	4.0 - 6.4
	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	157.07 ^H	mg/dL	60.00 - 140.00
	AS PER AMERICAN D	ABETES ASSOCIAT	ION (ADA):	
		GLYCOSYLATED HEMOGLOGIB (HBAIC) in %		
	REFERENCE GROUP	GLYC		(HBAIC) IN %
Non dia	REFERENCE GROUP abetic Adults >= 18 years	GLYC	<5.7	
Non dia A	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	GLYC	<5.7 5.7 – 6. 4	
Non dia A	REFERENCE GROUP abetic Adults >= 18 years	GLYC	<5.7 5.7 - 6.4 >= 6.5	
Non dia A	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)		<5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	
Non dia A D	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes) iagnosing Diabetes	Goals of	<5.7 5.7 - 6.4 >= 6.5 Age > 19 Years Therapy:	< 7.0
Non dia A D	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	Goals of	<5.7 5.7 - 6.4 >= 6.5 Age > 19 Years Therapy: Suggested:	
Non dia A D	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes) iagnosing Diabetes	Goals of Actions S	<5.7 5.7 - 6.4 >= 6.5 Age > 19 Years Therapy:	< 7.0

KOS Diagnostic Lab

(A Unit of KOS Healthcare)

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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Test Name		Value	Unit	Biological Reference interval
	ERYTHRO	CYTE SEDIMENT	ATION RATE (ESR)
mmune disease, but 2. An ESR can be affe- as C-reactive protein 3. This test may also I systemic lupus erythe CONDITION WITH LOV A low ESR can be see (polycythaemia), sign as sickle cells in sickl NOTE: 1. ESR and C - reactive 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevate 5. Women tend to ha 5. Drugs such as dext	does not tell the health practitione cted by other conditions besides in be used to monitor disease activity ematosus V ESR n with conditions that inhibit the n ificantly high white blood cell cour e cell anaemia) also lower the ESR e protein (C-RP) are both markers o s not change as rapidly as does CRF by as many other factors as is ESR, by as many other factors as is ESR, by as many other factors as is ESR, by as many other factors as is CRF by as many other factors as is CSR.	er exactly where the inf flammation. For this re- and response to thera formal sedimentation on the (leucocytosis), and s finflammation. P, either at the start of making it a better marl bes of proteins, globulir and pregnancy can cau	lammation is in the eason, the ESR is typ py in both of the a of red blood cells, su some protein abno inflammation or as ker of inflammatior is or fibrinogen. se temporary eleva	picallý 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 (such s it resolves. n .





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CLIENT ADDRESS	: 6349/1, NICHOI	SON ROAD, AMBALA CA	NTT	
Test Name		Value	Unit	Biological Reference interval
		CLINICAL CHEN	AISTRY/BIOCHEMIST	ſRY
		GLUCO	DSE FASTING (F)	
GLUCOSE FASTING	G (F): PLASMA E - peroxidase (god	1 07.2	H mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

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





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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	Biological Reference interval
	G	LUCOSE POS	T PRANDIAL (PP)	
	ANDIAL (PP): PLASMA E - PEROXIDASE (GOD-POD)	189.54 ^H	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > 0R = 200.0

INTERPRETATION IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES: 1. A post-prandial plasma glucose level below 140 mg/dl is considered normal. 2. A post-prandial glucose level between 140 - 200 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 post-prandial plasma glucose level of above 200 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.



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SO 9001:2008 CERT	IFIED LAB		EXCELLENCE IN HEALTHCARE	& DIAGNOSTICS
	Dr. Vinay Cl MD (Pathology & Chairman & Cor			(Pathology)
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Test Name		Value	Unit	Biological Reference interval
CHOLESTEROL TO by CHOLESTEROL OX		LIPID PRO 163.05)FILE : BASIC mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
TRIGLYCERIDES: S by GLYCEROL PHOSE	ERUM PHATE OXIDASE (ENZYMATIC)	166.61 ^H	mg/dL	240.0 OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERO by SELECTIVE INHIBIT	L (DIRECT): SERUM TION	57.83	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERO		71.9	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		105.22	mg/dL	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		33.32	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEP by CALCULATED, SPE	RUM	492.71	mg/dL	350.00 - 700.00
CHOLESTEROL/HI		2.82	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		1.24	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		2.88 ^L	RATIO	3.00 - 5.00

INTERPRETATION:

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

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

3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues. 4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL

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





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LIVER	FUNCTION TE	ST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.41	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.12	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.29	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	25.3	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	28.4	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.89	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para Nitrophenyl phosphatase by amino methyl propanol	81.95	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	26.33	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.03	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.18	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.85	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.47	RATIO	1.00 - 2.00

INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range. USE: - Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:

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





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





	Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist				
NAME	: Mr. OM PARKASH GAMBHIR				
AGE/ GENDER	: 62 YRS/MALE	PATIENT ID	: 1673436		
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012411160024		
REFERRED BY	:	REGISTRATION DATE	: 16/Nov/2024 10:23 AM		
BARCODE NO.	: 01520900	COLLECTION DATE	: 16/Nov/2024 10:30AM		
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 16/Nov/2024 11:42AM		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	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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Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist					
NAME	: Mr. OM PARKASH GAMBHIR				
AGE/ GENDER	: 62 YRS/MALE	PAT	IENT ID	: 1673436	
COLLECTED BY	: SURJESH	REG.	NO./LAB NO.	:012411160024	
REFERRED BY	:	REG	STRATION DATE	: 16/Nov/2024 10:23 AM	
BARCODE NO.	: 01520900	COLL	LECTION DATE	: 16/Nov/2024 10:30AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REP	DRTING DATE	: 16/Nov/2024 01:22PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
	KIDNE	Y FUNCTION T	EST (COMPLETE)		
UREA: SERUM		33.87	mg/dL	10.00 - 50.00	
	MATE DEHYDROGENASE (GLDH)		Ũ	0.40 1.40	
CREATININE: SERU		1.64 ^H	mg/dL	0.40 - 1.40	
	ROGEN (BUN): SERUM	15.83	mg/dL	7.0 - 25.0	
by CALCULATED, SPE BLOOD UREA NITE	ECTROPHOTOMETRY ROGEN (BUN)/CREATININE	9.65 ^L	RATIO	10.0 - 20.0	
RATIO: SERUM		9.05-	101110	10.0 20.0	
by CALCULATED, SPE		20.05	DATIO		
UREA/CREATININ by CALCULATED, SPE		20.65	RATIO		
URIC ACID: SERUM		7.71 ^H	mg/dL	3.60 - 7.70	
by URICASE - OXIDAS CALCIUM: SERUM	SE PEROXIDASE	9.45	mg/dL	8.50 - 10.60	
by ARSENAZO III, SPE	CTROPHOTOMETRY		ing/ uL		
PHOSPHOROUS: SE	ERUM DATE, SPECTROPHOTOMETRY	3.49	mg/dL	2.30 - 4.70	
ELECTROLYTES	ATE, SI LOTKOI HOTOMETRI				
SODIUM: SERUM		145.4	mmol/L	135.0 - 150.0	
by ISE (ION SELECTIV					
POTASSIUM: SERU		4.74	mmol/L	3.50 - 5.00	
CHLORIDE: SERUM	1	109.05	mmol/L	90.0 - 110.0	
by ISE (ION SELECTIV	'E ELECTRODE) 1ERULAR FILTERATION RATE				
	ERULAR FILTERATION RATE	17			
(eGFR): SERUM	ERULAR FILTERATION KATE	47			
by CALCULATED					
INTERPRETATION:	yoon pro, and post ronal azotomia				

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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





		Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		t CEO & Consultant Pathologist					
IAME	: Mr. OM PAR	KASH GAMBHIR							
AGE/ GENDER	: 62 YRS/MAL	Ξ	I	PATIENT ID	: 10	673436			
COLLECTED BY	: SURJESH		I	REG. NO./LAB NO.	:0	124111600	24		
REFERRED BY	·			REGISTRATION D		3/Nov/2024			
BARCODE NO.	:01520900			COLLECTION DAT		3/Nov/2024			
CLIENT CODE.	: KOS DIAGNO			REPORTING DATI	E : 10	6/Nov/2024 (J1:22PM		
CLIENT ADDRESS	: 6349/1, NIC	HOLSON ROAD, AMB	ALA CANTT						
Fest Name			Value	Un	it	Biolog	ical Refe	rence inte	erval
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia PCREASED RATIO (<1 Acute tubular necr 	ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis.	creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease.	ELS:	e) (e.g. obstructive	e uropathy).				
A. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1 G2	ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. 2: creased urea syr urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/crea apy (interferes v LAR FILTERATIO	creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : The sis. The creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creating treatinine). That failure. The causes false increase extinine ratio). with creatinine measu NATE: DESCRIPTION mal kidney function dney damage with prmal or high GFR	ELS: than creatinin but of extrace blood). due to tubula e to creatinine e in creatinin rement).	Ilular fluid). ar secretion of urea e). e with certain met <u>L/min/1.73m2)</u> >90 >90	hodologies,r ASSOCIA No p Presenc	esulting in no TED FINDINGS Toteinuria e of Protein , or cast in urin	<u>;</u>	when deh	ydrat
B. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rabdomyolysis (r Nuscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther <u>STIMATED GLOMERU</u> <u>G1</u> <u>G2</u> <u>G3a</u>	ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. 2: creased urea syn urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/crea apy (interferes v LAR FILTERATIO	creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : The creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creating treatinine). hal failure. Causes false increase extinine ratio). with creatinine measu V RATE: DESCRIPTION mal kidney function dney damage with prmal or high GFR_ Id decrease in GFR	ELS: than creatinin but of extrace blood). due to tubula e to creatinine rement).	Ilular fluid). ar secretion of urea e). e with certain met <u>L/min/1.73m2) >90 >90 60 -89</u>	hodologies,r ASSOCIA No p Presenc	FED FINDINGS roteinuria e of Protein ,	<u>;</u>	when deh	ydrat
G1 G2	ass (subnormal tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. creased urea syn urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/creased apy (interferes w LAR FILTERATIO Nor Kin Model Model	creatinine production cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : The sis. The creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creating treatinine). That failure. The causes false increase extinine ratio). with creatinine measu NATE: DESCRIPTION mal kidney function dney damage with prmal or high GFR	ELS: than creatinin but of extrace blood). due to tubula e to creatinine rement).	Ilular fluid). ar secretion of urea e). e with certain met <u>L/min/1.73m2)</u> >90 >90	hodologies,r ASSOCIA No p Presenc	FED FINDINGS roteinuria e of Protein ,	<u>;</u>	when deh	ydrat



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA 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

End Of Report ***





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