



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)	Dr. Yugam MD (f CEO & Consultant F	Pathology)
NAME	: Mr. BINTU HANDA			
AGE/ GENDER	: 56 YRS/MALE	PA	TIENT ID	: 1784621
COLLECTED BY	: SURJESH	RE	G. NO./LAB NO.	: 012503090033
REFERRED BY	:	RE	GISTRATION DATE	: 09/Mar/2025 10:16 AM
BARCODE NO.	: 01526792	CO	LLECTION DATE	:09/Mar/2025 10:30AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		PORTING DATE	: 09/Mar/2025 10:47AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
	T2 A W2	'HVA WFI I	LNESS PANEL: G	
			D COUNT (CBC)	
RED BLOOD CELLS	S (RBCS) COUNT AND INDICES	LETEDLOU		
HAEMOGLOBIN (H		11.5 ^L	gm/dL	12.0 - 17.0
by CALORIMETRIC			Ŭ	
RED BLOOD CELL (by hydro dynamic f	RBC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	3.31 ^L	Millions/c	mm 3.50 - 5.00
PACKED CELL VOL		34.4 ^L	%	40.0 - 54.0
	AUTOMATED HEMATOLOGY ANALYZER AR VOLUME (MCV)	103.9 ^H	fL	80.0 - 100.0
	AUTOMATED HEMATOLOGY ANALYZER		nd	27.0 - 34.0
	AR HAEMOGLOBIN (MCH)	34.7 ^H	pg	27.0 - 34.0
	AR HEMOGLOBIN CONC. (MCHC)	33.4	g/dL	32.0 - 36.0
RED CELL DISTRIB	UTION WIDTH (RDW-CV)	15.5	%	11.00 - 16.00
	AUTOMATED HEMATOLOGY ANALYZER UTION WIDTH (RDW-SD)	an all	fL	35.0 - 56.0
by CALCULATED BY A	AUTOMATED HEMATOLOGY ANALYZER	60.2 ^H		
MENTZERS INDEX by CALCULATED		31.39	RATIO	BETA THALASSEMIA TRAIT: < 13.0
.,				IRON DEFICIENCY ANEMIA:
	NEV	49.50	RATIO	>13.0
ODEEN & VINCINI	JEX	48.59	RATIO	BETA THALASSEMIA TRAIT:< 65.0
GREEN & KING INI				
	LLS (WBCS)			IRON DEFICIENCY ANEMIA: > 65.0
by calculated white blood ce fotal leucocyte	E COUNT (TLC)	6340	/cmm	
by CALCULATED WHITE BLOOD CE FOTAL LEUCOCYTE by FLOW CYTOMETR		6340 NIL	/cmm	65.0
by CALCULATED WHITE BLOOD CE FOTAL LEUCOCYTE by FLOW CYTOMETR NUCLEATED RED E by AUTOMATED 6 PAI	E COUNT (TLC) y by sf cube & microscopy		/cmm %	65.0 4000 - 11000

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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



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Dr. Vinay Chopra

MD (Pathology & Microbiology)



Dr. Yugam Chopra

MD (Pathology)

Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. BINTU HANDA AGE/ GENDER : 56 YRS/MALE **PATIENT ID** :1784621 **COLLECTED BY** : SURJESH :012503090033 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** :09/Mar/2025 10:16 AM : **BARCODE NO.** :01526792 **COLLECTION DATE** :09/Mar/2025 10:30AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :09/Mar/2025 10:47AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC)** NEUTROPHILS 59 % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 28 % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY 6H EOSINOPHILS % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 7 % 2 - 12by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE LEUKOCYTES (WBC) COUNT** ABSOLUTE NEUTROPHIL COUNT 3741 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 1775 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 380 /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 444 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE IMMATURE GRANULOCYTE COUNT 0.0 - 999.00 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 171000 /cmm 150000 - 450000 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.21% 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 12^H fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) 68000 /cmm 30000 - 90000 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) 39.7 % 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE

16

PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE



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

%



15.0 - 17.0





	Dr. Vinay Chopra MD (Pathology & Microbio Chairman & Consultant Pat	logy) MD	n Chopra 9 (Pathology) t Pathologist
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Test Name	Val	ue Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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	01526792		LLECTION DATE	: 09/Mar/2025 10:10/10/10/
				: 09/Mar/2025 01:34PM
	KOS DIAGNOSTIC LAB		PORTING DATE	: 09/Mar/2025 01:34PM
CLIENT ADDRESS :	6349/1, NICHOLSON ROAD,			
Test Name		Value	Unit	Biological Reference interval
	GLY	COSYLATED HAEN	AOGLOBIN (HBA1C)	
GLYCOSYLATED HAEMO		4.9	%	4.0 - 6.4
WHOLE BLOOD				
by HPLC (HIGH PERFORMANC ESTIMATED AVERAGE PI	CE LIQUID CHROMATOGRAPHY)	93.93	mg/dL	60.00 - 140.00
	CE LIQUID CHROMATOGRAPHY)	00.00	ing, ui	00.00 110.00
INTERPRETATION:				
	AS PER AMERICAN DIAE	BETES ASSOCIATION (AD)	A):	
REFER	ENCE GROUP	GLYCOSYLAT	ED HEMOGLOGIB (HBAIC) i	n %
Non diabetic	Adults >= 18 years	<5.7		
At Risk	(Prediabetes)	5.7 - 6.4		
Diagno	sing Diabetes		>= 6.5	
			Age > 19 Years	
		Goals of Therapy)
Therapeutic goa	Ils for glycemic control	Actions Suggestee	d: >8.0	
			Age < 19 Years	
		Goal of therapy	: <7.5	

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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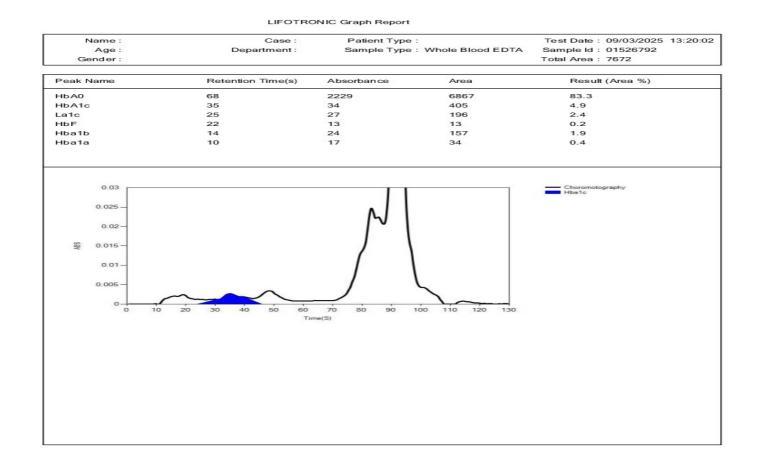


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Test Name	Va	lue Unit	Biological Reference interval
CLIENT ADDRESS	. 0549/ 1, NICHOLSON KOAD, AMDALA	CANTI	
CLIENT ADDRESS	: 6349/1. NICHOLSON ROAD. AMBALA	CANTT	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 09/Mar/2025 01:34PM
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NAME	: Mr. BINTU HANDA		
	MD (Pathology & Microbi Chairman & Consultant Pa	ology) MD	(Pathology)
	Dr. Vinay Chopra	Dr. Yugan	n Chopra







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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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BARCODE NO.	: 01526792	COLL	ECTION DATE	:09/Mar/2025 10:30AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	:09/Mar/2025 11:32AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	ERYTHRO	CYTE SEDIMENT	TATION RATE (ES	R)
by RED CELL AGGREG INTERPRETATION: 1. ESR is a non-specifi immune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythe CONDITION WITH LOV A low ESR can be see (polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactive 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dext	does not tell the health practition cted by other conditions besides in be used to monitor disease activity ematosus W ESR n with conditions that inhibit the r ificantly high white blood cell cou e cell anaemia) also lower the ESF e protein (C-RP) are both markers of s not change as rapidly as does CR by as many other factors as is ESR, ded, it is typically a result of two typ ye a higher ESR, and menstruation	often indicates the pre- er exactly where the ir nflammation. For this r y and response to ther normal sedimentation int (leucocytosis), and R. of inflammation. P, either at the start o , making it a better ma bes of proteins, globul and pregnancy can ca	nflammation is in the boreason, the ESR is typic rapy in both of the abore of red blood cells, such some protein abnorm of inflammation or as it rker of inflammation. ins or fibrinogen. use temporary elevatio	n associated with infection, cancer and auto- ody or what is causing it. ally used in conjunction with other test such ve diseases as well as some others, such as n as a high red blood cell count alities. Some changes in red cell shape (such resolves.





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CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	:09/Mar/2025 12:23PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLIN		Y/BIOCHEMIST	TRY
		GLUCOSE FA	STING (F)	
GLUCOSE FASTING by glucose oxidas	F (F): PLASMA E - PEROXIDASE (GOD-POD)	78.71	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

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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LIENT ADDRESS	: 6349/1, NICHOLSON ROA	AD, AMBALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
		LIPID PRO	FILE : BASIC	
CHOLESTEROL TO	TAL: SERUM	106.44	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		100.11	ing/ uL	BORDERLINE HIGH: 200.0 -
				239.0
				HIGH CHOLESTEROL: > OR = 240.0
RIGLYCERIDES: S		178.25 ^H	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSP	PHATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 -
				199.0 HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
IDL CHOLESTERO	L (DIRECT): SERUM	29.16 ^L	mg/dL	LOW HDL: < 30.0
by SELECTIVE INITIDITY	ION			BORDERLINE HIGH HDL: 30.0 60.0
				HIGH HDL: $> OR = 60.0$
DL CHOLESTEROI		41.63	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.
by GALGOLATED, SI L	CINCINICIOMETICI			BORDERLINE HIGH: 130.0 -
				159.0
				HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLEST	FEROL: SERUM	77.28	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPE	CTROPHOTOMETRY		Ū	ABOVE OPTIMAL: 130.0 - 159.
				BORDERLINE HIGH: 160.0 - 189.0
				HIGH: 190.0 - 219.0
		07.07	/ 1-	VERY HIGH: > OR = 220.0
LDL CHOLESTER(by CALCULATED, SPE		35.65	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER	CUM	391.13	mg/dL	350.00 - 700.00
by CALCULATED, SPE CHOLESTEROL/HD		3.65	RATIO	LOW RISK: 3.30 - 4.40
by CALCULATED, SPE		0.00	IATIO	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		1.43	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	6.11 ^H	RATIO	3.00 - 5.00

INTERPRETATION:

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

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

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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Test Name		Value	Unit	Biological Reference interval
	LIVER	FUNCTIO	ON TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, S	: SERUM PECTROPHOTOMETRY	0.38	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	Г (CONJUGATED): SERUM spectrophotometry	0.11	mg/dL	0.00 - 0.40
	ECT (UNCONJUGATED): SERUM	0.27	mg/dL	0.10 - 1.00
SGOT/AST: SERUN by IFCC, WITHOUT P	I (RIDOXAL PHOSPHATE	18.1	U/L	7.00 - 45.00
SGPT/ALT: SERUM	[/RIDOXAL PHOSPHATE	12.4	U/L	0.00 - 49.00
AST/ALT RATIO: S by calculated, spi	ERUM ECTROPHOTOMETRY	1.46	RATIO	0.00 - 46.00
ALKALINE PHOSP by para nitrophen propanol	HATASE: SERUM IYL PHOSPHATASE BY AMINO METHYL	87.23	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM	14.09	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		4.78 ^L	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL		3.18 ^L	gm/dL	3.50 - 5.50
GLOBULIN: SERUM		1.6 ^L	gm/dL	2.30 - 3.50
A : G RATIO: SERU		1.99	RATIO	1.00 - 2.00

by CALCULATED, SPECTROPHOTOMETRY

NOTE: To be correlated in individuals having SGOT and SGPT values higher than Normal Reference 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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INTERPRETATION





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Test Name	Valu	ie 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 Cho MD (Pathology & N Chairman & Consu	1icrobiology)		(Pathology)
NAME	: Mr. BINTU HANDA			
AGE/ GENDER	: 56 YRS/MALE		PATIENT ID	: 1784621
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012503090033
REFERRED BY	:		REGISTRATION DATE	: 09/Mar/2025 10:16 AM
BARCODE NO.	: 01526792		COLLECTION DATE	:09/Mar/2025 10:30AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 09/Mar/2025 01:47PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AN	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
	KIDNE	EY FUNCTIO	N TEST (COMPLETE))
UREA: SERUM by UREASE - GLUTAM	IATE DEHYDROGENASE (GLDH)	57.53 ^H	mg/dL	10.00 - 50.00
CREATININE: SERU		2.38 ^H	mg/dL	0.40 - 1.40
BLOOD UREA NITR by CALCULATED, SPE	COGEN (BUN): SERUM	26.88 ^H	mg/dL	7.0 - 25.0
BLOOD UREA NITE RATIO: SERUM by CALCULATED, SPE	COGEN (BUN)/CREATININE	11.29	RATIO	10.0 - 20.0
UREA/CREATININ by CALCULATED, SPE		24.17	RATIO	
URIC ACID: SERUM by URICASE - OXIDAS		7.04	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE		9.1	mg/dL	8.50 - 10.60
	ERUM DATE, SPECTROPHOTOMETRY	3.39	mg/dL	2.30 - 4.70
ELECTROLYTES		1.10.0	1.0	
SODIUM: SERUM by ISE (ION SELECTIV	'E ELECTRODE)	140.8	mmol/L	135.0 - 150.0
POTASSIUM: SERUE by ISE (ION SELECTIV	M	4.94	mmol/L	3.50 - 5.00
CHLORIDE: SERUM	1	105.6	mmol/L	90.0 - 110.0
ESTIMATED GLOM	IERULAR FILTERATION RATE			
ESTIMATED GLOM (eGFR): SERUM by CALCULATED INTERPRETATION:	ERULAR FILTERATION RATE	31.2		

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		Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist				
IAME	: Mr. BINTU	HANDA						
AGE/ GENDER	: 56 YRS/MAI	Æ		PATIENT ID	: 1	784621		
COLLECTED BY	: SURJESH			REG. NO./LAB NO.	•	01250309003	23	
	. SOIWLDII							
REFERRED BY	:			REGISTRATION D)9/Mar/2025 1		
BARCODE NO.	:01526792			COLLECTION DAT)9/Mar/2025 1		
CLIENT CODE.	: KOS DIAGNO	OSTIC LAB		REPORTING DATE	E : ()9/Mar/2025 0	1:47PM	
CLIENT ADDRESS	: 6349/1, NIC	HOLSON ROAD, AMI	BALA CANTT					
Test Name			Value	Uni	it	Biologi	ical Reference	e interval
9. Certain drugs (e.g. INCREASED RATIO (>20 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necro	0:1) WITH ELEV (BUN rises disp superimposed (0:1) WITH DECF	ATED CREATININE LEV proportionately more on renal disease.		ne) (e.g. obstructive	e uropathy).			
NCREASED RATIO (>20 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1	0:1) WITH ELEV. (BUN rises disperimposed of 0:1) WITH DECF osis. d starvation. creased urea sy urea rather tha monemias (urea f inappropiate 0:1) WITH INCR oy (accelerates eleases muscle who develop reasily sis (acetoacetai creased BUN/cr apy (interferes LAR FILTERATIO No No	ATED CREATININE LEV proportionately more on renal disease. REASED BUN : In creatinine diffuses a is virtually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). enal failure. te causes false increat reatinine ratio). with creatinine meas ON RATE: DESCRIPTION rmal kidney function idney damage with	than creatini out of extrac h blood).) due to tubul ne to creatinin se in creatinin urement).	ellular fluid). Iar secretion of urea ne).	hodologies,	resulting in nor ATED FINDINGS proteinuria ce of Protein ,		n dehydrai
NCREASED RATIO (>24 Postrenal azotemia Perenal azotemia CECREASED RATIO (<1 Acute tubular necro Low protein diet an Severe liver disease Other causes of dec Repeated dialysis (i CINHERITED GLOMERU CECREASED RATIO (<1 Phenacimide therap Rhabdomyolysis (re MAPPROPIATE RATIO Diabetic ketoacidos hould produce an inc Cephalosporin thera STIMATED GLOMERU CKD STAGE G1 G2	0:1) WITH ELEV. (BUN rises disperimposed of 0:1) WITH DECF osis. d starvation. creased urea sy urea rather tha monemias (urea f inappropiate 0:1) WITH INCR oy (accelerates eleases muscle who develop reasily sis (acetoacetai creased BUN/cr apy (interferes LAR FILTERATIONAL NO NO K	ATED CREATININE LEV proportionately more on renal disease. REASED BUN : In creatinine diffuses a is virtually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). enal failure. te causes false increat reatinine ratio). with creatinine meas ON RATE: DESCRIPTION rmal kidney function idney damage with tormal or high GFR_	than creatini out of extrac h blood).) due to tubul ne to creatinin se in creatinin urement).	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90 >90	hodologies, ASSOCI Presen	ATED FINDINGS		n dehydra
NCREASED RATIO (>24 1. Postrenal azotemia 2. Prerenal azotemia 3. Perenal azotemia 4. Acute tubular necro 5. Low protein diet an 3. Severe liver disease 4. Other causes of dec 5. Repeated dialysis (16 5. Inherited hyperami 7. SIADH (syndrome o 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide therap 2. Rhabdomyolysis (re 3. Muscular patients v NAPPROPIATE RATIO 2. Cephalosporin thera 5. STIMATED GLOMERU CKD STAGE G1 G2 G3a	0:1) WITH ELEV. (BUN rises disperimposed of 0:1) WITH DECF osis. d starvation. creased urea sy urea rather tha monemias (ure f inappropiate 0:1) WITH INCR oy (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes LAR FILTERATIC No K No K M	ATED CREATININE LEV proportionately more on renal disease. REASED BUN : In creatinine diffuses a is virtually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). enal failure. te causes false increat reatinine ratio). with creatinine meas IN RATE: DESCRIPTION rmal kidney function idney damage with iormal or high GFR ild decrease in GFR	than creatini out of extrac n blood).) due to tubul ne to creatinin se in creatinin urement). GFR (m	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90 >90 60 -89	hodologies, ASSOCI Presen	ATED FINDINGS proteinuria ce of Protein ,		n dehydra
NCREASED RATIO (>24 1. Postrenal azotemia 2. Prerenal azotemia 3. Prerenal azotemia 4. Acute tubular necro 5. Low protein diet an 6. Severe liver disease 6. Other causes of dec 6. Repeated dialysis (16 6. Inherited hyperami 7. SIADH (syndrome o 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide therap 2. Rhabdomyolysis (ref 8. Muscular patients v NAPPROPIATE RATIO 2. Cephalosporin therap 3. Cephalosporin therap 5. Cephalosporin therap 5. CENTATED GLOMERU 6. CAL STAGE 6. G1 6. G2	0:1) WITH ELEV. (BUN rises disp superimposed of 0:1) WITH DECF osis. d starvation. creased urea sy urea rather tha monemias (urea f inappropiate 0:1) WITH INCR oy (accelerates eleases muscle who develop reasily creased BUN/cr apy (interferes LAR FILTERATION NO K MO MO MO MO	ATED CREATININE LEV proportionately more on renal disease. REASED BUN : In creatinine diffuses a is virtually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). enal failure. te causes false increat reatinine ratio). with creatinine meas ON RATE: DESCRIPTION rmal kidney function idney damage with tormal or high GFR_	than creatini out of extrac n blood).) due to tubul ne to creatinin se in creatinin urement). GFR (m	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90 >90	hodologies, ASSOCI Presen	ATED FINDINGS proteinuria ce of Protein ,		n dehydra





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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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