



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultan	obiology)	Dr. Yugam MD ( CEO & Consultant	Pathology)
GE/ GENDER : 33 COLLECTED BY : REFERRED BY : CARCODE NO. : A CLIENT CODE. : K	ir. INDERJEET SINGH 5 YRS/MALE 1260454 OS DIAGNOSTIC SHAHBAD 349/1, NICHOLSON ROAD, AMB/	REG REG COJ REJ	FIENT ID G. NO./LAB NO. GISTRATION DATE LLECTION DATE PORTING DATE	: 1310915 <b>: 042502080002</b> : 08/Feb/2025 08:57 AM : 08/Feb/2025 03:46PM : 08/Feb/2025 04:13PM
Cest Name	949/ 1, NICHOLSON KOAD, AMD	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RE			IESS PANEL: 15.( D COUNT (CBC)	
IAEMOGLOBIN (HB)		15.5	gm/dL	12.0 - 17.0
ED BLOOD CELL (RBC)		5.68 <sup>H</sup>	Millions/o	cmm 3.50 - 5.00
ACKED CELL VOLUME	SING, ELECTRICAL IMPEDENCE (PCV) MATED HEMATOLOGY ANALYZER	48	%	40.0 - 54.0
IEAN CORPUSCULAR V		84.5	fL	80.0 - 100.0
IEAN CORPUSCULAR H		27.4	pg	27.0 - 34.0
	IEMOGLOBIN CONC. (MCHC) MATED HEMATOLOGY ANALYZER	32.4	g/dL	32.0 - 36.0
ED CELL DISTRIBUTIO		14.2	%	11.00 - 16.00
ED CELL DISTRIBUTIO		45.2	fL	35.0 - 56.0
AENTZERS INDEX		14.88	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
REEN & KING INDEX by calculated WHITE BLOOD CELLS	WRCS)	21.21	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
OTAL LEUCOCYTE COU	JNT (TLC)	7500	/cmm	4000 - 11000
IUCLEATED RED BLOO	D CELLS (nRBCS)	NIL		0.00 - 20.00
•	D CELLS (nRBCS) %	NIL	%	< 10 %





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Dr. Yugam Chopra

MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. INDERJEET SINGH AGE/ GENDER : 35 YRS/MALE **PATIENT ID** :1310915 **COLLECTED BY** :042502080002 REG. NO./LAB NO. : **REFERRED BY REGISTRATION DATE** :08/Feb/202508:57 AM **BARCODE NO. COLLECTION DATE** :08/Feb/202503:46PM :A1260454 CLIENT CODE. : KOS DIAGNOSTIC SHAHBAD **REPORTING DATE** :08/Feb/202504:13PM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC)** NEUTROPHILS 49<sup>L</sup> % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY 45<sup>H</sup> LYMPHOCYTES % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 1 % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 5 % 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 3675 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 3375 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 75 /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 375 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 302000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) % 0.10 - 0.36 0.38<sup>H</sup> by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 12<sup>H</sup> MEAN PLATELET VOLUME (MPV) fL. 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) 30000 - 90000 130000<sup>H</sup> /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) 43 % 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 16.3% 15.0 - 17.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE

Dr. Vinay Chopra



NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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	MD	Vinay Chopra (Pathology & Microbiology) rman & Consultant Patholo		(Pathology)
NAME	: Mr. INDERJEET	SINGH		
AGE/ GENDER	: 35 YRS/MALE		PATIENT ID	: 1310915
COLLECTED BY	:		REG. NO./LAB NO.	: 042502080002
REFERRED BY	:		<b>REGISTRATION DATE</b>	: 08/Feb/2025 08:57 AM
BARCODE NO.	: A1260452		<b>COLLECTION DATE</b>	:08/Feb/202503:46PM
CLIENT CODE.	: KOS DIAGNOSTIO	C SHAHBAD	<b>REPORTING DATE</b>	: 08/Feb/2025 04:34PM
CLIENT ADDRESS	: 6349/1, NICHOL	SON ROAD, AMBALA CAN	ТТ	
Test Name		Value	Unit	Biological Reference interval
		CLINICAL CHEM	ISTRY/BIOCHEMIST	TRY
		GLUCO	SE FASTING (F)	
GLUCOSE FASTING	G (F): PLASMA Se - peroxidase (god-	84.79	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0

**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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IAME	: Mr. INDERJEET SINGH			
GE/ GENDER	: 35 YRS/MALE	PAT	IENT ID	: 1310915
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SARCODE NO.	: A1260453		LECTION DATE	:08/Feb/202503:46PM
LIENT CODE.	: KOS DIAGNOSTIC SHAHBAD		ORTING DATE	: 08/Feb/2025 06:23PM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
		LIPID PROFIL	E : BASIC	
HOLESTEROL TOT	AI · SFRIM	199.37	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		155.57	ilig/ uL	BORDERLINE HIGH: 200.0 -
				239.0
				HIGH CHOLESTEROL: > OR = 240.0
RIGLYCERIDES: SI		125.32	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSP	HATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 - 199.0
				HIGH: 200.0 - 499.0
				VERY HIGH: > OR = 500.0
IDL CHOLESTEROI by SELECTIVE INHIBITI	L (DIRECT): SERUM	41.31	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0
.,				60.0
	(FRU) (		( 17	HIGH HDL: $> OR = 60.0$
DL CHOLESTEROL by CALCULATED, SPE		133 <sup>H</sup>	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.
•				BORDERLINE HIGH: 130.0 -
				159.0 HIGH: 160.0 - 189.0
				VERY HIGH: > OR = 190.0
NON HDL CHOLEST		158.06 <sup>H</sup>	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPE	CTROPHOTOMETRY			ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 -
				189.0
				HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
LDL CHOLESTER	DL: SERUM	25.06	mg/dL	0.00 - 45.00
by CALCULATED, SPE	CTROPHOTOMETRY			
OTAL LIPIDS: SER by CALCULATED, SPE		524.06	mg/dL	350.00 - 700.00
CHOLESTEROL/HD	L RATIO: SERUM	4.83 <sup>H</sup>	RATIO	LOW RISK: 3.30 - 4.40
by CALCULATED, SPE	UIKUPHUIUMEIRY			AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





	<b>Dr. Vinay Ch</b> MD (Pathology & Chairman & Con			(Pathology)
NAME	: Mr. INDERJEET SINGH			
AGE/ GENDER	: 35 YRS/MALE		PATIENT ID	: 1310915
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		3.22 <sup>H</sup>	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		3.03	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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CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	F	REPORTING DATE	: 08/Feb/2025 07:04PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AME	BALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
			TEST (COMPLETE)	
BILIRUBIN TOTAL	: SERUM PECTROPHOTOMETRY	1.54 <sup>H</sup>	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.24	mg/dL	0.00 - 0.40
-	CT (UNCONJUGATED): SERUM	1.3 <sup>H</sup>	mg/dL	0.10 - 1.00
SGOT/AST: SERUM		44.8	U/L	7.00 - 45.00
SGPT/ALT: SERUM		32.5	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE	ERUM	1.38	RATIO	0.00 - 46.00
ALKALINE PHOSPH		120.02	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTROF	L TRANSFERASE (GGT): SERUM	21.23	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO	SERUM	7.48	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		4.4	gm/dL	3.50 - 5.50
GLOBULIN: SERUM	1	3.08	gm/dL	2.30 - 3.50
A : G RATIO: SERUN	M	1.43	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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CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	<b>REPORTING DATE</b>	: 08/Feb/2025 07:04PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBAL	A CANTT		

Test Name	Value	Unit	<b>Biological Reference interval</b>

## 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 SIGNIFICANO	:Е:

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



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



100 3001 . 2000 CENT					
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Test Name		Value	Unit	Biological Reference inte	erval
	KIDNI	EY FUNCTION	TEST (COMPLETE)		
UREA: SERUM		36.57	mg/dL	10.00 - 50.00	
	NATE DEHYDROGENASE (GLDH)	1.07		0.40 1.40	
CREATININE: SER		1.27	mg/dL	0.40 - 1.40	
	ROGEN (BUN): SERUM	17.09	mg/dL	7.0 - 25.0	
	ROGEN (BUN)/CREATININE	13.46	RATIO	10.0 - 20.0	
RATIO: SERUM by CALCULATED, SPE	ECTROPHOTOMETRY				
UREA/CREATININ	E RATIO: SERUM	28.8	RATIO		
by CALCULATED, SPE URIC ACID: SERUM	ECTROPHOTOMETRY 1	5.46	mg/dL	3.60 - 7.70	
by URICASE - OXIDAS					
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	10.03	mg/dL	8.50 - 10.60	
PHOSPHOROUS: SH		2.86	mg/dL	2.30 - 4.70	
	DATE, SPECTROPHOTOMETRY		U		
ELECTROLYTES					
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	139.6	mmol/L	135.0 - 150.0	
POTASSIUM: SERU	М	3.95	mmol/L	3.50 - 5.00	
by ISE (ION SELECTIV CHLORIDE: SERUM		104.7	mmol/L	90.0 - 110.0	
by ISE (ION SELECTIV	/E ELECTRODE)		IIIII01/ L	JU.U - 11U.U	
ESTIMATED GLON	MERULAR FILTERATION RATE				
(eGFR): SERUM by CALCULATED	IERULAR FILTERATION RATE	75.6			
INTERPRETATION					

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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LIENT CODE.		STIC SHAHBAD		EPORTING DATE	:08	/Feb/2025 06	5:23PM		
LIENT ADDRESS	: 6349/1, NICI	IOLSON ROAD, AMBA	ALA CANTT						
Fest Name			Value	Uni	t	Biologi	cal Refere	ence inter	val
9. Certain drugs (e.g. <b>NCREASED RATIO (&gt;2</b>	tetracycline, glu 0:1) WITH ELEVA	TED CREATININE LEVE	LS:	) (e.a. obstructive	uropathy)				
<ol> <li>Certain drugs (e.g.,</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Perenal azotemia</li> <li>CEREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>SIADH (syndrome of SIADH (syndrome of Pregnancy.</li> <li>Pregnancy.</li> <li>PECREASED RATIO (</li> <li>Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>hould produce an in</li> <li>Cephalosporin thera</li> <li>STAGE</li> </ol>	tetracycline, glu <b>io:1) WITH ELEVA</b> a (BUN rises dispi- superimposed o <b>io:1) WITH DECRI</b> osis. ad starvation. e. creased urea syr- urea rather thar monemias (urea of inappropiate a <b>io:1) WITH INCRE</b> py (accelerates of eleases muscle of who develop rer : sis (acetoacetate creased BUN/cre- rapy (interferes v <b>JLAR FILTERATION</b>	cocorticoids) <b>TED CREATININE LEVE</b> roportionately more t in renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creatine reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measure <b>NATE:</b> <b>DESCRIPTION</b>	LS: han creatinine) ut of extracellu blood). due to tubular to creatinine). e in creatinine rement).	ular fluid). secretion of urea. with certain meth min/1.73m2 )	nodologies,re ASSOCIAT	ED FINDINGS	mal ratio v	vhen dehy	dratic
Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Low protein diet ar     Severe liver diseas     Other causes of de     Repeated dialysis     Inherited hyperam     SIADH (syndrome of     Pregnancy.     DECREASED RATIO (<         Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido hould produce an in     Cephalosporin ther     STIMATED GLOMERL     CKD STAGE     G1	tetracycline, glu <b>io:1) WITH ELEVA</b> a (BUN rises dispi- superimposed o <b>io:1) WITH DECRI</b> osis. ad starvation. e. creased urea syr- urea rather thar monemias (urea of inappropiate a <b>io:1) WITH INCRE</b> py (accelerates of eleases muscle of who develop rer : sis (acetoacetate creased BUN/cre- apy (interferes v JLAR FILTERATION Nor	cocorticoids) <b>TED CREATININE LEVE</b> roportionately more t in renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creatine reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measure <b>NATE:</b> <b>DESCRIPTION</b> mal kidney function	LS: han creatinine) ut of extracellu blood). due to tubular to creatinine). e in creatinine rement).	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 )</u> >90	nodologies,re ASSOCIAT	ED FINDINGS oteinuria	mal ratio v	vhen dehy	dratic
Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Low protein diet ar     Severe liver diseas     Other causes of de     Repeated dialysis     Inherited hyperam     SIADH (syndrome of     Pregnancy.     DECREASED RATIO (<         Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido hould produce an in     Cephalosporin ther     STADE GLOMERL     CKD STAGE	tetracycline, glu tetracycline, glu to:1) WITH ELEVA (BUN rises dispi- superimposed o to:1) WITH DECRI osis. ad starvation. e. creased urea syr- urea rather thar monemias (urea of inappropiate a to:1) WITH INCRE py (accelerates of eleases muscle of who develop rer : sis (acetoacetate creased BUN/cre- apy (interferes v JLAR FILTERATION Nor- Kid	cocorticoids) <b>TED CREATININE LEVE</b> roportionately more t in renal disease. <b>EASED BUN :</b> ASED BUN : in tridiuretic harmone) <b>ASED CREATININE:</b> onversion of creatine reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measure <b>NATE:</b> <b>DESCRIPTION</b> mal kidney function Iney damage with	LS: han creatinine) ut of extracellu blood). due to tubular to creatinine). e in creatinine rement).	ular fluid). secretion of urea. with certain meth min/1.73m2 )	nodologies,re ASSOCIAT No pr Presence	ED FINDINGS oteinuria of Protein ,		vhen dehy	dratic
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Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Low protein diet ar     Severe liver diseas     Other causes of de     Repeated dialysis     Inherited hyperam     SIADH (syndrome of     Pregnancy.     DECREASED RATIO (<         Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido hould produce an in     Cephalosporin ther     STIMATED GLOMERL     CKD STAGE     G1	tetracycline, glu 10:1) WITH ELEVA 1 (BUN rises dispi- superimposed o 10:1) WITH DECRI osis. 10:1) WITH DECRI osis. 10:1) WITH INCRE 10:1) WITH INC	cocorticoids) <b>TED CREATININE LEVE</b> roportionately more t in renal disease. <b>EASED BUN :</b> ASED BUN : in tridiuretic harmone) <b>ASED CREATININE:</b> onversion of creatine reatinine natio). vith creatinine measure vith creatinine measure <b>NATE:</b> DESCRIPTION mal kidney function drey damage with ormal or high GFR	LS: han creatinine) ut of extracellublood). due to tubular to creatinine). e in creatinine rement). GFR ( mL/	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 )</u> >90 >90	nodologies,re ASSOCIAT No pr Presence	ED FINDINGS oteinuria of Protein ,		vhen dehy	dratic
Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     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 (<         Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido     hould produce an in     Cephalosporin ther     STIMATED GLOMERL     G1     G2	tetracycline, glu tetracycline, glu to:1) WITH ELEVA (BUN rises dispi- superimposed o to:1) WITH DECRI osis. ad starvation. e. creased urea syr- urea rather thar monemias (urea of inappropiate a for appropriate a to:1) WITH INCRE py (accelerates co- eleases muscle co- who develop rer sis (acetoacetate creased BUN/cre- apy (interferes v JLAR FILTERATION Nor- Nor- Nor- Mid Mode	cocorticoids) TED CREATININE LEVE roportionately more t in renal disease. EASED BUN : ASED BUN : ASED CREATININE: onversion of creatine reatinine (). hal failure. ASED CREATININE: onversion of creatine reatinine ratio). //ith creatinine measure //ith creatinine measure MATE: DESCRIPTION mal kidney function diney damage with ormal or high GFR d decrease in GFR	LS: han creatinine) ut of extracellublood). due to tubular to creatinine). e in creatinine rement). GFR ( mL/ 6 3	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 ) &gt;90 &gt;90 0 -89</u>	nodologies,re ASSOCIAT No pr Presence	ED FINDINGS oteinuria of Protein ,		vhen dehy	dratic





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NAME	: Mr. INDERJEET SINGH		
AGE/ GENDER	: 35 YRS/MALE	PATIENT ID	: 1310915
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 042502080002
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 08/Feb/2025 08:57 AM
BARCODE NO.	: A1260453	COLLECTION DATE	:08/Feb/202503:46PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	<b>REPORTING DATE</b>	: 08/Feb/2025 06:23PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT	
Test Name		Value Unit	Biological Reference interva

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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