



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)	Dr. Yugam MD (CEO & Consultant F	Pathology)
IAME	: Mr. DINESH VERMA			
AGE/ GENDER	: 61 YRS/MALE	PA	TIENT ID	: 1668173
COLLECTED BY	: SURJESH	RE	G. NO./LAB NO.	:012411110063
REFERRED BY	:	RE	GISTRATION DATE	: 11/Nov/2024 01:20 PM
BARCODE NO.	: 01520591		LLECTION DATE	: 11/Nov/2024 01:30PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		PORTING DATE	: 11/Nov/2024 01:51PM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	ALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
RED BLOOD CELLS			LNESS PANEL: G D COUNT (CBC)	
HAEMOGLOBIN (H		12.5	gm/dL	12.0 - 17.0
by CALORIMETRIC		4.00	Ű	
RED BLOOD CELL (KBC) COUN I OCUSING, ELECTRICAL IMPEDENCE	4.26	Millions/c	2mm 3.50 - 5.00
PACKED CELL VOLU	UME (PCV) UTOMATED HEMATOLOGY ANALYZER	39 ^L	%	40.0 - 54.0
MEAN CORPUSCUL	AR VOLUME (MCV)	91.5	fL	80.0 - 100.0
MEAN CORPUSCUL	UTOMATED HEMATOLOGY ANALYZER AR HAEMOGLOBIN (MCH)	29.4	pg	27.0 - 34.0
	UTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC)	32.2 ^L	g/dL	32.0 - 36.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER		0	
	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	14.9	%	11.00 - 16.00
	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	51	fL	35.0 - 56.0
MENTZERS INDEX	OTOMATED HEMATOLOGT ANALTZER	21.48	RATIO	BETA THALASSEMIA TRAIT: <
by CALCULATED				13.0 IDON DEFICIENCY ANEMIA.
				IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING IND	DEX	32.07	RATIO	BETA THALASSEMIA TRAIT:<=
by CALCULATED				65.0 IRON DEFICIENCY ANEMIA: >
				65.0
NHITE BLOOD CE				
FOTAL LEUCOCYTE by FLOW CYTOMETRY	E COUNT (TLC) (by sf cube & microscopy	6710	/cmm	4000 - 11000
NUCLEATED RED B	BLOOD CELLS (nRBCS)	NIL		0.00 - 20.00
by AUTOMATED 6 PAP	RT HEMATOLOGY ANALYZER	NIL	%	< 10 %
NUCLEATED RED B	UTOMATED HEMATOLOGY ANALYZER	IVIL	70	10/0





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

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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT







Dr. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. DINESH VERMA AGE/ GENDER : 61 YRS/MALE **PATIENT ID** :1668173 **COLLECTED BY** : SURJESH REG. NO./LAB NO. :012411110063 **REFERRED BY REGISTRATION DATE** : 11/Nov/2024 01:20 PM : **BARCODE NO.** :01520591 **COLLECTION DATE** :11/Nov/2024 01:30PM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :11/Nov/2024 01:51PM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC) NEUTROPHILS** 74^H % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 13^L % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 6 % 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 4965 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 872 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 403 /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 470 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 71000^L /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.1^L % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) fL 14^H 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 30000 - 90000 PLATELET LARGE CELL COUNT (P-LCC) 39000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE % PLATELET LARGE CELL RATIO (P-LCR) 55.5^H 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 15.0 - 17.0 % 17.1^H by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE

ADVICE



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

KINDLY CORRELATE CLINICALLY

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA C	ANTT	
Test Name	Valu	e Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED.



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BARCODE NO.	: 01520591			: 11/Nov/2024 01:30PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB			: 11/Nov/2024 02:28PM
			NIING DATE	. 11/ NOV/ 2024 02.201 M
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANT I		
Test Name		Value	Unit	Biological Reference interva
WHOLE BLOOD	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY)	7.9 ^H	%	4.0 - 6.4
	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	180.03 ^H	mg/dL	60.00 - 140.00
	AS PER AMERICAN	DIABETES ASSOCIATION	(404).	
	REFERENCE GROUP		LATED HEMOGLOGIB (H	BAIC) in %
Non dia	abetic Adults >= 18 years	<5.7		
	Risk (Prediabetes)	5.7 - 6.4		
D	agnosing Diabetes	>= 6.5		
			Age > 19 Years	
		Goals of The	1.5	< 7.0
T				
Therapeut	ic goals for glycemic control	Actions Sugg		>8.0
Therapeut	ic goals for glycemic control	Actions Sugg Goal of the	Age < 19 Years	<7.5

KOS Diagnostic Lab

(A Unit of KOS Healthcare)

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 appropiate.

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 faisely 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 PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



NAME M. DINESH VERMA AGE/ GENDER :61 YRS/MALE PATIENT ID :1668173 COLLECTED BY :SURJESH REG. NO./LAB NO. :012411110063 REFERRED BY : REGISTRATION DATE :11/Nov/2024 01:20 PM BARCODE NO. :01520591 COLLECTION DATE :11/Nov/2024 01:30PM CLIENT CODE :KOS DIAGNOSTIC LAB REPORTING DATE :11/Nov/2024 01:59PM CLIENT ADDRESS :6349/1, NICHOLSON ROAD, AMBALA CANTT Biological Reference interval CERTHROCYTE SEDIMENTATION RATE (ESR) BERYTHROCYTE SEDIMENTATION RATE (ESR) 28 ^d mm/1st hr 0 - 20 by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY INTERRETATION INTERRETATION 1.581 ka non beselfs in fidammation. For this reason, the ESR is typically used in conjunction with other test such as Creative protein Structure of the month of the normal sedimentation of red blood cells, such as a high red blood cell count (polycythamita), significantly high withe blood cell count (leuccytosis), and some protein abnormalities. Some changes in red cell shape (such as skice cells in sickle cell anaemia) also lower the ESR. ONOTION WITHOW CSN A resk can be seen with conditions that inhibit the normal sedimentation		Dr. Vinay Cho MD (Pathology & Chairman & Cons	Microbiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
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Cest Name Value Unit Biological Reference interval ERYTHROCYTE SEDIMENTATION RATE (ESR) BERYTHROCYTE SEDIMENTATION RATE (ESR) by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY Description of the second of t	LIENT CODE.	: KOS DIAGNOSTIC LAB	I	REPORTING DATE	: 11/Nov/2024 01:59PM
ERYTHROCYTE SEDIMENTATION RATE (ESR) ERYTHROCYTE SEDIMENTATION RATE (ESR) by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY NTERPRETATION: • SR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto mmune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it. • An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such so C-reactive protein • This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as ystemic lupus erythematosus ONDITION WITH LOW ESR • Now ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count polycythaemia), significantly high white blood cell count (leucocytosis) , and some protein abnormalities. Some changes in red cell shape (such so fick cells in sickle cell anaemia) also lower the ESR. OTE: • ESR and C - reactive protein (C-RP) are both markers of inflammation. • CAP is not affected by as many other factors as is ESR, making it a better marker of inflammation. • CAP is not affected by as many other factors as is ESR, making it a better marker of inflammation. • CAP is not affected by as many other factory as is ESR, making it a better marker of inflammati	LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
EXPTTHROCYTE SEDIMENTATION RATE (ESR) 28 ^H mm/1st hr 0 - 20 by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY NTERPRETATION: 1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto mmune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it. 2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein. 3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus CONDITION WITH LOW ESR Alow ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count polycythaemia), significantly high white blood cell count (leucocytosis) , and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR. VOTE: 1. ESR and C - reactive protein (C-RP) are both markers of inflammation. 2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves. 3. The ESR is levated, it is typically a result of two types of proteins, globulins or fibrinogen. 4. WOTE: 5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations. 5. Women tend to h	Fest Name		Value	Unit	Biological Reference interval
by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY NTERPRETATION:ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto mmune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it. 2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such s C-reactive protein 3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as ystemic lupus erythematosus CONDITION WITH LOW ESR A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count polycythaemia), significantly high white blood cell count (leucocytosis), and some protein abnormalities. Some changes in red cell shape (suc s scicle cells in sickle cell anaemia) also lower the ESR. UOTE:ESR and C - reactive protein (C-RP) are both markers of inflammationESR and C - reactive protein (C-RP) are both markers of inflammationESR and C - reactive protein (C-RP) are both markers of inflammationESR and C - reactive protein (C-RP) are both markers of proteins, globulins or fibrinogenESR and C - reactive protein (C-RP) are both markers of proteins, globulins or fibrinogenESR and C - reactive protein (C-RP) are both markers of proteins, globulins or fibrinogenESR and C - reactive protein (C-RP) are both markers of proteins, globulins or fibrinogenESR and C - reactive protein (C-RP) are both markers of proteins, globulins or fibrinogenESR and C - reactive protein (C-RP) are both markers of proteins, globulins or fibrinogenESR and C - reactive protein (C-RP) are both markers of proteins, globulins or fibrinogenESR and C - reactive protein (C-RP) are both markers of proteins, globulins or fibrinogenESR and C - reactive protein (C-RP)		ERYTHR	OCYTE SEDIM	ENTATION RATE (ESR)
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen. 5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations. 6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while	as C-reactive protein 3. This test may also systemic lupus eryth CONDITION WITH LO A low ESR can be see (polycythaemia), sig as sickle cells in sick NOTE:	be used to monitor disease activit ematosus W ESR en with conditions that inhibit the nificantly high white blood cell cou le cell anaemia) also lower the ES re protein (C-RP) are both markers	y and response to normal sedimenta unt (leucocytosis) R. of inflammation. RP, either at the s	o therapy in both of the a ation of red blood cells, si , and some protein abno	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
	2. Generally, ESR doe	billion of the second state of the second stat			





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MBBS, MD (PATHOLOGY)







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CLIENT ADDRESS	: 6349/1, NICHO	DLSON ROAD,	AMBALA CANTT		
Test Name			Value	Unit	Biological Reference interval
		CLINI	CAL CHEMIS	TRY/BIOCHEMIST	'RY
			GLUCOSE	FASTING (F)	
GLUCOSE FASTING		D-POD)	137.64 ^H	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.

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
			FILE : BASIC	
CUOLESTEDOL TO	TAL. CEDIM			OPTIMAL: < 200.0
CHOLESTEROL TO by CHOLESTEROL O		132.78	mg/dL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSE	ERUM PHATE OXIDASE (ENZYMATIC)	178.63 ^H	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
HDL CHOLESTERO by SELECTIVE INHIBIT	L (DIRECT): SERUM Ton	38.58	mg/dL	VERY HIGH: > OR = 500.0 LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERO		58.47	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		94.2	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(35.73	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEF	RUM	444.19	mg/dL	350.00 - 700.00
CHOLESTEROL/HI by CALCULATED, SPE	DL RATIO: SERUM	3.44	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 PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





		Chopra & Microbiology) onsultant Patholog		(Pathology)
NAME	: Mr. DINESH VERMA			
AGE/ GENDER	: 61 YRS/MALE		PATIENT ID	: 1668173
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012411110063
REFERRED BY	:		REGISTRATION DATE	: 11/Nov/2024 01:20 PM
BARCODE NO.	: 01520591		COLLECTION DATE	: 11/Nov/2024 01:30PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 11/Nov/2024 04:49PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANT	Т	
Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		1.52	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		4.63	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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	Dr. Vinay Chop MD (Pathology & Mid Chairman & Consulta	crobiology)	Dr. Yugam MD (CEO & Consultant	(Pathology)
NAME	: Mr. DINESH VERMA			
AGE/ GENDER	: 61 YRS/MALE]	PATIENT ID	: 1668173
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMI	BALA CANTT		
Test Name		Value	Unit	Biological Reference interval
BILIRUBIN DIRECT	: SERUM pectrophotometry [(CONJUGATED): SERUM spectrophotometry	1.27^H 0.3	TEST (COMPLETE) mg/dL mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20 0.00 - 0.40
BILIRUBIN INDIRE by CALCULATED, SPE	CCT (UNCONJUGATED): SERUM	0.97	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	[/RIDOXAL PHOSPHATE	27.4	U/L	7.00 - 45.00
SGPT/ALT: SERUM		18.3	U/L	0.00 - 49.00
AST/ALT RATIO: S	ERUM	1.5	RATIO	0.00 - 46.00
ALKALINE PHOSPI		114.52	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTROF	L TRANSFERASE (GGT): SERUM	50.39	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO	SERUM	6.99	gm/dL	6.20 - 8.00
ALBUMIN: SERUM	REEN	3.46 ^L	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	1	3.53 ^H	gm/dL	2.30 - 3.50
A : G RATIO: SERUN	M	0.98 ^L	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:

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



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Tost Namo		Valuo Unit	Biological Poforanco interval

Test Name Value Unit	Biological Reference interval
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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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	MD (Pathology & N	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		(Pathology) Pathologist
NAME	: Mr. DINESH VERMA			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AN	DAD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	KIDNE		N TEST (COMPLETE)	
UREA: SERUM		51.11 ^H	mg/dL	10.00 - 50.00
•	MATE DEHYDROGENASE (GLDH)	U		0.40 1.40
CREATININE: SERU by ENZYMATIC, SPEC		1.77 ^H	mg/dL	0.40 - 1.40
	ROGEN (BUN): SERUM	23.88	mg/dL	7.0 - 25.0
by CALCULATED, SPE BLOOD URFA NITE	CCTROPHOTOMETRY ROGEN (BUN)/CREATININE	13.49	RATIO	10.0 - 20.0
RATIO: SERUM		10.10	101110	10.0 20.0
by CALCULATED, SPE UREA/CREATININ		28.88	RATIO	
by CALCULATED, SPE		20.00	KATIO	
URIC ACID: SERUM by URICASE - OXIDAS		9.79 ^H	mg/dL	3.60 - 7.70
CALCIUM: SERUM	SE PEROXIDASE	9.31	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE			-	
PHOSPHOROUS: SE	ERUM DATE, SPECTROPHOTOMETRY	3.28	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM		146	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV		4.05		
POTASSIUM: SERU by ISE (ION SELECTIV		4.65	mmol/L	3.50 - 5.00
CHLORIDE: SERUM	1	109.5	mmol/L	90.0 - 110.0
by ISE (ION SELECTIV	'E ELECTRODE) IERULAR FILTERATION RATE			

ESTIMATED GLOMERULAR FILTERATION RATE

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.

4. High protein intake.

5. Impaired renal function plus

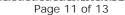
6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet,



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9001:2008 CERT							
		Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant	obiology)	Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist			
IAME	: Mr. DINES	H VERMA					
AGE/ GENDER	: 61 YRS/MA	LE	PATIENT ID	: 166817	73		
COLLECTED BY	: SURJESH		REG. NO./LAB N	0. :01241	11110063		
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BARCODE NO.	:01520591		COLLECTION DA		v/2024 01:30		
LIENT CODE.	: KOS DIAGN		REPORTING DA	TE : 11/No [*]	v/2024 05:44	4PM	
CLIENT ADDRESS	: 6349/1, NI	CHOLSON ROAD, AMBA	LA CANTT				
Fest Name			Value I	J nit	Biological	Reference inte	rval
 Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia 	ass (subnorma tetracycline, g 0:1) WITH ELEV (BUN rises dis superimposed	ATED CREATININE LEVEL proportionately more th on renal disease.		ive uropathy).			
 Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia PecREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome c Pregnancy. PECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido cephalosporin ther 	ass (subnorma tetracycline, g 0:1) WITH ELEV (BUN rises dis superimposed 0:1) WITH DEC osis. Id starvation. 2: creased urea s urea rather th monemias (ure f inappropiate 0:1) WITH INC oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/o apy (interferes	I creatinine production) lucocorticoids) /ATED CREATININE LEVEL proportionately more th on renal disease. REASED BUN : ynthesis. an creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d REASED CREATININE: s conversion of creatine e creatinine). enal failure. the causes false increase treatinine ratio). with creatinine measure ON RATE:	S: han creatinine) (e.g. obstruct ut of extracellular fluid). blood). lue to tubular secretion of ur to creatinine).	ea. ethodologies,resulti		I ratio when dehy	dratior
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 c Pregnancy. DECREASED RATIO (<1 Nuscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERL OKD STAGE	ass (subnorma tetracycline, g 0:1) WITH ELEV (BUN rises dis superimposed 0:1) WITH DEC osis. Id starvation. 2: creased urea s urea rather th monemias (uro f inappropiate 0:1) WITH INC oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/o apy (interferes LAR FILTERATI	I creatinine production) Iucocorticoids) /ATED CREATININE LEVEL proportionately more th on renal disease. REASED BUN : ynthesis. an creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d REASED CREATININE: s conversion of creatine e creatinine). enal failure. ate causes false increase creatinine ratio). with creatinine measure DISCRIPTION	S: han creatinine) (e.g. obstruct ut of extracellular fluid). blood). lue to tubular secretion of ur to creatinine). in creatinine with certain m ement). GFR (mL/min/1.73m2)	ea. ethodologies,resulti ASSOCIATED F	INDINGS	l ratio when dehy	dratio
 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 c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther 	ass (subnorma tetracycline, g 0:1) WITH ELEV (BUN rises dis superimposed 0:1) WITH DEC osis. Id starvation. 2: creased urea s urea rather th monemias (uro f inappropiate 0:1) WITH INC oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/o apy (interferes LAR FILTERATI	I creatinine production) Iucocorticoids) /ATED CREATININE LEVEL proportionately more th on renal disease. REASED BUN : ynthesis. an creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d REASED CREATININE: s conversion of creatine e creatinine). enal failure. Atte causes false increase areatinine ratio). with creatinine measure DESCRIPTION prmal kidney function	S: han creatinine) (e.g. obstruct ut of extracellular fluid). blood). lue to tubular secretion of ur to creatinine).	ea. ethodologies,resulti	TINDINGS	I ratio when dehy	dratio
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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 c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in CEPhalosporin ther STIMATED GLOMERL CKD STAGE G1 G2	ass (subnorma tetracycline, g 0:1) WITH ELEV (BUN rises dis superimposed 0:1) WITH DEC osis. Id starvation. 2. creased urea s urea rather th monemias (uro f inappropiate 0:1) WITH INC oy (accelerate eleases muscle who develop r sis (acetoaceta creased BUN/c apy (interferes LAR FILTERATI	I creatinine production) Iucocorticoids) /ATED CREATININE LEVEL proportionately more th on renal disease. REASED BUN : ynthesis. an creatinine diffuses out a si virtually absent in th antidiuretic harmone) of REASED CREATININE: s conversion of creatine e creatinine). enal failure. REASED CREATININE: s conversion of creatine e creatinine ratio). with creatinine measure DESCRIPTION ormal kidney function Kidney damage with normal or high GFR	S: han creatinine) (e.g. obstruct ut of extracellular fluid). blood). lue to tubular secretion of ur to creatinine). in creatinine with certain m ement). GFR (mL/min/1.73m2) >90 >90	ea. ethodologies,resulti ASSOCIATED F No protein Presence of F	INDINGS nuria Protein ,	I ratio when dehy	dratio





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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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