

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



<b>Dr. Vinay Cl</b> MD (Pathology Chairman & Co		Dr. Yugam MD (1 CEO & Consultant F	Pathology)
NAME : Mr. YASHPAL PARMAR			
AGE/ GENDER : 71 YRS/MALE	P	ATIENT ID	: 1592828
COLLECTED BY : SURJESH	R	EG. NO./LAB NO.	: 012408270025
REFERRED BY :		EGISTRATION DATE	: 27/Aug/2024 10:15 AM
<b>BARCODE NO.</b> : 01515795	_	OLLECTION DATE	: 27/Aug/2024 10:33AM
CLIENT CODE.: KOS DIAGNOSTIC LABCLIENT ADDRESS: 6349/1, NICHOLSON ROAD		EPORTING DATE	: 27/Aug/2024 11:27AM
Test Name	Value	Unit	Biological Reference interval
S	WASTHYA WFI	LNESS PANEL: G	
	COMPLETE BLOC		
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB)	12.9	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (RBC) COUNT	4.41	Millions/cn	nm 3.50 - 5.00
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCI PACKED CELL VOLUME (PCV)	e 40.5	%	40.0 - 54.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ	ZER		
MEAN CORPUSCULAR VOLUME (MCV) by calculated by automated hematology analyz	91.8 ZER	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH)	29.3	pg	27.0 - 34.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHO	C) 31.9 <sup>L</sup>	g/dL	32.0 - 36.0
by calculated by automated hematology analy RED CELL DISTRIBUTION WIDTH (RDW-CV)	12.9	%	11.00 - 16.00
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ RED CELL DISTRIBUTION WIDTH (RDW-SD)	ZER 44.1	fL	35.0 - 56.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ	ZER		
MENTZERS INDEX by CALCULATED	20.82	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX	26.9	RATIO	BETA THALASSEMIA TRAIT:<= 65.0
			IRON DEFICIENCY ANEMIA: > 65.0
<u>WHITE BLOOD CELLS (WBCS)</u> TOTAL LEUCOCYTE COUNT (TLC)	71630 <sup>H</sup>	/cmm	4000 - 11000
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY		/ (1111	
NUCLEATED RED BLOOD CELLS (nRBCS) by AUTOMATED 6 PART HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) %	NIL	%	< 10 %
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZ DIFFERENTIAL LEUCOCYTE COUNT (DLC)	ZER		
NEUTROPHILS	13 <sup>L</sup>	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			



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

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

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Dr. Yugam Chopra Dr. Vinay Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. YASHPAL PARMAR **AGE/ GENDER** : 71 YRS/MALE **PATIENT ID** :1592828 **COLLECTED BY** : SURJESH :012408270025 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 27/Aug/2024 10:15 AM **BARCODE NO.** :01515795 **COLLECTION DATE** : 27/Aug/2024 10:33AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 27/Aug/2024 11:27AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** LYMPHOCYTES 86<sup>H</sup> % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 0<sup>L</sup> % 1-6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 1<sup>L</sup> % 2 - 12 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LEUKOCYTES (WBC) COUNT **ABSOLUTE NEUTROPHIL COUNT** 2000 - 7500 /cmm 9312<sup>H</sup> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 61602<sup>H</sup> /cmm 800 - 4900 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE EOSINOPHIL COUNT** 40 - 440 0L /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 716 80 - 880 /cmm 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 /cmm 0.0 - 999.0 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) /cmm 150000 - 450000 116000<sup>L</sup> by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.13 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 6.50 - 12.0 12<sup>H</sup> fl by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) 30000 - 90000 44000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) 41.1 % 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 16.2 % 15.0 - 17.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED.



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







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Test Name	Value	Unit	Biological Reference interval





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CLIENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTT         Test Name       Value       Unit       Biological Reference         GLYCOSYLATED HAEMOGLOBIN (HBA1c):         GLYCOSYLATED HAEMOGLOBIN (HbA1c):       5.2       %       4.0 - 6.4         WHOLE BLOOD       by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)       Biological Reference         ESTIMATED AVERAGE PLASMA GLUCOSE       102.54       mg/dL       60.00 - 140.00         by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)       Biological Reference       60.00 - 140.00         INTERPRETATION:       SPER AMERICAN DIABETES ASSOCIATION (ADA):       60.00 - 140.00         REFERENCE GROUP       GLYCOSYLATED HEMOGLOGIB (HBAIC) in %       60.00 - 140.00         Non diabetic Adults >= 18 years       <5.7	
Test Name       Value       Unit       Biological Reference         GLYCOSYLATED HAEMOGLOBIN (HBA1C)         GLYCOSYLATED HAEMOGLOBIN (HBA1c):         S.2       %       4.0 - 6.4         WHOLE BLOOD         by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)         ESTIMATED AVERAGE PLASMA GLUCOSE       102.54       mg/dL       60.00 - 140.00         by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)         INTERPRETATION:         STIMATED AVERAGE PLASMA GLUCOSE       102.54       mg/dL       60.00 - 140.00         by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)         INTERPRETATION:         INTERPRETATION:         STIMATED AVERAGE PLASMA GLUCOSE         INTERPRETATION:         INTERPRETATION:         INTERPRETATION:         STIMATED HEMOGLOGIB (HBAIC) in %         NON diabetic Adults >= 18 years         <5.7	
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GLYCOSYLATED HAEMOGLOBIN (HbA1c):       5.2       %       4.0 - 6.4         WHOLE BLOOD       by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)       mg/dL       60.00 - 140.00         ESTIMATED AVERAGE PLASMA GLUCOSE       102.54       mg/dL       60.00 - 140.00         by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)       Interpretation:       60.00 - 140.00         INTERPRETATION:       See AMERICAN DIABETES ASSOCIATION (ADA):       See AMERICAN DIABETES ASSOCIATION (ADA):         REFERENCE GROUP       GLYCOSYLATED HEMOGLOGIB (HBAIC) in %       See AMERICAN DIABETES ASSOCIATION (ADA):         Non diabetic Adults >= 18 years       <5.7	ence interval
WHOLE BLOOD by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY) ESTIMATED AVERAGE PLASMA GLUCOSE 102.54 mg/dL 60.00 - 140.00 by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY) INTERPRETATION: NINTERPRETATION: REFERENCE GROUP GLYCOSYLATED HEMOGLOGIB (HBAIC) in % Non diabetic Adults >= 18 years <5.7 At Risk (Prediabetes) 5.7 - 6.4	
ESTIMATED AVERAGE PLASMA GLUCOSE 102.54 mg/dL 60.00 - 140.00 by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY) INTERPRETATION:	
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At Risk (Prediabetes) 5.7 – 6.4	
Diagnosing Diabetes >= 6.5	
Age > 19 Years	
Goals of Therapy:         < 7.0           Therapeutic goals for glycemic control         Actions Suggested:         >8.0	

#### 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

Goal of therapy:

Age < 19 Years

<7.5

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 HhA1c (-9.0.-9.5 %) is strongly associated with risk of development and ranid progression of microvascular and perve complications

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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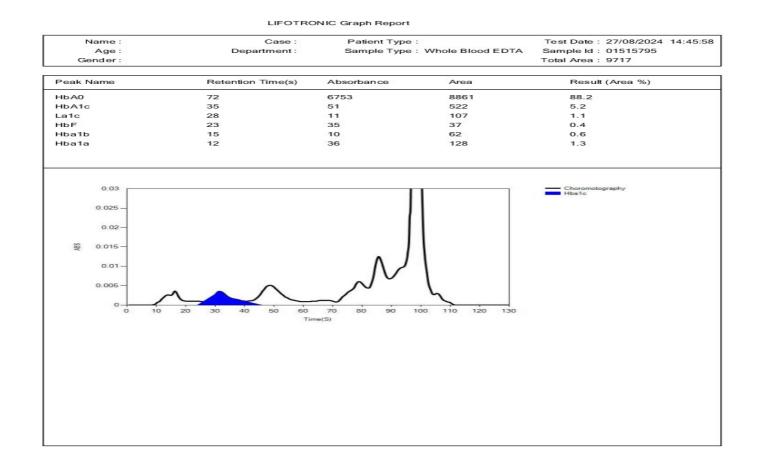
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	<b>Dr. Vinay Chopra</b> MD (Pathology & Micro Chairman & Consultan	obiology) MI	m Chopra D (Pathology) ht Pathologist
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Test Name		Value Unit	<b>Biological Reference interval</b>







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est Name		Value	Unit	Biological Reference interval
	ERYTH	HROCYTE SEDI	MENTATION RATE (ES	R)
	MENTATION RATE (ESR)	20	mm/1st h	
NOTE: 1. ESR and C - reactiv 2. Generally, ESR doo 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dex	le cell anaemia) also lower the E re protein (C-RP) are both marker es not change as rapidly as does ( I by as many other factors as is ES red, it is typically a result of two i ave a higher ESR, and menstruatio tran, methyldopa, oral contracep nd quinine may decrease it	rs of inflammation CRP, either at the <b>SR, making it a be</b> types of proteins on and pregnancy	e start of inflammation or as <b>tter marker of inflammatior</b> , globulins or fibrinogen. ( can cause temporary eleva	h.
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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)







Dr. Vinay Chopra

MD (Pathology & Microbiology)

Chairman & Consultant Pathologist



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

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## PERIPHERAL BLOOD SMEAR

## TEST NAME:

## PERIPHERAL BLOOD FILM/SMEAR (PBF)

## RED BLOOD CELLS (RBC'S):

RBCs mostly appear normocytic & normochromic.No polychromatic cells or normoblastic activity evident.

# WHITE BLOOD CELLS (WBC'S):

Smear show leucocytosis with lymphocytosis.

### **PLATELETS:**

Platelets appear slightly reduced on smear.

# **HEMOPARASITES**:

NOT SEEN.

## **IMPRESSION:**

Normocytic normochromic picture & Chronic lymphocytic leukemia.





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Test Name		Value	Unit	Biological Reference interval
	CLI	NICAL CHEMIST	RY/BIOCHEMISTR	Y
			ASTING (F)	
		GLUCUJLI		

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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.
 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
		LIPID PROFIL	E : BASIC	
CHOLESTEROL TOTAL by CHOLESTEROL OXI		154.74	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.
TRIGLYCERIDES: SERU by GLYCEROL PHOSPH	JM iate oxidase (enzymatic)	108.4	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (E by SELECTIVE INHIBITIC		55.62	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SI by CALCULATED, SPEC		77.44	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 CHOLESTER by CALCULATED, SPEC		99.12	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 CHOLESTEROL: S		21.68	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUN by CALCULATED, SPEC	1	417.88	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL R by CALCULATED, SPEC	ATIO: SERUM	2.78	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERU by calculated, spec		1.39	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





	Dr. Vinay Cl MD (Pathology Chairman & Co		Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. YASHPAL PARMAR			
AGE/ GENDER	: 71 YRS/MALE	PATI	ENT ID	: 1592828
COLLECTED BY	: SURJESH	REG.	NO./LAB NO.	: 012408270025
REFERRED BY	:	REGI	STRATION DATE	: 27/Aug/2024 10:15 AM
BARCODE NO.	:01515795	COLL	ECTION DATE	: 27/Aug/2024 10:33AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 27/Aug/2024 11:55AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD		1.95 <sup>L</sup>	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 Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist

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

NAME	: Mr. YASHPAL PARMAR		
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Т	
			/
Test Name	Value	Unit	<b>Biological Reference interval</b>

LIV	ER FUNCTION T	EST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.86	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.25	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.61	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	17.81	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	14.82	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.2	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	95.45	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by szasz, spectrophtometry	14.4	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	5.63 <sup>L</sup>	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by bromocresol green	4.01	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by calculated, spectrophotometry	1.62 <sup>L</sup>	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.48 <sup>H</sup>	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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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)







	<b>Dr. Vinay Chop</b> MD (Pathology & Mic Chairman & Consult	crobiology) MI	m Chopra D (Pathology) nt Pathologist
NAME	: Mr. YASHPAL PARMAR		
AGE/ GENDER	: 71 YRS/MALE	PATIENT ID	: 1592828
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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).

GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6



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Dr. Yugam Chopra

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NAME	: Mr. YASHPAL PARMAR			
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Test Name		Value	Unit	Biological Reference interval
	КІ	ONEY FUNCTION	TEST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAM	ATE DEHYDROGENASE (GLDH)	27.07	mg/dL	10.00 - 50.00
CREATININE: SERUN	1	0.93	mg/dL	0.40 - 1.40
BLOOD UREA NITRO	GEN (BUN): SERUM	12.65	mg/dL	7.0 - 25.0
	GEN (BUN)/CREATININE	13.6	RATIO	10.0 - 20.0
by CALCULATED, SPE	CTROPHOTOMETRY			
UREA/CREATININE R by CALCULATED, SPE		29.11	RATIO	
URIC ACID: SERUM		5.06	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE		8.96	mg/dL	8.50 - 10.60
PHOSPHOROUS: SER by PHOSPHOMOLYBD		3.02	mg/dL	2.30 - 4.70
<b>ELECTROLYTES</b>				
SODIUM: SERUM by ISE (ION SELECTIV	E ELECTRODE)	141.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUM		4.72	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIV	E ELECTRODE)	106.13	mmol/L	90.0 - 110.0
	RULAR FILTERATION RATE			
ESTIMATED GLOMER (eGFR): SERUM by CALCULATED	RULAR FILTERATION RATE	87.8		

Dr. Vinay Chopra

## 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.



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





NAME : Mr. YASHPAL PARMAR AGE/ GENDER : 7.1 YRS/MALE PATIENT ID : 1592828 COLLECTED BY : SURJESH REG. NO./LAB NO. : 012408270025 REFERED BY : REGISTRATION DATE : 27/Aug/2024 10:5 AM BARCODE NO. : 01515795 COLLECTION DATE : 27/Aug/2024 10:5 AM BARCODE NO. : 01515795 COLLECTION DATE : 27/Aug/2024 10:5 AM CLIENT CODE : KOS DIAGNOSTIC LAB REPORTING DATE : 27/Aug/2024 11:55 AM CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit Biological Reference interval 3. Glaemorrhage. 4. High protein intake. 5. Impaired renal function plus 6. Excess protein intake. 5. Impaired renal function plus 6. Excess protein intake. 5. Impaired renal function plus 6. Excess protein intake. 7. Urine reabsorption (e.g., ureter colosionny) 8. Reduced muscle mass (subnormal creatinine production) 9. Gertain drugs (e.g. tetracycline, glucocorticods) WRCRASDE RATIO (-20:0) WITH IEVENED IN EXCESS 1. Postnenia superfinguoed on renal disease. 1. Postnenia superfinguoed on renal disease. 2. Orportein diet and starvation. 3. Swere liver disease. 2. Own protein diet and starvation. 3. Swere liver disease. 3. Orportein diet and starvation. 4. Other causes of decreased urea synthesis. 5. Repeated diskys (urear atrihum than creatinine diffuses out of extracellular fluid). 6. Inherited hyperammonemias (ureal is virtually absent in blood). 7. JOHC (synthem ef inappropiate antiline diffuses out of extracellular fluid). 6. Inherited hyperammonemias (ureal si virtually absent in blood). 7. JOHC (Synthem ef inappropiate antiline diffuses out of extracellular fluid). 6. Inherited hyperammonemias (ureal si virtually absent in blood). 7. JOHC (Synthem ef inappropiate antiline diffuses out of extracellular fluid). 6. Inherited hyperammonemias (ureal si virtually absent in blood). 7. JOHC (Synthem ef inappropiate antiline diffuses out of extracellular fluid). 6. Inherited hyperammonemias (ureal si virtually absent in blood). 7. JOHC (Synthem ef inappropiate antiline reference in creatinine with certain		MD (Patholog	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologist		Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist	
CULLECTED BY       SURJESH       REG. NO./LAB NO.       SURJESA         REFERRED BY       :       .       REGISTRATION DATE       :       27/Aug/2024 10:15 AM         BARCODE NO.       :01515795       COLLECTION DATE       :       27/Aug/2024 10:33AM         CLIENT CODE       :KOS DIAGNOSTIC LAB       REPORTING DATE       :       27/Aug/2024 10:33AM         CLIENT ADRESS       :6349/1, NICHOLSON ROAD, AMBALA CANTT       .       .       .       .         3. GI haemorrhage.       <	NAME	: Mr. YASHPAL PARMAR				
REFERED BY       ::::::::::::::::::::::::::::::::::::	AGE/ GENDER	: 71 YRS/MALE	P	ATIENT ID	: 1592828	
REFERED BY:       ::::::::::::::::::::::::::::::::::::	COLLECTED BY	: SURJESH	R	EG. NO./LAB NO.	: 012408270025	5
BARCODE NO. : : 01515795						
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CLIENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTT         Test Name       Value       Unit       Biological Reference interval         3. Gi haemorrhage.       4. High protein intake.       5. Impaired renal function plus         6. Excess protein intake or production or tissue breakdown (e.g. infection, Gl bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).       7. Urine reabsorption (e.g. ureter colostomy)         8. Reduced muscle mass (subnormal creatinine production)       9. Certain drugs (e.g. tetracycline, glucocorticoids)         NICREASED RATIO (-20:1) WITH ELVATED CREATININE LEVELS:       1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         9. Perenal azotemia (SURT Sirvilauly absent in blood).       2. Perenal azotemia (SURT Sirvilauly absent in blood).         9. Other disease.       4. Other causes of decreased urea synthesis.         9. Other disease.       4. Other causes of decreased urea synthesis.         9. Pregnancy.       2. Reduced muscle mass (surge is virtually absent in blood).         7. SkaDH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.         8. Pregnancy.       2. Reduced muscle mass (scena sirvitually absent in blood).         7. SkaDH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.         8. Pregnancy.       2. Reduced muscle secretarinely.         9. Rotading bysis (re					0	
Test Name       Value       Unit       Biological Reference interval         3. Gl haemorrhage.       4. High protein intake.       5. Impaired renal function plus         6. Excess protein intake or production or tissue breakdown (e.g. infection, Gl bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).         7. Urine reabsorption (e.g. ureter colostomy)       8. Reduced muscle mass (subormal creatinine production)         9. Cartain drugs (e.g. tetracycline, gluccorticoids)       9. Cartain drugs (e.g. tetracycline, gluccorticoids)         INCREASED RATIO (<20:1) WITH ELEVATED CREATININE LEVELS:				EPORTING DATE	: 27/Aug/2024 11	:55AM
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<ul> <li>4. High protein intake.</li> <li>5. Impaired renal function plus</li> <li>6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, purns, surgery, cachexia, high fever).</li> <li>7. Urine reabsorption (e.g. ureter colostomy)</li> <li>8. Reduced muscle mass (subnormal creatinine production)</li> <li>9. Certain drugs (e.g. tetracycline, glucocorticoids)</li> <li>MCREASED RATIO (-20:1) WITH ELEVATED CREATININE LEVELS:</li> <li>1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).</li> <li>2. Prerenal azotemia superimposed on renal disease.</li> <li>DECREASED RATIO (-10:1) WITH DECREASED BUN :</li> <li>1. Acute tubular necrosis.</li> <li>2. Low protein diet and starvation.</li> <li>3. Severe liver disease.</li> <li>3. Severe liver disease.</li> <li>4. Other causes of decreased urea synthesis.</li> <li>5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).</li> <li>5. Inherited hyperanmonemias (urea is virtually absent in blood).</li> <li>7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.</li> <li>8. Pregnancy.</li> <li>DECREASED RATIO (-10:1) WITH INCREASED CREATININE:</li> <li>1. Phenacimide therapy (accelerates conversion of creatine to creatinine).</li> <li>2. Rhabdomyolysis (releases muscle creatinine).</li> <li>3. Muscular patients who develop renal failure.</li> <li>NAPPROPIATE RATIO:</li> <li>1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydra should produce an increased BUN/creatinine measurement).</li> <li>2. EstimArtEO GLOMERULAR HILERATION RATE:</li> <li>CKO STAGE DESCRIPTION AFE:</li> <li>G1 Normal kidney function &gt;90 Ne proteinuria</li> <li>G2 Kidney damage with &gt;90 Presence of Protein , Albumin or cast in urine</li> <li>G3a Mild decrease in GFR 6089</li> <li>G3b Moderate decrease in GFR 3059<td>Test Name</td><td></td><td>Value</td><td>Unit</td><td>Biologica</td><td>al Reference interval</td></li></ul>	Test Name		Value	Unit	Biologica	al Reference interval
G2Kidney damage with normal or high GFR>90Presence of Protein , Albumin or cast in urineG3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59	8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia	(e.g. ureter colostomy) ass (subnormal creatinine pro tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATIN</b> (BUN rises disproportionatel	INE LEVELS: y more than creatinine	) (e.g. obstructive urop	pathy).	
normal or high GFRAlbumin or cast in urineG3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59	<ol> <li>B. Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>INCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;1</li> <li>Acute tubular necro</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of det</li> <li>Repeated dialysis (</li> <li>Inherited hyperami</li> <li>SIADH (syndrome o</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;1</li> <li>Phenacimide thera</li> <li>Rabdomyolysis (reasonable the caused of the cause of a standard the caused of the cause of a standard the caused of the cause of a standard the cause of a standard the cause of the cause of a standard the cause of the cause of a standard the cause o</li></ol>	(e.g. ureter colostomy) ass (subnormal creatinine pro tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATIN</b> (BUN rises disproportionatel superimposed on renal diseas <b>0:1) WITH DECREASED BUN :</b> Disis. d starvation. creased urea synthesis. urea rather than creatinine d monemias (urea is virtually al f inappropiate antidiuretic ha <b>0:1) WITH INCREASED CREATIN</b> by (accelerates conversion of eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine LAR FILTERATION RATE: DESCRIPTION	INE LEVELS: y more than creatinine se. iffuses out of extracell osent in blood). irmone) due to tubular NINE: creatine to creatinine) : increase in creatinine ). e measurement).	ular fluid). secretion of urea. with certain methodo	logies,resulting in norn	mal ratio when dehydrat
G3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59	<ol> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;1</li> <li>Acute tubular necro</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of dei</li> <li>Repeated dialysis (</li> <li>Inherited hyperami</li> <li>SIADH (syndrome o</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;1</li> <li>Phenacimide thera</li> <li>Rabdomyolysis (ref</li> <li>Muscular patients of</li> <li>Muscular patients of</li> <li>Cephalosporin thera</li> <li>Cephalosporin thera</li> <li>CKD STAGE</li> <li>G1</li> </ol>	(e.g. ureter colostomy) ass (subnormal creatinine pro tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATIN</b> (BUN rises disproportionatel superimposed on renal diseas <b>0:1) WITH DECREASED BUN :</b> Disis. d starvation. creased urea synthesis. urea rather than creatinine d monemias (urea is virtually al f inappropiate antidiuretic ha <b>0:1) WITH INCREASED CREATIN</b> by (accelerates conversion of eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine <b>LAR FILTERATION RATE:</b> <u>DESCRIPTION</u>	INE LEVELS: y more than creatinine se. iffuses out of extracell osent in blood). urmone) due to tubular NINE: creatine to creatinine) e increase in creatinine measurement). NGFR ( mL/ nction	ular fluid). secretion of urea. with certain methodo <u>(min/1.73m2 ) A</u> >90	logies,resulting in norn ISSOCIATED FINDINGS	mal ratio when dehydrat
G3b Moderate decrease in GFR 30-59	B. Reduced muscle m     Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<1     Acute tubular necro     Low protein diet ar     Severe liver disease     Other causes of der     Severe liver disease     Other causes of der     SIADH (syndrome o     Pregnancy.     DECREASED RATIO (<1     Phenacimide thera     Rhabdomyolysis (re     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido     hould produce an ind     SETIMATED GLOMERU     CKD STAGE	(e.g. ureter colostomy) ass (subnormal creatinine pro tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATIN</b> (BUN rises disproportionatel superimposed on renal diseas <b>0:1) WITH DECREASED BUN :</b> Disis. d starvation.  creased urea synthesis. urea rather than creatinine d monemias (urea is virtually al f inappropiate antidiuretic ha <b>0:1) WITH INCREASED CREATIN</b> by (accelerates conversion of eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine LAR FILTERATION RATE: DESCRIPTION Normal kidney fu Kidney damage	INE LEVELS: y more than creatinine se. iffuses out of extracell osent in blood). urmone) due to tubular NINE: creatine to creatinine) e increase in creatinine e measurement). N GFR ( mL/ mction with	ular fluid). secretion of urea. with certain methodo <u>(min/1.73m2 ) A</u> >90	logies,resulting in norr SSOCIATED FINDINGS No proteinuria Presence of Protein ,	
	B. Reduced muscle m     Certain drugs (e.g.     NCREASED RATIO (>2     Prerenal azotemia     Prerenal azotemia     DECREASED RATIO (<1     Acute tubular necro     Low protein diet ar     Severe liver disease     Other causes of dec     Repeated dialysis (     SiADH (syndrome o     Pregnancy.     DECREASED RATIO (<1     Phenacimide thera     Rhabdomyolysis (re     RapPROPIATE RATIO     Diabetic ketoacido     should produce an ind     CEphalosporin ther     ESTIMATED GLOMERU     G1     G2	(e.g. ureter colostomy) ass (subnormal creatinine pro- tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATIN</b> (BUN rises disproportionatel superimposed on renal diseas <b>0:1) WITH DECREASED BUN :</b> Disis. d starvation.  creased urea synthesis. urea rather than creatinine d monemias (urea is virtually al f inappropiate antidiuretic ha <b>0:1) WITH INCREASED CREATIN</b> by (accelerates conversion of eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine LAR FILTERATION RATE: DESCRIPTION Normal kidney fu Kidney damage normal or high	INE LEVELS: y more than creatinine se. iffuses out of extracell osent in blood). irmone) due to tubular NINE: creatine to creatinine) e increase in creatinine e measurement). N GFR ( mL/ mction With GFR	ular fluid). secretion of urea. with certain methodo (min/1.73m2) A >90 Al	logies,resulting in norr SSOCIATED FINDINGS No proteinuria Presence of Protein ,	
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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)

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	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiol Chairman & Consultant Pat		(Pathology)
NAME	: Mr. YASHPAL PARMAR		
AGE/ GENDER	: 71 YRS/MALE	PATIENT ID	: 1592828
COLLECTED BY	: SURJESH	<b>REG. NO./LAB NO.</b>	: 012408270025
REFERRED BY	:	<b>REGISTRATION DATE</b>	: 27/Aug/2024 10:15 AM
BARCODE NO.	: 01515795	<b>COLLECTION DATE</b>	: 27/Aug/2024 10:33AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 27/Aug/2024 11:55AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA (	CANTT	
Test Name	Valu	ue 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

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

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	MD (Pathology &	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		n <b>Chopra</b> (Pathology) : Pathologist
AME	: Mr. YASHPAL PARMAR			
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LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
est Name		Value	Unit	Biological Reference interval
	LACT	ATE DEHYDROGEN	ASE (LDH): SERUN	Λ
ITERPRETATION:- Lactate dehydroger tythrocytes. The test can be use ancer are too erration ICREASED (MARKED Megaloblastic aner Untreated pernicio Hodgkins disease. Abdominal and lun Severe shock. Hypoxia.	d for monitoring changes in tumo c to be of use in the diagnosis of ):- mia. us anemia. g cancers.	or burden after chemo		ons in heart, liver, muscle, kidney, lung, and tate dehydrogenase elevations in patients with
NCREASED (MODERA Myocardial infarcti Pulmonary infarctio Leukemia. Hemolytic anemia. Infectious mononu	ion (MI). on and pulmonary embolism.			

KOS Diagnostic Lab (A Unit of KOS Healthcare)

\*\*\* End Of Report \*\*\*





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