



	Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta	robiology)		a m Chopra MD (Pathology) tant Pathologist	
NAME	: Miss. AARYANA YADAV				
AGE/ GENDER	: 20 YRS/FEMALE		PATIENT ID	: 1583063	3
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012408	8170006
REFERRED BY	:		REGISTRATION DAT	E : 17/Aug/	/2024 09:08 AM
BARCODE NO.	:01515194		COLLECTION DATE	: 17/Aug/	/2024 09:17AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 17/Aug/	/2024 10:44AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AME	BALA CANT	Т		
Test Name		Value	Unit		Biological Reference interval
	SWAS	STHYA V	VELLNESS PANEL:	D	
	CON	/IPLETE B	LOOD COUNT (CBC)		
RED BLOOD CELLS (I	RBCS) COUNT AND INDICES				
HAEMOGLOBIN (HB		10.8 ^L	gm/d	L	12.0 - 16.0
by CALORIMETRIC RED BLOOD CELL (R		3.81	Millio	ns/cmm	3.50 - 5.00
PACKED CELL VOLUM		34.6 ^L	%		37.0 - 50.0
MEAN CORPUSCULA		90.7	fL		80.0 - 100.0
	AUTOMATED HEMATOLOGY ANALYZER	28.3	pg		27.0 - 34.0
by CALCULATED BY	AUTOMATED HEMATOLOGY ANALYZER	ام د ما			32.0 - 36.0
	AR HEMOGLOBIN CONC. (MCHC) AUTOMATED HEMATOLOGY ANALYZER	31.2 ^L	g/dL		32.0 - 30.0
	TION WIDTH (RDW-CV) AUTOMATED HEMATOLOGY ANALYZER	13.2	%		11.00 - 16.00
-	TION WIDTH (RDW-SD)	44.6	fL		35.0 - 56.0
	AUTOMATED HEMATOLOGY ANALYZER	22.01)	
MENTZERS INDEX by CALCULATED		23.81	RATIC		BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING IND	EX	31.37	RATIC		BETA THALASSEMIA TRAIT: < =
by CALCULATED					65.0
WHITE BLOOD CELL	s (WBCS)				IRON DEFICIENCY ANEMIA: > 65.0
TOTAL LEUCOCYTE (5500	/cmm		4000 - 11000
by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY				
NUCLEATED RED BL by CALCULATED BY A MICROSCOPY	OOD CELLS (NRBCS) AUTOMATED HEMATOLOGY ANALYZER &	NIL			0.00 - 20.00
	OOD CELLS (nRBCS) %	NIL	%		< 10 %
	AUTOMATED HEMATOLOGY ANALYZER &				
DIFFERENTIAL LEUC	<u>OCYTE COUNT (DLC)</u>				



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



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

NAME	: Miss. AARYANA YADAV		
AGE/ GENDER	: 20 YRS/FEMALE	PATIENT ID	: 1583063
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Test Name	Value	Unit	Biological Reference interval

			•
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	51	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	37	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3	%	1 - 6
MONOCYTES	9	%	2 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2805	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2035	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	165	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	495	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIVE MARKED	<u>RS.</u>		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	278000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.27	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	10	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	60000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	21.5	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	15.9	%	15.0 - 17.0



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 17/Aug/2024 11:42AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT			
Test Name		Value	Unit	Biological Reference	interval
by MODIFIED WESTE	ERYT MENTATION RATE (ESR) rgren automated method	HROCYTE SEDIMENT 23 ^H	ATION RATE (ESR) mm/1st hr	0 - 20	
immune disease, but 2. An ESR can be affe as C-reactive protein	does not tell the health practiti acted by other conditions beside be used to monitor disease acti ematosus	oner exactly where the ir is inflammation. For this r	flammation is in the b eason, the ESR is typic	n associated with infection, canc ody or what is causing it. ally used in conjunction with oth ve diseases as well as some othe	her test such
A low ESR can be see (polycythaemia), sign	n with conditions that inhibit th	count (leucocytosis), and	of red blood cells, suc some protein abnorm	n as a high red blood cell count alities. Some changes in red cel	I shape (such
1. ESR and C - reactiv 2. Generally, ESR doe	e protein (C-RP) are both marke as not change as rapidly as does by as many other factors as is E	CRP, either at the start o	f inflammation or as it rker of inflammation.	resolves.	

 CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.
 If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
 Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
 Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while entities entities entities entities entities entities. aspirin, cortisone, and quinine may decrease it





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CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 17/Aug/2024 11:02AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMISTR	Y/BIOCHEMISTR	Y
		GLUCOSE FA	STING (F)	
): Plasma	84.46	mg/dL	NORMAL: < 100.0

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 PROFILE	: BASIC	
CHOLESTEROL TOTAL by CHOLESTEROL OXI		132.65	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239. HIGH CHOLESTEROL: > OR = 240
TRIGLYCERIDES: SERI	JM HATE OXIDASE (ENZYMATIC)	90.35	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (E		51.11	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: S by CALCULATED, SPEC		63.47	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159. HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTER by CALCULATED, SPEC		81.54	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: by CALCULATED, SPEC		18.07	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUN by CALCULATED, SPEC	1	355.65	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL R by CALCULATED, SPEC	ATIO: SERUM	2.6	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: SERI		1.24	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD	L RATIO: SERUM	1.77 ^L	RATIO	3.00 - 5.00

INTERPRETATION:

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

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

 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 Dr. Yugam Chopra MD (Pathology) MD (Pathology & Microbiology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Miss. AARYANA YADAV AGE/ GENDER : 20 YRS/FEMALE **PATIENT ID** :1583063 **COLLECTED BY** : SURJESH :012408170006 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 17/Aug/2024 09:08 AM : **BARCODE NO.** :01515194 **COLLECTION DATE** :17/Aug/2024 09:17AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :17/Aug/2024 11:02AM : 6349/1, NICHOLSON ROAD, AMBALA CANTT **CLIENT ADDRESS** Test Name Value Unit LIVER FUNCTION TEST (COMPLETE)

LIVE			
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.44	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by diazo modified, spectrophotometry	0.13	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.31	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	28.4	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	12.8	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.22	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	55.52	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by szasz, spectrophtometry	9.66	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.23	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by bromocresol green	4.21	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.02 ^L	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by calculated, spectrophotometry	2.08 ^H	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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Biological Reference interval

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT E E

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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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	K	DNEY FUNCTION	TEST (COMPLETE)	
UREA: SERUM		19.5	mg/dL	10.00 - 50.00
-	ATE DEHYDROGENASE (GLDH)			
CREATININE: SERUM		0.79	mg/dL	0.40 - 1.20
by ENZYMATIC, SPECTROPHOTOMETERY BLOOD UREA NITROGEN (BUN): SERUM		9.11	mg/dL	7.0 - 25.0
by CALCULATED, SPE				
BLOOD UREA NITRO RATIO: SERUM	GEN (BUN)/CREATININE	11.53	RATIO	10.0 - 20.0
by CALCULATED, SPE	CTROPHOTOMETRY			
UREA/CREATININE RATIO: SERUM		24.68	RATIO	
by CALCULATED, SPE URIC ACID: SERUM	CTROPHOTOMETRY	4.03	mg/dL	2.50 - 6.80
by URICASE - OXIDAS	E PEROXIDASE	4.03	iiig/uL	2.30 - 0.60
CALCIUM: SERUM		9.2	mg/dL	8.50 - 10.60
by ARSENAZO III, SPECTROPHOTOMETRY		3.73	ma/dl	2 20 4 70
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY		3.73	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM		140.1	mmol/L	135.0 - 150.0
by ISE (ION SELECTIVE ELECTRODE)		4.17		
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)		4.16	mmol/L	3.50 - 5.00
CHLORIDE: SERUM	- /	105.07	mmol/L	90.0 - 110.0
by ISE (ION SELECTIVE ELECTRODE)				
	RULAR FILTERATION RATE			
ESTIMATED GLOMERULAR FILTERATION RATE		109.8		
(eGFR): SERUM by CALCULATED				

by CALCULATED **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 Name		Value Unit	t Biolog	ical Reference interval
8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia	superimposed on renal disease.		uropathy).	
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8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1 G2	(e.g. ureter colostomy) ass (subnormal creatinine product tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATININE L a (BUN rises disproportionately mo superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine diffusi- monemias (urea is virtually absen- of inappropiate antidiuretic harmon 10:1) WITH INCREASED CREATININE py (accelerates conversion of crea- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false incr creased BUN/creatinine ratio). apy (interferes with creatinine me <u>JLAR FILTERATION RATE:</u> <u>DESCRIPTION</u> Normal kidney function Kidney damage with normal or high GFR	EVELS: ore than creatinine) (e.g. obstructive of the extracellular fluid). t in blood). ne) due to tubular secretion of urea. : tine to creatinine). ease in creatinine with certain meth asurement). on >90 20 >90 21 60 - 89 GFR 30-59	odologies,resulting in no ASSOCIATED FINDINGS No proteinuria Presence of Protein ,	δ

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	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Patholog		(Pathology)
NAME	: Miss. AARYANA YADAV		
AGE/ GENDER	: 20 YRS/FEMALE	PATIENT ID	: 1583063
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012408170006
REFERRED BY	:	REGISTRATION DATE	: 17/Aug/2024 09:08 AM
BARCODE NO.	: 01515194	COLLECTION DATE	: 17/Aug/2024 09:17AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 17/Aug/2024 11:02AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	ГТ	
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





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



		nay Chopra hology & Microbiology) an & Consultant Patholog	gy & Microbiology) ME		m Chopra D (Pathology) nt Pathologist		
NAME	: Miss. AARYANA YA)		5				
GE/ GENDER	: 20 YRS/FEMALE		PATIENT	D	: 1583063		
OLLECTED BY	: SURJESH		REG. NO./		:012408170	1006	
EFERRED BY	. SUMEST			TION DATE	: 17/Aug/2024		
BARCODE NO.	: 01515194		COLLECTI		8		
CLIENT CODE.	: KOS DIAGNOSTIC LA	D	REPORTI		: 17/Aug/202		
LIENT ADDRESS		N ROAD, AMBALA CANT		IG DATE	: 17/Aug/2024	4 11.JJAW	
LIENI ADDRESS	. 0349/1, MCHOLSOI	N KOAD, AMDALA CAN I	. 1				
Test Name		Value		Unit	Biolo	ogical Reference interval	
		VI	ITAMINS				
		VITAMIN D/25	HYDROXY V	ITAMIN D3			
/ITAMIN D (25-HYDI	ROXY VITAMIN D3): SE	RUM 7.8 ^L		ng/mL	DEFI	CIENCY: < 20.0	
	IESCENCE IMMUNOÁSSA			3	INSU	IFFICIENCY: 20.0 - 30.0	
						ICIENCY: 30.0 - 100.0	
					TOXI	CITY: > 100.0	
<u>NTERPRETATION:</u> DEFIC	CIENT:	< 20		nc	ı/mL		
INSUFF	FICIENT:	21 - 29		ng	ı/mL		
PREFFERED RANGE: INTOXICATION:		<u> </u>			g/mL		
Vitamin D plays a p hosphate reabsorpt .Severe deficiency m ECREASED: .Lack of sunshine ex .Inadequate intake, .Depressed Hepatic .Secondary to advan .Osteoporosis and S .Enzyme Inducing dr VCREASED: . Hypervitaminosis E evere hypercalcemia AUTION : Replaceme ypervitaminosis D	ion, skeletal calcium de hay lead to failure to mi malabsorption (celiac o Vitamin D 25- hydroxyla ced Liver disease econdary Hyperparathr ugs: anti-epileptic drug D is Rare, and is seen on a and hyperphophatemia nt therapy in deficient i individuals as compare to	tenance of calcium hom position, calcium mobili neralize newly formed c lisease) ase activity oidism (Mild to Modera s like phenytoin, phenol ly after prolonged expos a. ndividuals must be mon	ization, mainh osteoid in bon te deficiency) barbital and c sure to extren nitored by peri	y regulated by p e, resulting in r arbamazepine, f nely high doses odic assessmen	arathyroid harm ckets in children hat increases Vit of Vitamin D. Wh t of Vitamin D lev	and osteomalacia in adults. tamin D metabolism. hen it occurs, it can result in	
		*** End Of I	Report **	*			
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