

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta	robiology)		Pathology)
NAME	: Mr. RAJESH KAPOOR			
AGE/ GENDER	: 70 YRS/MALE		PATIENT ID	: 1614321
COLLECTED BY	:		REG. NO./LAB NO.	: 012409160017
REFERRED BY	:		REGISTRATION DATE	: 16/Sep/2024 08:57 AM
BARCODE NO.	: 01517048		COLLECTION DATE	: 16/Sep/2024 09:02AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 16/Sep/2024 09:49AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AME	ALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	SWAS		LLNESS PANEL: DT	
			DOD COUNT (CBC)	
RED BLOOD CELLS (R	BCS) COUNT AND INDICES			
HAEMOGLOBIN (HB)		13.8	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (RB		4.7	Millions/cr	nm 3.50 - 5.00
by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE IE (PCV)	43.8	%	40.0 - 54.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
MEAN CORPUSCULAI	R VOLUME (MCV) UTOMATED HEMATOLOGY ANALYZER	93.3	fL	80.0 - 100.0
MEAN CORPUSCULA	R HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	29.5	pg	27.0 - 34.0
MEAN CORPUSCULA	R HEMOGLOBIN CONC. (MCHC)	31.6 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUT	UTOMATED HEMATOLOGY ANALYZER	12.8	%	11.00 - 16.00
	UTOMATED HEMATOLOGY ANALYZER ION WIDTH (RDW-SD)	44.9	fL	35.0 - 56.0
by CALCULATED BY A MENTZERS INDEX	UTOMATED HEMATOLOGY ANALYZER	19.85	RATIO	BETA THALASSEMIA TRAIT: < 13.0
by CALCULATED		17.00	NATIO 1	IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDE	X	25.53	RATIO	BETA THALASSEMIA TRAIT:<= 65.
by CALCULATED WHITE BLOOD CELLS	(WBCS)			IRON DEFICIENCY ANEMIA: > 65.0
TOTAL LEUCOCYTE CO		8170	/cmm	4000 - 11000
by FLOW CYTOMETRY	BY SF CUBE & MICROSCOPY		, smith	
NUCLEATED RED BLC by AUTOMATED 6 PAR	OOD CELLS (NRBCS)	NIL		0.00 - 20.00
NUCLEATED RED BLC	OD CELLS (nRBCS) %	NIL	%	< 10 %
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
NEUTROPHILS		53	%	50 - 70
	BY SF CUBE & MICROSCOPY	00	70	00 /0



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







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Test Name		Value	Unit	Biological Reference interval
	' BY SF CUBE & MICROSCOPY	36	%	20 - 40
EOSINOPHILS	BY SF CUBE & MICROSCOPY	5	%	1 - 6
MONOCYTES	BY SF CUBE & MICROSCOPY	6	%	2 - 12
BASOPHILS	BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCY				
ABSOLUTE NEUTROP	HIL COUNT BY SF CUBE & MICROSCOPY	4330	/cmm	2000 - 7500
ABSOLUTE LYMPHOC		2941	/cmm	800 - 4900
ABSOLUTE EOSINOPH		408	/cmm	40 - 440
ABSOLUTE MONOCY by FLOW CYTOMETRY	TE COUNT ' by sf cube & microscopy	490	/cmm	80 - 880
ABSOLUTE BASOPHIL by FLOW CYTOMETRY	. COUNT Y BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTH	IER PLATELET PREDICTIVE MARKE	<u>RS.</u>		
PLATELET COUNT (PL by HYDRO DYNAMIC F	T) OCUSING, ELECTRICAL IMPEDENCE	197000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE	0.24	%	0.10 - 0.36
MEAN PLATELET VOL by HYDRO DYNAMIC F	UME (MPV) ocusing, electrical impedence	12 ^H	fL	6.50 - 12.0
PLATELET LARGE CEL by HYDRO DYNAMIC F	L COUNT (P-LCC) ocusing, electrical impedence	81000	/cmm	30000 - 90000
PLATELET LARGE CEL by HYDRO DYNAMIC F	L RATIO (P-LCR) ocusing, electrical impedence	41.2	%	11.0 - 45.0
	ION WIDTH (PDW) ocusing, electrical impedence CTED ON EDTA WHOLE BLOOD	16.4	%	15.0 - 17.0



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est Name		Value	Unit	Biological Reference interval
	ERYTH	ROCYTE SEDIMENT	ATION RATE (ES	R)
	MENTATION RATE (ESR) RGREN AUTOMATED METHOD	28 ^H	mm/1st l	hr 0 - 20
mmune disease, but	does not tell the health practition	ner exactly where the inf inflammation. For this re	flammation is in the eason, the ESR is ty	ion associated with infection, cancer and auto- e body or what is causing it. pically used in conjunction with other test such bove diseases as well as some others, such as

 2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
 3. CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.
 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 aspiring cortisonal and quipino may decrease it. aspirin, cortisone, and quinine may decrease it





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Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMISTR	Y/BIOCHEMISTR	Y
		GLUCOSE FA	STING (F)	
GLUCOSE FASTING (by glucose oxidas	F): PLASMA E - PEROXIDASE (GOD-POD)	196.25 ^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0
 A fasting plasma g A fasting plasma g test (after consumpti 	on of 75 gms of glucose) is recor	considered normal. mg/dl is considered as nmended for all such	patients.	prediabetic. A fasting and post-prandial bloc at post-prandial is strongly recommended fo

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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFILE	: BASIC	
CHOLESTEROL TOTA	L: SERUM	194.67	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX				BORDERLINE HIGH: 200.0 - 239 HIGH CHOLESTEROL: > OR = 24
TRIGLYCERIDES: SER by GLYCEROL PHOSP	UM HATE OXIDASE (ENZYMATIC)	88.44	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (by SELECTIVE INHIBIT		62.28	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0
				HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: S by CALCULATED, SPE		114.7	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 CHOLESTE by calculated, spe		132.39 ^H	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, SPE		17.69	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUI by CALCULATED, SPE		477.78	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL I by CALCULATED, SPE		3.13	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: SER by calculated, spe		1.84	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	AD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD		1.42 ^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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			1	
Test Name		Value	Unit	Biological Reference interval
1	10/2			
			ON TEST (COMPLETE)	
BILIRUBIN TOTAL: S		0.75	mg/dL	INFANT: 0.20 - 8.00
				ADULT: 0.00 - 1.20
	CONJUGATED): SERUM	0.2	mg/dL	0.00 - 0.40
	(UNCONJUGATED): SERUM	0.55	mg/dL	0.10 - 1.00
by CALCULATED, SPE				
SGOT/AST: SERUM		23.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM	RIDOXAL PHOSPHATE	19.5	U/L	0.00 - 49.00
	RIDOXAL PHOSPHATE	17.5	0/1	0.00 - 47.00
AST/ALT RATIO: SER		1.22	RATIO	0.00 - 46.00
by CALCULATED, SPE		110 17	11/1	40.0.400.0
ALKALINE PHOSPHA by PARA NITROPHEN PROPANOL	TASE: SERUIVI YL PHOSPHATASE BY AMINO METHYL	113.17	U/L	40.0 - 130.0
GAMMA GLUTAMYL by szasz, spectrof	. TRANSFERASE (GGT): SERUM PHTOMETRY	54	U/L	0.00 - 55.0
TOTAL PROTEINS: SI		6.1 ^L	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		3.71	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPE	CTROPHOTOMETRY	2.39	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPE		1.55	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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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).

PROGNOSTIC SIGNIFICANCE:

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



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Test Name		Value	Unit	Biological Reference interval
	KI		N TEST (COMPLETE)	
UREA: SERUM		30.81	mg/dL	10.00 - 50.00
•	NATE DEHYDROGENASE (GLDH)			
CREATININE: SERUN by ENZYMATIC, SPEC		0.86	mg/dL	0.40 - 1.40
-	DGEN (BUN): SERUM	14.4	mg/dL	7.0 - 25.0
by CALCULATED, SPE	ECTROPHOTOMETRY			
BLOOD UREA NITRC RATIO: SERUM	OGEN (BUN)/CREATININE	16.74	RATIO	10.0 - 20.0
	ECTROPHOTOMETRY			
UREA/CREATININE F		35.83	RATIO	
by CALCULATED, SPE URIC ACID: SERUM	ECTROPHOTOMETRY	3.65	mg/dL	3.60 - 7.70
by URICASE - OXIDAS	SE PEROXIDASE	5.05	ing/uL	5.00 - 7.70
CALCIUM: SERUM		9.47	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE PHOSPHOROUS: SEF		3.24	mg/dL	2.30 - 4.70
	DATE, SPECTROPHOTOMETRY	5.24	ing/uL	2.30 - 4.70
ELECTROLYTES				
sodium: serum		138.2	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV POTASSIUM: SERUM		4.33	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV		4.33	THITIOI/L	5.50 - 5.00
CHLORIDE: SERUM		103.65	mmol/L	90.0 - 110.0
by ISE (ION SELECTIV FSTIMATED GLOME	'E ELECTRODE) RULAR FILTERATION RATE			
	RULAR FILTERATION RATE	93.1		
(eGFR): SERUM		73.1		
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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LIENI ADDRESS	. 0349/1, MIC	HOLSON KOAD, AMDAI	LA CANTI			
Test Name			Value	Unit	Biological	Reference interval
7. Urine reabsorption 3. Reduced muscle m 9. Certain drugs (e.g. NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< ⁷	ass (subnormal tetracycline, gli 0:1) WITH ELEV (BUN rises disp superimposed (0:1) WITH DECF	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more th on renal disease.	S:		osis, Cushing's syndron ithy).	
7. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. NCREASED RATIO (>2 4. Postrenal azotemia DECREASED RATIO (~ 5. Low protein diet ar 6. Severe liver disease 6. Other causes of de 6. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (~ 1. Phenacimide thera 2. Rhabdomyolysis (r	(e.g. ureter colu ass (subnormal tetracycline, glu 0:1) WITH ELEV (BUN rises disp superimposed of 0:1) WITH DECF osis. d starvation. creased urea sy urea rather tha monemias (ure of inappropiate 0:1) WITH INCR py (accelerates eleases muscle	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more th on renal disease. EASED BUN : n creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d EASED CREATININE: conversion of creatine s creatinine).	S: an creatinine) (e.g. It of extracellular flu lood). ue to tubular secret	obstructive uropa iid).		
. Urine reabsorption . Reduced muscle m . Certain drugs (e.g. VCREASED RATIO (>2 . Postrenal azotemia Perenal azotemia PECREASED RATIO (< . Acute tubular necr . Low protein diet ar . Severe liver disease . Other causes of de . Repeated dialysis (. Inherited hyperam . SIADH (syndrome of . Pregnancy. PECREASED RATIO (< . Phenacimide thera . Rhabdomyolysis (r . Muscular patients VAPPROPIATE RATIO . Diabetic ketoacido hould produce an in . Cephalosporin ther	(e.g. ureter colu ass (subnormal tetracycline, glu 0:1) WITH ELEV. (BUN rises disp superimposed of 0:1) WITH DECF osis. ad starvation. b creased urea sy urea rather tha monemias (urea of inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoacetatic creased BUN/cr apy (interferes	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more the on renal disease. EASED BUN : n creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d EASED CREATININE: conversion of creatine for creatinine). nal failure. re causes false increase eatinine ratio). with creatinine measure	S: an creatinine) (e.g. it of extracellular flu lood). ue to tubular secret to creatinine). in creatinine with c	obstructive uropa iid). ion of urea.	ithy).	al ratio when dehydratic
. Urine reabsorption . Reduced muscle m . Certain drugs (e.g. VCREASED RATIO (>2 . Postrenal azotemia Perenal azotemia DECREASED RATIO (< . Acute tubular necr . Low protein diet ar . Severe liver disease . Other causes of de . Repeated dialysis (. Inherited hyperam . SIADH (syndrome c . Pregnancy. DECREASED RATIO (< . Phenacimide thera . Rhabdomyolysis (r . Muscular patients VAPPROPIATE RATIO . Diabetic ketoacido hould produce an in . Cephalosporin ther	(e.g. ureter colu ass (subnormal tetracycline, glu 0:1) WITH ELEV. (BUN rises disp superimposed of 0:1) WITH DECF osis. ad starvation. b creased urea sy urea rather tha monemias (urea of inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoacetatic creased BUN/cr apy (interferes	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more the on renal disease. EASED BUN : n creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d EASED CREATININE: conversion of creatine for creatinine). nal failure. re causes false increase eatinine ratio). with creatinine measure	S: an creatinine) (e.g. it of extracellular flu lood). ue to tubular secret to creatinine). in creatinine with c	obstructive uropa nid). ion of urea. ertain methodolo	ithy).	
. Urine reabsorption . Reduced muscle m . Certain drugs (e.g. . VCREASED RATIO (>2 . Postrenal azotemia Prerenal azotemia ECREASED RATIO (<' . Acute tubular necr . Low protein diet ar . Severe liver disease . Other causes of de . Repeated dialysis (. Inherited hyperam . SIADH (syndrome of . Pregnancy. ECREASED RATIO (<' . Phenacimide thera . Rhabdomyolysis (r . Muscular patients VAPPROPIATE RATIO . Diabetic ketoacido nould produce an in . Cephalosporin ther STIMATED GLOMERI CKD STAGE G1	(e.g. ureter colu ass (subnormal tetracycline, glu 0:1) WITH ELEV (BUN rises disp superimposed of 0:1) WITH DECF osis. ad starvation. creased urea sy urea rather tha monemias (urea of inappropiate of inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoacetat creased BUN/cr apy (interferes DLAR FILTERATIC No	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more the on renal disease. EASED BUN : In thesis. In creatinine diffuses out a is virtually absent in b antidiuretic harmone) d EASED CREATININE: conversion of creatine for creatinine). nal failure. In a failure.	S: an creatinine) (e.g. it of extracellular flu lood). ue to tubular secret to creatinine). in creatinine with c ement). GFR (mL/min/1 >90	obstructive uropa nid). ion of urea. ertain methodolo 73m2) AS	ogies,resulting in norma SOCIATED FINDINGS No proteinuria	
. Urine reabsorption . Reduced muscle m . Certain drugs (e.g. VCREASED RATIO (>2 . Postrenal azotemia Perenal azotemia PECREASED RATIO (< . Acute tubular necr . Low protein diet ar . Severe liver disease . Other causes of de . Repeated dialysis (. Inherited hyperam . SIADH (syndrome of . Pregnancy. PECREASED RATIO (< . Phenacimide thera . Rhabdomyolysis (r . Muscular patients VAPPROPIATE RATIO . Diabetic ketoacido hould produce an in . Cephalosporin ther STIMATED GLOMERL CKD STAGE	(e.g. ureter colu ass (subnormal tetracycline, glu 0:1) WITH ELEV (BUN rises disp superimposed of 0:1) WITH DECF osis. ad starvation. creased urea sy urea rather tha monemias (urea of inappropiate of inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoacetat creased BUN/cr apy (interferes DLAR FILTERATIC No	ostomy) creatinine production) ucocorticoids) ATED CREATININE LEVEL proportionately more the on renal disease. EASED BUN : n creatinine diffuses ou a is virtually absent in b antidiuretic harmone) d EASED CREATININE: conversion of creatine for creatinine). nal failure. re causes false increase eatinine ratio). with creatinine measure N RATE: DESCRIPTION	S: an creatinine) (e.g. it of extracellular flu lood). ue to tubular secret to creatinine). in creatinine with c ement). GFR (mL/min/1	obstructive uropa nid). ion of urea. ertain methodolo 73m2) AS	ogies,resulting in norma	

Moderate decrease in GFR
Severe decrease in GFR
Kidney failure

30-59

15-29

<15

DR.YUGAM CHOPRA



G3b

G4

G5



CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)







	Dr. Vinay Chopra MD (Pathology & Microbiolo Chairman & Consultant Path		(Pathology)
NAME	: Mr. RAJESH KAPOOR		
AGE/ GENDER	: 70 YRS/MALE	PATIENT ID	: 1614321
COLLECTED BY	:	REG. NO./LAB NO.	: 012409160017
REFERRED BY	:	REGISTRATION DATE	: 16/Sep/2024 08:57 AM
BARCODE NO.	: 01517048	COLLECTION DATE	: 16/Sep/2024 09:02AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 16/Sep/2024 11:36AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA C	ANTT	
Test Name	Valu	e 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)

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana 0171-2643898, +91 99910 43898 care@koshealthcare.com www.koshealthcare.com







	MD (Pathology & Mi	Dr. Vinay Chopra ID (Pathology & Microbiology) hairman & Consultant Pathologist CEC		n Chopra (Pathology) Pathologist	
NAME	: Mr. RAJESH KAPOOR				
AGE/ GENDER	: 70 YRS/MALE		PATIENT ID	: 1614321	
COLLECTED BY	:	REG. NO./LAB NO.		: 012409160017	
REFERRED BY	:		REGISTRATION DATE	: 16/Sep/2024 08:57 AM	
BARCODE NO.	: 01517048		COLLECTION DATE	: 16/Sep/2024 09:02AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 16/Sep/2024 10:35AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
		ENDOC	RINOLOGY		
	THY	ROID FUN	CTION TEST: TOTAL		
TRIIODOTHYRONINE		0.861	ng/mL	0.35 - 1.93	
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY) THYROXINE (T4): SERUM 7.74 by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)		7.74	µgm/dL	4.87 - 12.60	
	NG HORMONE (TSH): SERUM escent microparticle immunoassa rasensitive	3.766 (Y)	µIU/mL	0.35 - 5.50	

KOS Diagnostic Lab

(A Unit of KOS Healthcare)

CLINICAL CONDITION T3 T4 TSH Primary Hypothyroidism: Reduced Reduced Increased (Significantly) Subclinical Hypothyroidism: Normal or Low Normal Normal or Low Normal High Reduced (at times undetectable) Primary Hyperthyroidism: Increased Increased Subclinical Hyperthyroidism: Normal or High Normal Normal or High Normal Reduced

LIMITATIONS:-

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG), and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.

2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (eg: phenytoin , salicylates).

3. Serum T4 levles in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.

4. TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothroidism, pregnancy, phenytoin therapy.

TRIIODOTH	(RONINE (T3)	THYROXINE (T4)		THYROID STIMULATING HORMONE (TSH)	
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range (μIU/mL)
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 - 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 - 17.04	3 Days – 6 Months	0.70 - 8.40





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

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







0.30 - 4.10

		Dr. Vinay Chop MD (Pathology & M Chairman & Consul	icrobiology)		I gam Choj MD (Patholo ultant Patholo	ogy)	
NAME	: Mr. RAJESH	KAPOOR					
AGE/ GENDER	: 70 YRS/MAI	.E]	PATIENT ID	: 161	4321	
COLLECTED BY	:]	REG. NO./LAB NO.	:01	24091600	17
REFERRED BY	:]	REGISTRATION DA	TE : 16/	/Sep/2024 (08:57 AM
BARCODE NO.	:01517048			COLLECTION DATE	:16/	Sep/2024 (09:02AM
CLIENT CODE.	: KOS DIAGNO	OSTIC LAB]	REPORTING DATE	:16/	'Sep/2024 1	10:35AM
CLIENT ADDRESS	6349/1, NIC	HOLSON ROAD, AM	ÍBALA CANTT				
Test Name			Value	Unit		Biolog	ical Reference interval
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00		
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50		
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20	11 – 19 Years	0.50 - 5.50		
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35-5.50		
		MENDATIONS OF TSH LE	VELS DURING PREC				
	1st Trimester			0.10 - 2.50			ļ
2nd Trimester			0.20 – 3.00			1	

INCREASED TSH LEVELS:

1.Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.

2.Hypothyroid patients receiving insufficient thyroid replacement therapy.

3.Hashimotos thyroiditis

4.DRUGS: Amphetamines, idonie containing agents & dopamine antagonist.

5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

DECREASED TSH LEVELS:

1.Toxic multi-nodular goitre & Thyroiditis.

2. Over replacement of thyroid harmone in treatment of hypothyroidism.

3. Autonomously functioning Thyroid adenoma

4.Secondary pituatary or hypothalmic hypothyroidism

3rd Trimester

5. Acute psychiatric illness

6.Severe dehydration.

7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

8.Pregnancy: 1st and 2nd Trimester



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





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



NAME	: Mr. RAJESH KAPOOR			
AGE/ GENDER	: 70 YRS/MALE		PATIENT ID	: 1614321
COLLECTED BY	:		REG. NO./LAB NO.	: 012409160017
REFERRED BY	:		REGISTRATION DATE	: 16/Sep/2024 08:57 AM
BARCODE NO.	: 01517048		COLLECTION DATE	: 16/Sep/2024 09:02AM
CLIENT CODE. CLIENT ADDRESS	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD,	AMBALA CANT	REPORTING DATE T	: 16/Sep/2024 10:35AM
Test Name		Value	Unit	Piological Deference interval
		value	Unit	Biological Reference interval
		VI	TAMINS	
	VI	TAMIN D/25	HYDROXY VITAMIN D3	
	ROXY VITAMIN D3): SERUM	54.1	ng/mL	DEFICIENCY: < 20.0
by CLIA (CHEMILUMIN	IESCENCE IMMUNOASSAY)			INSUFFICIENCY: 20.0 - 30.0
				SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
NTERPRETATION:				
DEFI		< 20		g/mL
DEFI INSUF	ICIENT: FICIENT: ED RANGE:	< 20 21 - 29 30 - 100	n	g/mL g/mL g/mL
DEFI INSUF PREFFER INTOX	Ficient: Ed Range: Ication:	21 - 29 30 - 100 > 100	n n n	g/mL g/mL g/mL
DEFI INSUF PREFFER INTOX .Vitamin D compou onversion of 7- dipy	FICIENT: ED RANGE: ICATION: nds are derived from dietary ero vdrocholecalciferol to Vitamin D:	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upp	n plants, Vitamin D2), or cho	g/mL g/mL g/mL g/mL Ilecalciferol (from animals, Vitamin D3), or by
DEFI INSUF PREFFER INTOX .Vitamin D compou onversion of 7- dihy 2.25-OHVitamin D i issue and tightly bo	FICIENT: ED RANGE: ICATION: nds are derived from dietary ercy vdrocholecalciferol to Vitamin Di represents the main body resevo und by a transport protein while	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upo ir and transport in circulation.	n plants, Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans	g/mL g/mL g/mL Jecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose
DEFI INSUF PREFFER INTOX .Vitamin D compou conversion of 7- dih 2.25-OHVitamin D l issue and tightly bo 3.Vitamin D plays a p	FICIENT: ED RANGE: ICATION: nds are derived from dietary erc vdrocholecalciferol to Vitamin Di represents the main body resevo und by a transport protein while orimary role in the maintenance	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upc ir and transport in circulation. of calcium hom	n plants, Vitamin D2), or chc on Ultraviolet exposure. form of Vitamin D and trans eostatis. It promotes calciur	g/mL g/mL g/mL Jecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and
DEFI INSUF PREFFER INTOX I. Vitamin D compou conversion of 7- dihv 2.25-OHVitamin D p issue and tightly bo 3. Vitamin D plays a p bhosphate reabsorp 4. Severe deficiency f	FICIENT: ED RANGE: ICATION: nds are derived from dietary erc vdrocholecalciferol to Vitamin D represents the main body resevo und by a transport protein while primary role in the maintenance tion, skeletal calcium deposition	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upc ir and transport a in circulation. of calcium homili	n n n plants, Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans leostatis. It promotes calciur zation, mainly regulated by	g/mL g/mL g/mL lecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and
DEFI INSUF PREFFER INTOX I.Vitamin D compou conversion of 7- dihy 2.25-OHVitamin D r issue and tightly bo 3.Vitamin D plays a r ohosphate reabsorp 4.Severe deficiency r DECREASED: I.Lack of sunshine ex	FICIENT: ED RANGE: ICATION: nds are derived from dietary ero vdrocholecalciferol to Vitamin D represents the main body resevo und by a transport protein while primary role in the maintenance tion, skeletal calcium deposition may lead to failure to mineralize xposure.	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upc ir and transport a in circulation. of calcium homili	n n n plants, Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans leostatis. It promotes calciur zation, mainly regulated by	g/mL g/mL g/mL lecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH).
DEFI INSUE PREFFER INTOX I.Vitamin D compou conversion of 7- dihy 2.25-OHVitamin D f issue and tightly bo 3.Vitamin D plays a p ohosphate reabsorp 4.Severe deficiency f DECREASED: 1.Lack of sunshine e; 2.Inadeguate intake	FICIENT: ED RANGE: ICATION: ICATION: Icarion: Icari	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upo ir and transport e in circulation. of calcium hom , calcium mobili newly formed c	n n n plants, Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans leostatis. It promotes calciur zation, mainly regulated by	g/mL g/mL g/mL lecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH).
DEFI INSUF PREFFER INTOX I.Vitamin D compou conversion of 7- dihy 2.25-OHVitamin D plays a issue and tightly bo 3.Vitamin D plays a phosphate reabsorp 4.Severe deficiency n DECREASED: 1.Lack of sunshine e: 2.Inadequate intake 3.Depressed Hepatic 4.Secondary to adva	FICIENT: ED RANGE: ICATION: ICATION: Idva are derived from dietary ero vdrocholecalciferol to Vitamin D. represents the main body resevo und by a transport protein while primary role in the maintenance tion, skeletal calcium deposition may lead to failure to mineralize xposure. , malabsorption (celiac disease) : Vitamin D 25- hydroxylase activ nced Liver disease	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upo ir and transport e in circulation. of calcium hom , calcium mobili newly formed c	n n n plants, Vitamin D2), or chc on Ultraviolet exposure. form of Vitamin D and trans reostatis. It promotes calciur zation, mainly regulated by osteoid in bone, resulting in i	g/mL g/mL g/mL lecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH).
DEFI INSUF PREFFER INTOX I.Vitamin D compou conversion of 7- dihy 2.25-OHVitamin D plays a p dissue and tightly bo 3.Vitamin D plays a p dissphate reabsorp 4.Severe deficiency n DECREASED: 1.Lack of sunshine e: 2.Inadequate intake 3.Depressed Hepatic 4.Secondary to adva 5.Osteoporosis and 5 5.Enzyme Inducing d	FICIENT: ED RANGE: ICATION: ICATION: Idracode a content of the second represents the main body resevored und by a transport protein while primary role in the maintenance tion, skeletal calcium deposition may lead to failure to mineralize xposure. , malabsorption (celiac disease) : Vitamin D 25- hydroxylase active nced Liver disease Secondary Hyperparathroidism (21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upo ir and transport e in circulation. of calcium hom , calcium mobili newly formed c	n plants, Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans eostatis. It promotes calciur zation, mainly regulated by osteoid in bone, resulting in i	g/mL g/mL g/mL lecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH).
DEFI INSUF PREFFER INTOX I.Vitamin D compou conversion of 7- dihy 2.25-OHVitamin D r issue and tightly bo 3.Vitamin D plays a r phosphate reabsorp 4.Severe deficiency r DECREASED: I.Lack of sunshine ex 2.Inadequate intake 3.Depressed Hepatic 4.Secondary to adva 5.Osteoporosis and 5 5.Enzyme Inducing d NCREASED:	FICIENT: ED RANGE: ICATION: Inds are derived from dietary ero vdrocholecalciferol to Vitamin D. represents the main body resevo und by a transport protein while primary role in the maintenance tion, skeletal calcium deposition may lead to failure to mineralize xposure. , malabsorption (celiac disease) : Vitamin D 25- hydroxylase activ nced Liver disease Secondary Hyperparathroidism (lrugs: anti-epileptic drugs like ph	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin uponism ir and transport in circulation. of calcium hom , calcium mobili newly formed con ity Mild to Moderation enytoin, phenot	n plants, Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans reostatis. It promotes calciur zation, mainly regulated by osteoid in bone, resulting in r te deficiency) parbital and carbamazepine,	g/mL g/mL g/mL glecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH). rickets in children and osteomalacia in adults.
DEFI INSUE PREFFER INTOX 1. Vitamin D compou conversion of 7- dihy 2.25-OHVitamin D fi issue and tightly bo 3. Vitamin D plays a p ohosphate reabsorp 4. Severe deficiency f DECREASED: 1. Lack of sunshine ei 2. Inadeguate intake 3. Depressed Hepatic 4. Secondary to adva 5. Osteoporosis and S 6. Enzyme Inducing d NCREASED: 1. Hypervitaminosis severe hypercalcemi	FICIENT: ED RANGE: ICATION: ICATION: Inds are derived from dietary erc ydrocholecalciferol to Vitamin D: represents the main body resevo und by a transport protein while primary role in the maintenance tion, skeletal calcium deposition may lead to failure to mineralize xposure. , malabsorption (celiac disease) : Vitamin D 25- hydroxylase activ nced Liver disease Secondary Hyperparathroidism (lrugs: anti-epileptic drugs like ph D is Rare, and is seen only after a and hyperphophatemia.	21 - 29 30 - 100 > 100 localciferol (fror 3 in the skin upo ir and transport e in circulation. of calcium hom , calcium mobili newly formed c ity Mild to Modera enytoin, phenot prolonged expos	n plants. Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans eostatis. It promotes calciur zation, mainly regulated by osteoid in bone, resulting in r barbital and carbamazepine, sure to extremely high doses	g/mL g/mL g/mL lecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH). rickets in children and osteomalacia in adults. that increases Vitamin D metabolism. of Vitamin D. When it occurs, it can result in
DEFI INSUF PREFFER INTOX I.Vitamin D compou conversion of 7- dihy 2.25-OHVitamin D n issue and tightly bo 3.Vitamin D plays a p ohosphate reabsorp 4.Severe deficiency n DECREASED: 1.Lack of sunshine ex 2.Inadeguate intake 3.Depressed Hepatic 4.Secondarv to adva 5.Osteoporosis and S 5.Enzyme Inducing d NCREASED: 1. Hypervitaminosis Severe hypercalcemi CAUTION: Replaceme hypervitaminosis D	FICIENT: ED RANGE: ICATION: ICATION: Inds are derived from dietary ero vdrocholecalciferol to Vitamin D. represents the main body resevo und by a transport protein while primary role in the maintenance tion, skeletal calcium deposition may lead to failure to mineralize xposure. , malabsorption (celiac disease) vitamin D 25- hydroxylase active nced Liver disease Secondary Hyperparathroidism (rugs: anti-epileptic drugs like ph D is Rare, and is seen only after pa a and hyperphophatemia. ent therapy in deficient individual	21 - 29 <u>30 - 100</u> > 100 localciferol (fror 3 in the skin upo ir and transport e in circulation. of calcium hom , calcium mobili newly formed c ity Mild to Modera enytoin, phenot prolonged exposed als must be mon	n plants. Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans eostatis. It promotes calciur zation, mainly regulated by osteoid in bone, resulting in r barbital and carbamazepine, sure to extremely high doses itored by periodic assessmer	g/mL g/mL g/mL g/mL port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH). rickets in children and osteomalacia in adults. that increases Vitamin D metabolism. of Vitamin D. When it occurs, it can result in nt of Vitamin D levels in order to prevent
DEFI INSUF PREFFER INTOX 1.Vitamin D compou conversion of 7- dihy 2.25-OHVitamin D n tissue and tightly bo 3.Vitamin D plays a p phosphate reabsorp 4.Severe deficiency n DECREASED: 1.Lack of sunshine ex 2.Inadeguate intake 3.Depressed Hepatic 4.Secondary to adva 5.Osteoporosis and S 6.Enzyme Inducing d INCREASED: 1. Hypervitaminosis Severe hypercalcemi CAUTION: Replaceme hypervitaminosis D	FICIENT: ED RANGE: ICATION: ICATION: Inds are derived from dietary ero vdrocholecalciferol to Vitamin D. represents the main body resevo und by a transport protein while primary role in the maintenance tion, skeletal calcium deposition may lead to failure to mineralize xposure. , malabsorption (celiac disease) : Vitamin D 25- hydroxylase activ nced Liver disease Secondary Hyperparathroidism (Irugs: anti-epileptic drugs like ph D is Rare, and is seen only after p a and hyperphophatemia. ent therapy in deficient individual <i>individuals as compare to whites</i> ,	21 - 29 <u>30 - 100</u> > 100 localciferol (fror 3 in the skin upo ir and transport e in circulation. of calcium hom , calcium mobili newly formed c ity Mild to Modera enytoin, phenot prolonged exposed als must be mon	n plants. Vitamin D2), or cho on Ultraviolet exposure. form of Vitamin D and trans eostatis. It promotes calciur zation, mainly regulated by osteoid in bone, resulting in r barbital and carbamazepine, sure to extremely high doses itored by periodic assessmer	g/mL g/mL g/mL lecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH). rickets in children and osteomalacia in adults. that increases Vitamin D metabolism. of Vitamin D. When it occurs, it can result in





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



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	Dr. Vinay Cho MD (Pathology & Chairman & Const	Microbiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. RAJESH KAPOOR			
AGE/ GENDER	: 70 YRS/MALE	PATIEN	T ID	: 1614321
COLLECTED BY	:	REG. NO	./LAB NO.	: 012409160017
REFERRED BY	:	REGIST	RATION DATE	: 16/Sep/2024 08:57 AM
BARCODE NO.	: 01517048		FION DATE	: 16/Sep/2024 09:02AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		TING DATE	: 16/Sep/2024 10:47AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A			
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PATHO	DLOGY	
		OUTINE & MICROSCO		
PHYSICAL EXAMINAT				
		10		
QUANTITY RECIEVED	ANCE SPECTROPHOTOMETRY	10	ml	
COLOUR		PALE YELLOW		PALE YELLOW
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY				
TRANSPARANCY	ANCE SPECTROPHOTOMETRY	HAZY		CLEAR
SPECIFIC GRAVITY	ANOL OF LOTHON HOTOMETRY	1.02		1.002 - 1.030
	ANCE SPECTROPHOTOMETRY			
CHEMICAL EXAMINA	TION			
REACTION		ACIDIC		
PROTEIN	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	ANCE SPECTROPHOTOMETRY	Negative		
SUGAR		Negative		NEGATIVE (-ve)
	ANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5
pH by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	<=0.0		5.0 - 7.5
BILIRUBIN		Negative		NEGATIVE (-ve)
•	TANCE SPECTROPHOTOMETRY	Negotivo		
NITRITE by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0
	ANCE SPECTROPHOTOMETRY	Nogotius		
KETONE BODIES by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
BLOOD		Negative		NEGATIVE (-ve)
-	ANCE SPECTROPHOTOMETRY			
ASCORBIC ACID	ANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)

MICROSCOPIC EXAMINATION



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

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







Dr. Vinay Chopra



Dr. Yugam Chopra

MD (Pathology) MD (Pathology & Microbiology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. RAJESH KAPOOR **AGE/ GENDER** : 70 YRS/MALE **PATIENT ID** :1614321 **COLLECTED BY** :012409160017 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 16/Sep/2024 08:57 AM **BARCODE NO.** :01517048 **COLLECTION DATE** :16/Sep/2024 09:02AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :16/Sep/2024 10:47AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** NEGATIVE (-ve) **RED BLOOD CELLS (RBCs)** /HPF 0 - 3 by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT PUS CELLS 3-4 /HPF 0 - 5 by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT EPITHELIAL CELLS 1-2 /HPF ABSENT by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT CRYSTALS NEGATIVE (-ve) NEGATIVE (-ve) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT NEGATIVE (-ve) NEGATIVE (-ve) CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT BACTERIA NEGATIVE (-ve) **NEGATIVE** (-ve) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

A FEW MUCOUS THREADS SEEN

OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT TRICHOMONAS VAGINALIS (PROTOZOA)

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

*** End Of Report ***

ABSENT



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

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NEGATIVE (-ve)

ABSENT