

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



KOS Diagnostic Lab (A Unit of KOS Healthcare)

	Dr. Vinay Cl MD (Pathology of Chairman & Col		ME	m Chopra D (Pathology) ht Pathologist
NAME AGE/ GENDER	: Mr. RAM JOHAR : 74 YRS/MALE		PATIENT ID	: 1812192
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012503310026
REFERRED BY	:		REGISTRATION DATE	: 31/Mar/2025 10:28 AM
BARCODE NO.	: 01528059		COLLECTION DATE	: 31/Mar/2025 10:55AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 31/Mar/2025 11:18AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	SWA	ASTHYA WE	LLNESS PANEL:	D
	CO	OMPLETE BLO	DOD COUNT (CBC)	
RED BLOOD CELL	S (RBCS) COUNT AND INI	DICES		
HAEMOGLOBIN (HB)	12.4	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT cusing, electrical impedenci	4.36	Million	3.50 - 5.00
PACKED CELL VOL		39.2 ^L	%	40.0 - 54.0
MEAN CORPUSCUL	AR VOLUME (MCV) TOMATED HEMATOLOGY ANALY2	89.7 ZER	fL	80.0 - 100.0
	AR HAEMOGLOBIN (MCH)	28.4 ZER	pg	27.0 - 34.0
by CALCULATED BY AU	AR HEMOGLOBIN CONC. (TOMATED HEMATOLOGY ANALYZ		g/dL	32.0 - 36.0
by CALCULATED BY AU	UTION WIDTH (RDW-CV) TOMATED HEMATOLOGY ANALYZ		%	11.00 - 16.00
by CALCULATED BY AU	UTION WIDTH (RDW-SD) TOMATED HEMATOLOGY ANALYZ		fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		20.57	RATIO	BETA THALASSEMIA TRAIT: 13.0 IRON DEFICIENCY ANEMIA:
				>13.0
GREEN & KING IND by Calculated	EX	99.93	RATIO	BETA THALASSEMIA TRAIT: <= 74.1
				IRON DEFICIENCY ANEMIA: >= 74.1
WHITE BLOOD CE	LLS (WBCS)			
	E COUNT (TLC) BY SF CUBE & MICROSCOPY	9640	/cmm	4000 - 11000
				0.00 20.00
NUCLEATED RED B	LOOD CELLS (nRBCS) HEMATOLOGY ANALYZER	NIL		0.00 - 20.00





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	Dr. Vinay Chop MD (Pathology & Mic Chairman & Consulta	crobiology)	Dr. Yugam MD CEO & Consultant	(Pathology)	
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by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER				
DIFFERENTIAL LI	<u>EUCOCYTE COUNT (DLC)</u>				
NEUTROPHILS by FLOW CYTOMETRY	/ BY SF CUBE & MICROSCOPY	57	%	50 - 70	
LYMPHOCYTES		30	%	20 - 40	
	Y BY SF CUBE & MICROSCOPY	_			
EOSINOPHILS	Y BY SF CUBE & MICROSCOPY	5	%	1 - 6	
MONOCYTES		8	%	2 - 12	
	Y BY SF CUBE & MICROSCOPY	0	70	2 12	
BASOPHILS		0	%	0 - 1	
	BY SF CUBE & MICROSCOPY				
ABSOLUTE LEUK	OCYTES (WBC) COUNT				
ABSOLUTE NEUTR		5495	/cmm	2000 - 7500	
	A BY SF CUBE & MICROSCOPY	2802	10000	800 4000	
ABSOLUTE LYMPH	/ BY SF CUBE & MICROSCOPY	2892	/cmm	800 - 4900	
ABSOLUTE EOSIN		482 ^H	/cmm	40 - 440	
by FLOW CYTOMETRY	BY SF CUBE & MICROSCOPY				
ABSOLUTE MONO		771	/cmm	80 - 880	
ABSOLUTE BASOP	Y BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110	
	Y BY SF CUBE & MICROSCOPY	0	/ciiiii	0 - 110	
ABSOLUTE IMMAT	TURE GRANULOCYTE COUNT	0	/cmm	0.0 - 999.0	
	OTHER PLATELET PREDICTIV	E MARKERS.			
PLATELET COUNT		288000	/cmm	150000 - 450000	
	OCUSING, ELECTRICAL IMPEDENCE	200000	/emm	130000 - 430000	
PLATELETCRIT (P		0.36	%	0.10 - 0.36	
	OCUSING, ELECTRICAL IMPEDENCE				
MEAN PLATELET	VOLUME (MPV)	12 ^H	fL	6.50 - 12.0	
•	CELL COUNT (P-LCC)	101000H	/cmm	30000 - 90000	
	OCUSING, ELECTRICAL IMPEDENCE	121000 ^H	/Cillin	50000 - 20000	
PLATELET LARGE	CELL RATIO (P-LCR)	41.8	%	11.0 - 45.0	



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Test Name		Value	Unit	Biological Reference interval
PLATELET DISTR	FOCUSING, ELECTRICAL IMPEDENCE IBUTION WIDTH (PDW) FOCUSING, ELECTRICAL IMPEDENCE	15.9	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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CLIENT CODE.	: KOS DIAGN	OSTIC LAB	R	EPORTING DATE	: 31/Mar/2025 11:39AM
CLIENT ADDRESS	: 6349/1, NI	CHOLSON ROAD, AMI	BALA CANTT		
Test Name			Value	Unit	Biological Reference interval
		ERYTHROC	YTE SEDIM	IENTATION RATE	(ESR)
mmune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythe CONDITION WITH LOV A low ESR can be see (polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactive 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ba	GATION BY CAPIL ic test because does not tell th cted by other c be used to more ematosus W ESR n with condition ificantly high v e cell anaemia e protein (C-RP es not change as by as many oth ed, it is typicall ve a higher ESR ran, methyldog	an elevated result off he health practitioner onditions besides influ- nitor disease activity a ns that inhibit the noi vhite blood cell count also lower the ESR. are both markers of s rapidly as does CRP, her factors as is ESR , my a result of two type: , and menstruation ar ba, oral contraceptive	exactly where ammation. For nd response to rmal sedimenta (leucocytosis) inflammation. either at the si aking it a bette s of proteins, g d pregnancy c	the inflammation is in the this reason, the ESR is typ therapy in both of the a ation of red blood cells, su , and some protein abno tart of inflammation or as ar marker of inflammation lobulins or fibrinogen. an cause temporary eleva	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 uch as a high red blood cell count rmalities. Some changes in red cell shape (such s it resolves. n .

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Test Name		Value	Unit	Biological Reference interval
	CLINI	CAL CHEMISTR	Y/BIOCHEMIS	STRY
			STINC (E)	
		GLUCOSE FA	51ING (F)	

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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 PRO	OFILE : BASIC	
CHOLESTEROL TC by CHOLESTEROL OX		174.63	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: 5 by GLYCEROL PHOSP	SERUM PHATE OXIDASE (ENZYMATIC)	123.71	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERC by SELECTIVE INHIBIT	DL (DIRECT): SERUM ion	54.36	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERC by CALCULATED, SPE		95.53	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLES by CALCULATED, SPE		120.27	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTER by CALCULATED, SPE	CTROPHOTOMETRY	24.74	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SE by CALCULATED, SPE	-	472.97	mg/dL	350.00 - 700.00
CHOLESTEROL/HI	DL RATIO: SERUM	3.21	RATIO	LOW RISK: 3.30 - 4.40

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Test Name		Value	Unit	Biological Reference interval
L				MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: S by CALCULATED, SPE		1.76	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	IDL RATIO: SERUM	2.28 ^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.

 Cow 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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Test Name		Value	Unit	Biological Reference interval
			ON TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI	.: SERUM PECTROPHOTOMETRY	0.63	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	T (CONJUGATED): SERUM SPECTROPHOTOMETRY	0.17	mg/dL	0.00 - 0.40
	ECT (UNCONJUGATED): SERUM	0.46	mg/dL	0.10 - 1.00
SGOT/AST: SERUN by IFCC, WITHOUT PY	M /RIDOXAL PHOSPHATE	26	U/L	7.00 - 45.00
SGPT/ALT: SERUN by IFCC, WITHOUT PY	1 (RIDOXAL PHOSPHATE	32.6	U/L	0.00 - 49.00
AST/ALT RATIO: S	SERUM ECTROPHOTOMETRY	0.8	RATIO	0.00 - 46.00
ALKALINE PHOSP. by PARA NITROPHEN PROPANOL	HATASE: SERUM	93.04	U/L	40.0 - 130.0
GAMMA GLUTAM by SZASZ, SPECTRON	IYL TRANSFERASE (GGT): SERUN phtometry	M 24.36	U/L	0.00 - 55.0
TOTAL PROTEINS by BIURET, SPECTRO		6.4	gm/dL	6.20 - 8.00
ALBUMIN: SERUM	Γ	4.1	gm/dL	3.50 - 5.50
by BROMOCRESOL G GLOBULIN: SERUN		2.3	gm/dL	2.30 - 3.50
	ECTROPHOTOMETRY		8	··· · · · · · · ·
A : G RATIO: SERU		1.78	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





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HEPATOCELLULAR C	ARCINOMA & CHRONIC HEPATITIS	> 1.3	Slightly Increas	sed)

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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	KIDNE	Y FUNCTIO	N TEST (COMPLET)	E)
UREA: SERUM		27.5	mg/dL	10.00 - 50.00
•	IATE DEHYDROGENASE (GLDH)			
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY		1.08	mg/dL	0.40 - 1.40
•	ROGEN (BUN): SERUM	12.85	mg/dL	7.0 - 25.0
by CALCULATED, SPECTROPHOTOMETRY			8	
	ROGEN (BUN)/CREATININE	11.9	RATIO	10.0 - 20.0
RATIO: SERUM by CALCULATED, SPE	ECTROPHOTOMETRY			
UREA/CREATININ		25.46	RATIO	
by CALCULATED, SPE				
URIC ACID: SERUN by URICASE - OXIDAS		2.43 ^L	mg/dL	3.60 - 7.70
CALCIUM: SERUM		8.46 ^L	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE	CTROPHOTOMETRY		-	
PHOSPHOROUS: SI	ERUM DATE, SPECTROPHOTOMETRY	3.31	mg/dL	2.30 - 4.70
ELECTROLYTES	DATE, SPECIROPHOTOMETRY			
SODIUM: SERUM		143.8	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV	/E ELECTRODE)	145.8	IIIII01/L	135.0 - 150.0
POTASSIUM: SERU		4.37	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV CHLORIDE: SERUN	,	107.95	mm_1/I	90.0 110.0
by ISE (ION SELECTIV		107.85	mmol/L	90.0 - 110.0
ESTIMATED GLO	MERULAR FILTERATION RAT	<u>re</u>		
ESTIMATED GLON	MERULAR FILTERATION RATE	E 72		
(eGFR): SERUM				
by CALCULATED				
INTERPRETATION:	een pre- and post renal azotemia.			

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.



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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	: Mr. RAM JO	: Mr. RAM JOHAR				
AGE/ GENDER	: 74 YRS/MAI	LE]	PATIENT ID	: 1812192	
COLLECTED BY	: SURJESH		,	REG. NO./LAB NO.	:0125033100)26
REFERRED BY				REGISTRATION DAT		
	·					
BARCODE NO.	:01528059			COLLECTION DATE	: 31/Mar/2025	
CLIENT CODE.	: KOS DIAGN			REPORTING DATE	: 31/Mar/2025	02:20PM
CLIENT ADDRESS	: 6349/1, NIC	CHOLSON ROAD,	AMBALA CANTT			
Test Name			Value	Unit	Biolo	gical Reference interval
burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<	ction plus ke or productic xia, high fever) (e.g. ureter col ass (subnorma tetracycline, gl 0:1) WITH ELEV (BUN rises dis superimposed 0:1) WITH DECI	ostomy) I creatinine produ ucocorticoids) ATED CREATININE proportionately m on renal disease.	iction)	n, GI bleeding, thyroto e) (e.g. obstructive ur		drome, high protein diet,
5. Impaired renal fun 6. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r	ction plus ke or productic xia, high fever) (e.g. ureter col ass (subnorma tetracycline, gl 0:1) WITH ELEV (BUN rises dis superimposed 0:1) WITH DECI osis. d starvation. e. creased urea sy urea rather tha monemias (urea of inappropiate 0:1) WITH INCE py (accelerates eleases muscle	ostomy) I creatinine produ ucocorticoids) ATED CREATININE proportionately m on renal disease. REASED BUN : an creatinine diffu antidiuretic harm REASED CREATININ conversion of creatinine).	uction) E LEVELS: hore than creatinin uses out of extrace ent in blood). hone) due to tubula	e) (e.g. obstructive ur Ilular fluid). ar secretion of urea.		drome, high protein diet,
5. Impaired renal fun 6. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO	ction plus ke or production xia, high fever) (e.g. ureter col ass (subnormation tetracycline, gl 0:1) WITH ELEV (BUN rises dis superimposed 0:1) WITH DECI osis. d starvation. creased urea sy urea rather that monemias (urea of inappropiate 0:1) WITH INCE py (accelerates eleases muscle who develop recently a starvation creased urea sy urea rather that a starvation of inappropiate	ostomy) I creatinine produ ucocorticoids) ATED CREATININE proportionately m on renal disease. REASED BUN : an creatinine diffu a is virtually abse antidiuretic harm REASED CREATININ conversion of creatinine). enal failure.	uction) E LEVELS: hore than creatinin uses out of extrace ent in blood). hone) due to tubula IE: eatine to creatinin	e) (e.g. obstructive ur Ilular fluid). ar secretion of urea.	opathy).	
5. Impaired renal fun 6. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO	ction plus ke or production xia, high fever) (e.g. ureter col ass (subnormal tetracycline, gl 0:1) WITH ELEV (BUN rises disp superimposed 0:1) WITH DECI posis. Ind starvation. A starva	ostomy) I creatinine produ ucocorticoids) ATED CREATININE proportionately m on renal disease. REASED BUN : an creatinine diffu a is virtually abse antidiuretic harm REASED CREATININ conversion of creatinine). enal failure. te causes false ind reatinine ratio). with creatinine m	action) E LEVELS: hore than creatinin uses out of extrace ent in blood). hone) due to tubula IE: eatine to creatinin crease in creatinin	e) (e.g. obstructive ur Ilular fluid). ar secretion of urea.	opathy).	drome, high protein diet, ormal ratio when dehydratio

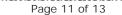
CKD STAGE	DESCRIPTION	GFR (mL/min/1.73m2)	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with	>90	Presence of Protein,
	normal or high GFR		Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	



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NAME	: Mr. RAM JOHAR		
AGE/ GENDER	: 74 YRS/MALE	PATIENT ID	: 1812192
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012503310026
REFERRED BY	:	REGISTRATION DATE	: 31/Mar/2025 10:28 AM
BARCODE NO.	: 01528059	COLLECTION DATE	: 31/Mar/2025 10:55AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 31/Mar/2025 02:20PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CAN	TT	
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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CLIENT CODE.	: KOS DIAGNOSTIC I	LAB	REPORTING DATE	: 31/Mar/2025 12:27PM
CLIENT ADDRESS	: 6349/1, NICHOLSO	ON ROAD, AMBALA CANT	TT	
Test Name		Value	Unit	Biological Reference interval
		VI	TAMINS	
		VITAMIN D/25 H	IYDROXY VITAMIN I)3
VITAMIN D (25-HYDROXY VITAMIN D3): SERUM		O3): SERUM 76.8	ng/mL	DEFICIENCY: < 20.0
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)				INSUFFICIENCY: 20.0 - 30.0
				SUFFICIENCY: 30.0 - 100.0
				TOXICITY: > 100.0
INTERPRETATION:				
DEFI	CIENT:	< 20	n	g/mL
INSUF	FICIENT:	21 - 29	n	g/mL

DEFICIENT:	< 20	ng/mL	
INSUFFICIENT:	21 - 29	ng/mL	
PREFFERED RANGE:	30 - 100	ng/mL	
INTOXICATION:	> 100	ng/mL	I

KOS Diagnostic Lab (A Unit of KOS Healthcare)

1. Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.

2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.

3. Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH). 4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults. DECREASED:

1.Lack of sunshine exposure.

2.Inadequate intake, malabsorption (celiac disease) 3.Depressed Hepatic Vitamin D 25- hydroxylase activity

4. Secondary to advanced Liver disease

5. Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)

6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED: 1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.

*** End Of Report ***



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