

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
AME	: Mrs. DALJIT KAUR			
GE/ GENDER	: 38 YRS/FEMALE	F	PATIENT ID	: 1684532
COLLECTED BY	:	F	REG. NO./LAB NO.	: 012411280008
REFERRED BY	:	F	REGISTRATION DATE	: 28/Nov/2024 09:18 AM
SARCODE NO.	: 01521581		COLLECTION DATE	: 28/Nov/2024 09:24AM
LIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 28/Nov/2024 10:17AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
	SW/A ST	HVA WFI	LNESS PANEL: 1.0	
			OD COUNT (CBC)	
PED BLOOD CELL	S (RBCS) COUNT AND INDICES	LETE DLU		
HAEMOGLOBIN (H		11 ^L	gm/dL	12.0 - 16.0
by CALORIMETRIC			U U	
RED BLOOD CELL ((RBC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	6.24 ^H	Millions/	cmm 3.50 - 5.00
PACKED CELL VOL	UME (PCV) AUTOMATED HEMATOLOGY ANALYZER	36.1 ^L	%	37.0 - 50.0
•	AR VOLUME (MCV)	58 ^L	fL	80.0 - 100.0
	AUTOMATED HEMATOLOGY ANALYZER AR HAEMOGLOBIN (MCH)		pď	27.0 - 34.0
	AUTOMATED HEMATOLOGY ANALYZER	17.6 ^L	pg	27.0 - 34.0
	AR HEMOGLOBIN CONC. (MCHC)	30.3 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIB	UTION WIDTH (RDW-CV)	18.1 ^H	%	11.00 - 16.00
	AUTOMATED HEMATOLOGY ANALYZER UTION WIDTH (RDW-SD)	39.3	fL	35.0 - 56.0
by CALCULATED BY A	AUTOMATED HEMATOLOGY ANALYZER			
MENTZERS INDEX		9.29	RATIO	BETA THALASSEMIA TRAIT: < 13.0
				IRON DEFICIENCY ANEMIA:
GREEN & KING INI)FX	16.8	RATIO	>13.0 BETA THALASSEMIA TRAIT:<
by CALCULATED		10.0	in the second se	65.0
				IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE	LLS (WBCS)			00.0
	E COUNT (TLC)	10210	/cmm	4000 - 11000
TOTAL LEUCOCYTI				
FOTAL LEUCOCYTI	Y BY SF CUBE & MICROSCOPY SLOOD CELLS (nRBCS)	NII.		(0.00 - 20.00)
TOTAL LEUCOCYTI by FLOW CYTOMETR NUCLEATED RED F by AUTOMATED 6 PAI	y by sf cube & microscopy BLOOD CELLS (nRBCS) rt hematology analyzer BLOOD CELLS (nRBCS) %	NIL NIL	%	0.00 - 20.00 < 10 %





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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	66	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	27	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LEUKOCYTES (WBC) COUNT	0	%	0 - 1
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6739	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by flow cytometry by sf cube & microscopy	2757	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by flow cytometry by sf cube & microscopy	102	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	613	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRANULOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	102	/cmm	0.0 - 999.0
PLATELETS AND OTHER PLATELET PREDICTIV	<u>E MARKERS.</u>		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	248000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.31	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	13 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	135000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by Hydro Dynamic Focusing, electrical impedence	54.5 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence	15	%	15.0 - 17.0



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Test Name	Value	Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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LIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 28/Nov/2024 10:39AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT		
'est Name		Value	Unit	Biological Reference interval
mune disease, but An ESR can be affe C-reactive protein This test may also stemic lupus eryth NDITION WITH LO ow ESR can be see olycythaemia), sig	does not tell the health practitione ected by other conditions besides inf be used to monitor disease activity ematosus W ESR In with conditions that inhibit the no	r exactly when flammation. F and response ormal sediment (leucocytos)	re the inflammation is in th or this reason, the ESR is ty to therapy in both of the a ntation of red blood cells, s	pically used in conjunction with other test such bove diseases as well as some others, such as
DTE: ESR and C - reactiv Generally, ESR doo CRP is not affected If the ESR is elevat Women tend to ha Drugs such as dex	e protein (C-RP) are both markers o sonot change as rapidly as does CRP l by as many other factors as is ESR, i ed, it is typically a result of two type we a higher ESR, and menstruation a tran, methyldopa, oral contraceptiv	f inflammation P, either at the making it a be es of proteins and pregnancy	e start of inflammation or a tter marker of inflammatio , globulins or fibrinogen. / can cause temporary elev	n.
spirin, cortisone, ar	id quinine may decrease it			





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BARCODE NO.	:01521581			COLLECTION DATE	: 28/Nov/2024 09:24AM
CLIENT CODE.	: KOS DIAGNOS	STIC LAB		REPORTING DATE	: 28/Nov/2024 11:50AM
CLIENT ADDRESS	: 6349/1, NICH	IOLSON ROAD,	AMBALA CANTT		
Test Name			Value	Unit	Biological Reference interval
		CLINI	CAL CHEMIS	FRY/BIOCHEMIST	'RY
			GLUCOSE	FASTING (F)	
GLUCOSE FASTING	G (F): PLASMA	OD-POD)	222.39 ^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

 A fasting plasma glucose level below 100 mg/dl is considered normal.
 A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.

test (after consumption of 75 gms of glucose) is recommended for all such patients. 3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.





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Test Name		Value	Unit	Biological Reference interval
			II E . DASIC	
	TAL CEDUM	LIPID PROF		OPTIMAL: < 200.0
CHOLESTEROL TO by CHOLESTEROL O		170.89	mg/dL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
FRIGLYCERIDES: S		342.45 ^H	mg/dL	240.0 OPTIMAL: < 150.0
by GLYCEROL PHOSE	PHATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
UDI CHOLESTEDO	L (DIRECT): SERUM	34.4	mg/dL	VERY HIGH: > OR = 500.0 LOW HDL: < 30.0
by SELECTIVE INHIBIT		34.4	ing/uL	BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERO by CALCULATED, SPE		68	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129. BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0
NON HDL CHOLES' by calculated, spe	TEROL: SERUM ECTROPHOTOMETRY	136.49 ^H	mg/dL	VERY HIGH: > OR = 190.0 OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTER		68.49 ^H	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEF		684.23	mg/dL	350.00 - 700.00
CHOLESTEROL/HI	ECTROPHOTOMETRY DL RATIO: SERUM ECTROPHOTOMETRY	4.97 ^H	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		1.98	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	9.95 ^H	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.

3. 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. 4. 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
	LIVER	FUNCTION '	TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, S.	: SERUM PECTROPHOTOMETRY	1.41 ^H	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	Г (CONJUGATED): SERUM spectrophotometry	0.25	mg/dL	0.00 - 0.40
	ECT (UNCONJUGATED): SERUM	1.16 ^H	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	I (RIDOXAL PHOSPHATE	17.1	U/L	7.00 - 45.00
SGPT/ALT: SERUM		17.8	U/L	0.00 - 49.00
AST/ALT RATIO: S	ERUM ECTROPHOTOMETRY	0.96	RATIO	0.00 - 46.00
ALKALINE PHOSP		100.24	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM	19.77	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		7.29	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		4.4	gm/dL	3.50 - 5.50

by CALCULATED, SPECTROPHOTOMETRY A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY

by BROMOCRESOL GREEN

GLOBULIN: SERUM

INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:

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

2.89

1.52





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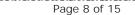
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gm/dL

RATIO

2.30 - 3.50

1.00 - 2.00







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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).

PROGNOSTIC SIGNIFICANCE:

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



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	KIDN	EY FUNCTIO	N TEST (COMPLETE)		
UREA: SERUM		25.92	mg/dL	10.00 - 50.00	
by UREASE - GLUTAMATE DEHYDROGENASE (GLDH) CREATININE: SERUM		0.78	mg/dL	0.40 - 1.20	
by ENZYMATIC, SPECTROPHOTOMETERY			-		
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY		12.11	mg/dL	7.0 - 25.0	
	ROGEN (BUN)/CREATININE	15.53	RATIO	10.0 - 20.0	
RATIO: SERUM					
by CALCULATED, SPE UREA/CREATININ		33.23	RATIO		
by CALCULATED, SPE	ECTROPHOTOMETRY				
URIC ACID: SERUM by URICASE - OXIDAS		3.93	mg/dL	2.50 - 6.80	
CALCIUM: SERUM		10.32	mg/dL	8.50 - 10.60	
by ARSENAZO III, SPE PHOSPHOROUS: SE		3.45	mg/dI	2.30 - 4.70	
	DATE, SPECTROPHOTOMETRY	3.45	mg/dL	2.30 - 4.70	
ELECTROLYTES					
SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)		137.9	mmol/L	135.0 - 150.0	
POTASSIUM: SERU		4	mmol/L	3.50 - 5.00	
by ISE (ION SELECTIV	/E ELECTRODE)				
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)		103.43	mmol/L	90.0 - 110.0	
	IERULAR FILTERATION RATE				
	ERULAR FILTERATION RATE	99.6			

INTERPRETATION:

To differentiate between pre- and post renal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE: 1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased

glomerular filtration rate.

2. Catabolic states with increased tissue breakdown.

3. GI haemorrhage.



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Test Name			Value	Uni	it	Biolog	ical Refere	ence interv
9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<'	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR	TED CREATININE LEVE roportionately more to n renal disease.) (e.g. obstructive	e uropathy).			
 P. Certain drugs (e.g., INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<' 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 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 1. Diabetic ketoacido should produce an in 2. Cephalosporin there ESTIMATED GLOMERI G1 G2 	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. 2. creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/crea apy (interferes v ILAR FILTERATIO Nor Kin Nor	cocorticoids) TED CREATININE LEVE roportionately more to n renal disease. EASED BUN : Athesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creatine creatinine). hal failure. a causes false increase extinine ratio). with creatinine measu <u>N RATE:</u> <u>DESCRIPTION</u> mal kidney function dney damage with prmal or high GFR	han creatinine but of extracell blood). due to tubular e to creatinine) e in creatinine rement). GFR (mL/	ular fluid). secretion of urea with certain meth <u>(min/1.73m2)</u> >90 >90	hodologies,r ASSOCIA No p Presenc	esulting in no TED FINDINGS roteinuria e of Protein , or cast in urin		vhen dehydr
 2. Certain drugs (e.g., NCREASED RATIO (>2 I. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<' I. Acute tubular necr 2. Low protein diet and 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (r 6. Inherited hyperam 7. SIADH (syndrome of 3. Pregnancy. DECREASED RATIO (<' 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients NAPPROPIATE RATIO 4. Cephalosporin there ESTIMATED GLOMERI G1 G2 G3a G3b 	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. 2. creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/crea apy (interferes v UAR FILTERATIO Nor Kin Mor	cocorticoids) TED CREATININE LEVE roportionately more to n renal disease. EASED BUN : Athesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creatine treatinine). hal failure. a causes false increase extinine ratio). vith creatinine measu V RATE: DESCRIPTION mal kidney function dney damage with	han creatinine but of extracell blood). due to tubular e to creatinine) e in creatinine rement). GFR (mL/	ular fluid). secretion of urea with certain meth <u>(min/1.73m2)</u> >90 >90 >90 0 -89 30-59	hodologies,r ASSOCIA No p Presenc	TED FINDINGS roteinuria e of Protein ,		vhen dehydr
 P. Certain drugs (e.g., INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<' 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 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 1. Diabetic ketoacido should produce an in 2. Cephalosporin there ESTIMATED GLOMERI G1 G2 	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 0:1) WITH DECR osis. Id starvation. 2. creased urea syn urea rather thar monemias (urea f inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop ren- sis (acetoacetate creased BUN/creased apy (interferes with LAR FILTERATION Nor Kin Model	cocorticoids) TED CREATININE LEVE roportionately more to n renal disease. EASED BUN : The thesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) CASED CREATININE: conversion of creatine treatinine). hal failure. the causes false increase extinine ratio). with creatinine measure NATE: DESCRIPTION mal kidney function dney damage with ormal or high GFR Id decrease in GFR	han creatinine but of extracell blood). due to tubular e to creatinine) e in creatinine rement). GFR (mL/	ular fluid). secretion of urea with certain meth <u>(min/1.73m2)</u> >90 >90	hodologies,r ASSOCIA No p Presenc	TED FINDINGS roteinuria e of Protein ,		vhen dehydr



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









	Dr. Vinay Chopra MD (Pathology & Microbio Chairman & Consultant Pa		(Pathology)
NAME	: Mrs. DALJIT KAUR		
AGE/ GENDER	: 38 YRS/FEMALE	PATIENT ID	: 1684532
COLLECTED BY	:	REG. NO./LAB NO.	: 012411280008
REFERRED BY	:	REGISTRATION DATE	: 28/Nov/2024 09:18 AM
BARCODE NO.	: 01521581	COLLECTION DATE	: 28/Nov/2024 09:24AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 28/Nov/2024 11:50AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA	CANTT	
Test Name	Val	lue 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

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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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MBBS, MD (PATHOLOGY)







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NAME	: Mrs. DALJIT KAUR				
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Test Name		Value	Unit	Biological Reference interval	
INTERPRETATION:-	IESCENT MICROPARTICLE IMMUNOA	ASSAY)			
	SED VITAMIN B12	1 Drognanov	DECREASED VITAMI	N B12	
1.Ingestion of Vitam 2.Ingestion of Estroy		1.Pregnancy	pirin, Anti-convulsants	Colchicipe	
3.Ingestion of Vitam		3.Ethanol Ig			
4.Hepatocellular injury			4. Contraceptive Harmones		
5.Myeloproliferativ	e disorder		5.Haemodialysis		
6.Uremia			6. Multiple Myeloma		
2.In humans, it is obt 3.The body uses its v excreted. 4.Vitamin B12 deficie ileal resection, small	ency may be due to lack of IF sec intestinal diseases).	is and requires intrins cally, reabsorbing vita cretion by gastric muce	ic factor (IF) for absor min B12 from the ileur osa (eg, gastrectomy, ç	n and returning it to the liver; very little is gastric atrophy) or intestinal malabsorption (eg.	
proprioception, poor the neurologic defect 6.Serum methylmalo	coordination, and affective beh s without macrocytic anemia. nic acid and homocysteine level	navioral changes. Thes is are also elevated in	e manifestations may vitamin B12 deficiency	weakness, hyperreflexia, ataxia, loss of occur in any combination; many patients have y states. al cause of vitamin B12 malabsorption	

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7. Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption. **NOTE:**A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.





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	Dr. Vinay Cho MD (Pathology & M Chairman & Consu	licrobiology)	Dr. Yugam MD D & Consultant	(Pathology)
NAME	: Mrs. DALJIT KAUR			
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Test Name		Value	Unit	Biological Reference interval
		CLINICAL PATHOI TINE & MICROSCOP		ATION
PHYSICAL EXAMIN	ATION			
QUANTITY RECIEVE		10	ml	
by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	AMBER YELLOW		PALE YELLOW
	ANCE SPECTROPHOTOMETRY			
TRANSPARANCY	ANCE SPECTROPHOTOMETRY	HAZY		CLEAR
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY SPECIFIC GRAVITY		<=1.005		1.002 - 1.030
	ANCE SPECTROPHOTOMETRY			
<u>CHEMICAL EXAMIN</u> REACTION	NATION	ACIDIC		
	ANCE SPECTROPHOTOMETRY	ACIDIC		
PROTEIN	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR	ANCE SI LOTION HOTOMETRI	3+		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5
pH by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	<=3.0		5.0 - 7.5
BILIRUBIN	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
NITRITE	ANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN	ANCE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0
KETONE BODIES		Negative		NEGATIVE (-ve)
by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	Nogativo		NEGATIVE (-ve)
DLOOD	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-VC)
ASCORBIC ACID	ANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
MICROSCOPIC EXA				
RED BLOOD CELLS		NEGATIVE (-ve)	/HPF	0 - 3





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Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist

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

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Test Name		Value	Unit	Biological Reference interval
PUS CELLS by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT	1-2	/HPF	0 - 5
EPITHELIAL CELLS	S CENTRIFUGED URINARY SEDIMENT	2-4	/HPF	ABSENT

by MICROSCOLL ON CENTRI OCED ORMART SEDIMENT		
CRYSTALS	NEGATIVE (-ve)	NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
CASTS	NEGATIVE (-ve)	NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
BACTERIA	NEGATIVE (-ve)	NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
OTHERS	NEGATIVE (-ve)	NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		
TRICHOMONAS VAGINALIS (PROTOZOA)	ABSENT	ABSENT

TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

*** End Of Report ***



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