



Dr. Vinay Chop MD (Pathology & Mic Chairman & Consulta	crobiology)	Dr. Yugam MD (I CEO & Consultant F	Pathology)
NAME : Mrs. PAMALJEET BHATIA			
AGE/ GENDER : 43 YRS/FEMALE	PAT	FIENT ID	: 1546476
COLLECTED BY :	REG	G. NO./LAB NO.	: 012407120031
REFERRED BY :	REG	GISTRATION DATE	: 12/Jul/2024 10:32 AM
BARCODE NO. : 01512979	COL	LLECTION DATE	: 12/Jul/2024 10:34AM
CLIENT CODE. : KOS DIAGNOSTIC LAB	REI	PORTING DATE	: 12/Jul/2024 11:18AM
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMI	BALA CANTT		
Test Name	Value	Unit	Biological Reference interval
SWAS	THYA WELLN	NESS PANEL: 1.2	
COL	MPLETE BLOOD	D COUNT (CBC)	
RED BLOOD CELLS (RBCS) COUNT AND INDICES	510 01	(
HAEMOGLOBIN (HB)	12.3	gm/dL	12.0 - 16.0
by CALORIMETRIC RED BLOOD CELL (RBC) COUNT	4.27	Millions/cm	nm 3.50 - 5.00
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PACKED CELL VOLUME (PCV)	38.6	%	37.0 - 50.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER MEAN CORPUSCULAR VOLUME (MCV)	90.3	fL	80.0 - 100.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER MEAN CORPUSCULAR HAEMOGLOBIN (MCH)	28.8	pg	27.0 - 34.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	31.8 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	14.7	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	49.5	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	21.15	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX	31.08	RATIO	BETA THALASSEMIA TRAIT: < =
by CALCULATED			65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	7570	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by calculated by automated hematology analyzer & microscopy	NIL	%	< 10 %

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Vinay Chopra Dr. MD (Pathology & Microbiology) Chairman & Consultant Pathologist CEO & Co LJEET BHATIA

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

NAME	: Mrs. PAMALJEET BHATIA		
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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	65	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	27	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	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	4921	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2044	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	303	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	303	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKER	<u>RS.</u>		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	194000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.29	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	15 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	118000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	61.2 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.1	%	15.0 - 17.0



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Test Name		Value Unit	Biological Reference interval







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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 12/Jul/2024 11:30AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	FRVTHR	OCYTE SEDIMENT	ΔΤΙΩΝ ΡΔΤΕ (FSE	2)
by MODIFIED WESTER INTERPRETATION: 1. ESR is a non-specifi mmune disease, but of 2. An ESR can be affect as C-reactive protein 3. This test may also be systemic lupus erythe CONDITION WITH LOV A low ESR can be seer (polycythaemia), sign as sickle cells in sickle NOTE: 1. ESR and C - reactive 2. Generally, ESR does 3. CRP is not affected 4. If the ESR is elevated 5. Women tend to hav 5. Drugs such as dextri	does not tell the health practitione ted by other conditions besides in be used to monitor disease activity matosus V ESR n with conditions that inhibit the n ificantly high white blood cell cour e cell anaemia) also lower the ESR e protein (C-RP) are both markers o is not change as rapidly as does CRF by as many other factors as is ESR, id, it is typically a result of two typ re a higher ESR. and menstruation a	er exactly where the ir flammation. For this r and response to ther ormal sedimentation nt (leucocytosis), and f inflammation. P, either at the start o making it a better ma es of proteins, globul and pregnancy can ca	flammation is in the eason, the ESR is typ apy in both of the all of red blood cells, su some protein abnor f inflammation or as rker of inflammation use temporary eleva	on associated with infection, cancer and auto- body or what is causing it. bically used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count malities. Some changes in red cell shape (such it resolves.





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CLIENT CODE.	: KOS DIAGNOSTIC LAB	RI	EPORTING DATE	: 12/Jul/2024 11:55AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
Test Name	CLIN			
Test Name	CLIN		RY/BIOCHEMISTR	

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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AGE/ GENDER: 43 YFCOLLECTED BY:REFERRED BY:BARCODE NO.: 0151	PAMALJEET BHATIA RS/FEMALE	PATI		
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REFERRED BY : BARCODE NO. : 0151			ENT ID	: 1546476
BARCODE NO. : 0151		REG.	NO./LAB NO.	: 012407120031
		REGI	STRATION DATE	: 12/Jul/2024 10:32 AM
CLIENT CODE	2979	COLI	LECTION DATE	: 12/Jul/2024 10:34AM
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CLIENT ADDRESS : 6349	/1, NICHOLSON ROAD), AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFILE	: BASIC	
CHOLESTEROL TOTAL: SERU by CHOLESTEROL OXIDASE P.		219.54 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE ON	(IDASE (ENZYMATIC)	174.97 ^H	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT) by SELECTIVE INHIBITION	: SERUM	49.07	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by calculated, spectroph	OTOMETRY	135.48 ^H	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 CHOLESTEROL: SE by CALCULATED, SPECTROPH		170.47 ^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM		34.99	mg/dL	0.00 - 45.00
by CALCULATED, SPECTROPH TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPH		614.05	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: S by CALCULATED, SPECTROPH	SERUM	4.47 ^H	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: SERUM by CALCULATED, SPECTROPH	OTOMETRY	2.76	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





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NAME	: Mrs. PAMALJEET BHATIA			
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Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HDL by CALCULATED, SPE		3.57	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 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
LIV	ER FUNCTION TE	EST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.76	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.31	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.45	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	34.47	U/L	7.00 - 45.00
SGPT/ALT: SERUM	52.3 ^H	U/L	0.00 - 49.00
by IFCC, WITHOUT PYRIDOXAL PHOSPHATE AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.66	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	132	U/L	40.0 - 150.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	32	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.63	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.7	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.93	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.6	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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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
1	



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NAME

BARCODE NO.

CLIENT CODE.

Test Name

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Dr. Yugam Chopra

CEO & Consultant Pathologist

MD (Pathology)

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Dr. Vinay Chopra

MD (Pathology & Microbiology)

Chairman & Consultant Pathologist

KIE	ONEY FUNCTION T	EST (COMPLETE)		
UREA: SERUM by urease - glutamate dehydrogenase (gldh)	28.33	mg/dL	10.00 - 50.00	
CREATININE: SERUM by enzymatic, spectrophotometery	0.69	mg/dL	0.40 - 1.20	
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	13.24	mg/dL	7.0 - 25.0	
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by Calculated, spectrophotometry	19.19	RATIO	10.0 - 20.0	
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	41.06	RATIO		
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	5.6	mg/dL	2.50 - 6.80	
CALCIUM: SERUM by Arsenazo III, spectrophotometry	9.2	mg/dL	8.50 - 10.60	
PHOSPHOROUS: SERUM by phosphomolybdate, spectrophotometry ELECTROLYTES	3.18	mg/dL	2.30 - 4.70	
SODIUM: SERUM	145.2	mmol/L	135.0 - 150.0	
POTASSIUM: SERUM by ise (ion selective electrode)	4.16	mmol/L	3.50 - 5.00	
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	108.9	mmol/L	90.0 - 110.0	
ESTIMATED GLOMERULAR FILTERATION RATE				
ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM	110.4			

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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NAME	: Mrs. PAMALJEET BHATIA			
AGE/ GENDER	: 43 YRS/FEMALE	PATIENT ID	: 1546476	3
COLLECTED BY	:	REG. NO./LAB NO). : 012407	7120031
	•			
REFERRED BY	:	REGISTRATION		2024 10:32 AM
BARCODE NO.	: 01512979	COLLECTION DA	FE : 12/Jul/2	2024 10:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DAT	: 12/Jul/2	2024 11:55AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
Test Name		Value U	nit	Biological Reference interval
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia				g's syndrome, high protein diet,
7. Urine reabsorption 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	(e.g. ureter colostomy) ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATININ py (accelerates conversion of crea eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false ind creased BUN/creatinine ratio). apy (interferes with creatinine m JLAR FILTERATION RATE:	LEVELS: ore than creatinine) (e.g. obstructive ses out of extracellular fluid). nt in blood). one) due to tubular secretion of ure E: atine to creatinine). crease in creatinine with certain me easurement).	re uropathy). ra.	ıg in normal ratio when dehydra
7. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. NCREASED 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 NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther	(e.g. ureter colostomy) ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATININ py (accelerates conversion of cree eleases muscle creatinine). who develop renal failure. 1: sis (acetoacetate causes false ind creased BUN/creatinine ratio). apy (interferes with creatinine m	LEVELS: ore than creatinine) (e.g. obstructive ses out of extracellular fluid). nt in blood). one) due to tubular secretion of ure E: atine to creatinine). crease in creatinine with certain me easurement).	re uropathy). ra.	ng in normal ratio when dehydra
7. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. NCREASED 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 of 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r 8. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther STIMATED GLOMERL OKD STAGE	(e.g. ureter colostomy) ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. d starvation. e. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse of inappropiate antidiuretic harm (0:1) WITH INCREASED CREATININ py (accelerates conversion of cree eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false ind creased BUN/creatinine ratio). apy (interferes with creatinine m ULAR FILTERATION RATE: DESCRIPTION Normal kidney funct Kidney damage wit	LEVELS: ore than creatinine) (e.g. obstructive ses out of extracellular fluid). nt in blood). one) due to tubular secretion of ure E: atine to creatinine). crease in creatinine with certain me easurement).	re uropathy). ea. ethodologies,resultin ASSOCIATED FII No proteinu Presence of Pr	ng in normal ratio when dehydra NDINGS uria otein ,
Y. Urine reabsorption Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome c Rhabdomyolysis (r Rhabdomyolysis (r Rhabdomyolysis (r Rhabdomyolysis (r Diabetic ketoacido hould produce an in CENTATED GLOMERL CKD STAGE G1 G2	(e.g. ureter colostomy) ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. d starvation. e. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse of inappropiate antidiuretic harm (0:1) WITH INCREASED CREATININ py (accelerates conversion of cree eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false ind creased BUN/creatinine ratio). apy (interferes with creatinine m ULAR FILTERATION RATE: DESCRIPTION Normal kidney funct Kidney damage wit normal or high GF	LEVELS: ore than creatinine) (e.g. obstructive ses out of extracellular fluid). nt in blood). one) due to tubular secretion of ure E: atine to creatinine). crease in creatinine with certain me easurement). ion >90 h >90 R >90	re uropathy). ea. ethodologies,resultin	ng in normal ratio when dehydra NDINGS uria otein ,
7. Urine reabsorption 3. Reduced muscle m 4. 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 3. 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 G1 G2 G3a	(e.g. ureter colostomy) ass (subnormal creatinine produ tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. d starvation. e. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually abse of inappropiate antidiuretic harm (0:1) WITH INCREASED CREATININ py (accelerates conversion of cree eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false ind creased BUN/creatinine ratio). apy (interferes with creatinine m UCR FILTERATION RATE: DESCRIPTION Normal kidney funct Kidney damage wit normal or high GF Mild decrease in Gi	LEVELS: ore than creatinine) (e.g. obstructive ses out of extracellular fluid). nt in blood). one) due to tubular secretion of ure E: atine to creatinine). crease in creatinine with certain me easurement). ion >90 h >90 R 60 - 89	re uropathy). ea. ethodologies,resultin ASSOCIATED FII No proteinu Presence of Pr	ng in normal ratio when dehydra NDINGS uria otein ,
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DR.VINAY CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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









	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant	biology) MI	m Chopra D (Pathology) ht Pathologist
NAME	: Mrs. PAMALJEET BHATIA		
AGE/ GENDER	: 43 YRS/FEMALE	PATIENT ID	: 1546476
COLLECTED BY	:	REG. NO./LAB NO.	: 012407120031
REFERRED BY	:	REGISTRATION DATE	: 12/Jul/2024 10:32 AM
BARCODE NO.	:01512979	COLLECTION DATE	: 12/Jul/2024 10:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 12/Jul/2024 11:55AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	LA CANTT	
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

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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)

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







	Dr. Vinay Ch MD (Pathology & Chairman & Con		Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. PAMALJEET BHATIA			
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BARCODE NO.	: 01512979	COL	LECTION DATE	: 12/Jul/2024 10:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REP	ORTING DATE	: 12/Jul/2024 12:08PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		ENDOCRIN	OLOGY	
		ENDOCRIN HYROID FUNCTIO		
	e (T3): Serum	HYROID FUNCTIO 0.794		0.35 - 1.93
THYROXINE (T4): SE	E (T3): SERUM NESCENT MICROPARTICLE IMMUNOA	0.794 0.899 0.89	N TEST: TOTAL	0.35 - 1.93 4.87 - 12.60

overproduction(hyperthyroidism) of T4 and/or T3. CLINICAL CONDITION T3 T4 TSH Primary Hypothyroidism Reduced Reduced Increased (Significantly) Subclinical Hypothyroidism: Normal or Low Normal Normal or Low Normal High

LIMITATIONS:-
LINITATIONS.

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

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.

Increased

Normal or High Normal

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 levies 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 STIMUL	ATING 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

Increased

Normal or High Normal





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Reduced (at times undetectable)

Reduced





	Dr. Vinay Chopra MD (Pathology & Microbi Chairman & Consultant P	G, /	(Pathology)
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Test Name			Value	Unit		Biological 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	
	RECOM	MENDATIONS OF TSH LE	EVELS DURING PREG	NANCY (µIU/mL)		
	1st Trimester			0.10 - 2.50		
	2nd Trimester		0.20 - 3.00			
	3rd Trimester			0.30 - 4.10		

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

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



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A			
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PA	THOLOGY	
		OUTINE & MICRO	SCOPIC EXAMINAT	ΓΙΟΝ
PHYSICAL EXAMINAT				
QUANTITY RECIEVED		10	ml	
	, TANCE SPECTROPHOTOMETRY	10		
COLOUR		PALE YELLOW		PALE YELLOW
-	TANCE SPECTROPHOTOMETRY			
TRANSPARANCY	TANCE SPECTROPHOTOMETRY	CLEAR		CLEAR
SPECIFIC GRAVITY		1.02		1.002 - 1.030
	TANCE SPECTROPHOTOMETRY			
CHEMICAL EXAMINA	TION			
REACTION		ACIDIC		
by DIP STICK/REFLECT PROTEIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR		Negative		NEGATIVE (-ve)
•	TANCE SPECTROPHOTOMETRY	5.0		
pH	TANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5
BILIRUBIN		Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
NITRITE	TANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN	TANGE OF LOTINOT HOTOMETRY.	Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
KETONE BODIES		Negative		NEGATIVE (-ve)
BLOOD	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY	Negative		
ASCORBIC ACID		NEGATIVE (-ve	2)	NEGATIVE (-ve)
by DIP STICK/REFLECT	TANCE SPECTROPHOTOMETRY			

MICROSCOPIC EXAMINATION



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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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	MBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
RED BLOOD CELLS (F	RBCs) CENTRIFUGED URINARY SEDIMENT	Value NEGATIVE (-ve)	Unit /HPF	Biological Reference interval 0 - 3	
RED BLOOD CELLS (F by MICROSCOPY ON O PUS CELLS					
RED BLOOD CELLS (F by MICROSCOPY ON (PUS CELLS by MICROSCOPY ON (EPITHELIAL CELLS	CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3	
RED BLOOD CELLS (F by MICROSCOPY ON (PUS CELLS by MICROSCOPY ON (EPITHELIAL CELLS by MICROSCOPY ON (CRYSTALS	CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve) 1-2	/HPF /HPF	0 - 3 0 - 5	

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS	1-2	/HPF	0 - 5
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
EPITHELIAL CELLS	2-4	/HPF	ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY 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
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			

End Of Report





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