



	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultan	obiology)		ugam Chopra MD (Pathology) sultant Pathologist	
NAME	: Mr. RAJAT JAIN				
AGE/ GENDER	: 37 YRS/MALE		PATIENT ID	: 169433	6
COLLECTED BY	:		REG. NO./LAB NO.	:01241	2090017
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBAI	LA CANTT)	REGISTRATION DA	ATE : 09/Dec	/2024 10:52 AM
BARCODE NO.	: 01522196		COLLECTION DATE		/2024 10:55AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 09/Dec	/2024 11:07AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA CANT'I			
Test Name		Value	Unit	it	Biological Reference interval
			ATOLOGY OOD COUNT (CB	BC)	
	(RBCS) COUNT AND INDICES				
HAEMOGLOBIN (HE by CALORIMETRIC	3)	14.4	gm.	/dL	12.0 - 17.0
RED BLOOD CELL (F	RBC) COUNT	5.07 ^H	Mill	llions/cmm	3.50 - 5.00
PACKED CELL VOLU		45.8	%		40.0 - 54.0
MEAN CORPUSCULA		90.5	fL		80.0 - 100.0
	AR HAEMOGLOBIN (MCH) JTOMATED HEMATOLOGY ANALYZER	28.5	pg		27.0 - 34.0
	AR HEMOGLOBIN CONC. (MCHC) JTOMATED HEMATOLOGY ANALYZER	31.5 ^L	g/d	dL	32.0 - 36.0
	TION WIDTH (RDW-CV) JTOMATED HEMATOLOGY ANALYZER	13.4	%		11.00 - 16.00
	TION WIDTH (RDW-SD) JTOMATED HEMATOLOGY ANALYZER	45.3	fL		35.0 - 56.0
MENTZERS INDEX		17.85	RAT	TIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING IND by CALCULATED	EX	24	RAT	TIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CEL	LS (WBCS)				
TOTAL LEUCOCYTE by FLOW CYTOMETRY	COUNT (TLC) by sf cube & microscopy	8430	/cn	nm	4000 - 11000
	LOOD CELLS (nRBCS) T HEMATOLOGY ANALYZER	NIL			0.00 - 20.00
	LOOD CELLS (nRBCS) % ITOMATED HEMATOLOGY ANALYZER	NIL	%		< 10 %





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





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

NAME	: Mr. RAJAT JAIN		
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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	57	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	31	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	7	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4805	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2613	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	422	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	590	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	166000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.23	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	14 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	87000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	52.5 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.6	%	15.0 - 17.0



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Test Name	Value	Unit	Biological Reference interval



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMIST	RY/BIOCHEMIST	TRY
		GLUCOSE F	ASTING (F)	
GLUCOSE FASTING by glucose oxidasi	(F): PLASMA - peroxidase (god-pod)	111.56 ^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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



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NAME



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100 1000

Test Name	Value	Unit	Biological Reference interval
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TIVED	CUNCTION TEST (CO)	MDIETE)	
LIVER	FUNCTION TEST (CO	MPLEIE)	
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.62	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.18	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by Calculated, spectrophotometry	0.44	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	40.3	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	42.2	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by calculated, spectrophotometry	0.95	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	113.83	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by szasz, spectrophtometry	40.63	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.5	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.38	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.12	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by calculated, spectrophotometry	1.4	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:

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





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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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Test Name		Value	Unit	Biological Reference interval
	KIDNI	EY FUNCTIO	N TEST (COMPLETE)
UREA: SERUM	IATE DEHYDROGENASE (GLDH)	19.33	mg/dL	10.00 - 50.00
CREATININE: SERI	UM	1.03	mg/dL	0.40 - 1.40
	ROGEN (BUN): SERUM	9.03	mg/dL	7.0 - 25.0
RATIO: SERUM	ROGEN (BUN)/CREATININE	8.77 ^L	RATIO	10.0 - 20.0
UREA/CREATININ by CALCULATED, SPE		18.77	RATIO	
URIC ACID: SERUM		3.78	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	10.04	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE by PHOSPHOMOLYBE	ERUM DATE, SPECTROPHOTOMETRY	2.66	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	137.2	mmol/L	135.0 - 150.0
POTASSIUM: SERU		4.09	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	102.9	mmol/L	90.0 - 110.0
	IERULAR FILTERATION RATE			
ESTIMATED GLOM (eGFR): SERUM by CALCULATED INTERPRETATION:	ERULAR FILTERATION RATE	95.9		

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 Unit	Biologie	cal Reference interval
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia 	superimposed on renal disease.		uropathy).	
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 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI G1 G2	ass (subnormal creatinine producti tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LE (BUN rises disproportionately mor superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. nd starvation. 2. creased urea synthesis. urea rather than creatinine diffuse monemias (urea is virtually absent of inappropiate antidiuretic harmon (0:1) WITH INCREASED CREATININE: py (accelerates conversion of creat eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false increat creased BUN/creatinine ratio). apy (interferes with creatinine mea JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR	EVELS: The than creatinine) (e.g. obstructive of extracellular fluid). es out of extracellular fluid). in blood). ne) due to tubular secretion of urea. tine to creatinine). ease in creatinine with certain methers assurement). GFR (mL/min/1.73m2) n >90 >90		
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 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERU G1 G2 G3a	ass (subnormal creatinine producti tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LE (BUN rises disproportionately mor superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. nd starvation. 2. creased urea synthesis. urea rather than creatinine diffuse monemias (urea is virtually absent of inappropiate antidiuretic harmon 0:1) WITH INCREASED CREATININE: py (accelerates conversion of creat eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false increat creased BUN/creatinine ratio). apy (interferes with creatinine meat JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR Mild decrease in GFR	EVELS: The than creatinine) (e.g. obstructive of the sout of extracellular fluid). In blood). The due to tubular secretion of urea. The to creatinine). The sease in creatinine with certain methods asurement). GFR (mL/min/1.73m2) n >90 60 - 89	odologies,resulting in nor <u>ASSOCIATED FINDINGS</u> <u>No proteinuria</u> Presence of Protein ,	
8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal 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 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI G1 G2	ass (subnormal creatinine producti tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LE (BUN rises disproportionately mor superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. nd starvation. 2. creased urea synthesis. urea rather than creatinine diffuse monemias (urea is virtually absent of inappropiate antidiuretic harmon (0:1) WITH INCREASED CREATININE: py (accelerates conversion of creat eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false increat creased BUN/creatinine ratio). apy (interferes with creatinine mea JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR	EVELS: The than creatinine) (e.g. obstructive of the sout of extracellular fluid). In blood). The due to tubular secretion of urea. The to creatinine). The surement). The surement of the surement of	odologies,resulting in nor <u>ASSOCIATED FINDINGS</u> <u>No proteinuria</u> Presence of Protein ,	





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Test Name	Value	Unit	Biological Reference interval

COMMENTS:

Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
 eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
 In patients, with eGFR creatinine between 45-59 ml/min/1.73 m2 (G3) and without any marker of Kidney damage, It is recommended to measure of CFD with the commended to measure

3. In patients, with eGFR cleaning between 45-59 minimit 1.73 m2 (G3) and without any marker of Kidney damage, it is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fulfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
7. A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).

ADVICE:

KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated



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Test Name	Value	Unit	Biological Reference interval
	IMMUNOPATH	OLOGY/SEROLOGY	Y
	ANTI SMOOTH MUSCLE	E ANTIBODY (ASMA)	: ELISA
ANTI CMOOTH MU	SCLE ANTIBODY (ASMA) - ELISA 62.7 ^H	IU/mL	NEGATIVE: 0.0 - 40.0

INTERPRETATION:

KOS Diagnostic Lab (A Unit of KOS Healthcare)

1. Smooth muscle autoantibodies (SMA) are found in approximately 3% of normal adult caucasians. 2. High titres (>=1:160) of SMA are found in approximately 97% of patients with autoimmune chronic active hepatitis. SMA are found less frequently in uveitis, drug induced hepatitis, alcoholic liver disease, primary pulmonary hypertension and transiently in acute hepatitis and other viral infections including infectious mononucleosis.
3.Low titer antibodies may be found in the sera of patients with viral infections, malignancies and in the normal population.
4.The presence of SMA is not predictive of the development of liver disease.
5.The absence of ASMA indicates non autoimmune forms of chronic hepatitis.





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TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT





NAME	: Mr. RAJAT JAIN			
AGE/ GENDER	: 37 YRS/MALE		PATIENT ID	: 1694336
COLLECTED BY	:		REG. NO./LAB NO.	: 012412090017
REFERRED BY	: CENTRAL PHOENIX CLUB (AM	MBALA CANTT)	REGISTRATION DATE	: 09/Dec/2024 10:52 AM
BARCODE NO.	:01522196		COLLECTION DATE	:09/Dec/2024 10:55AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 10/Dec/2024 05:58AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT	2	
Test Name		Value	Unit	Biological Reference interval

INTERPRETATION:-

1.For diagnostic purposes, ANA value should be used as an adjuvant to other clinical and laboratory data available.

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2. Measurement of antinuclear antibodies (ANAs) in serum is the most commonly performed screening test for patients suspected of having a systemic rheumatic disease, also referred to as connective tissue disease.

3.ANAs occur in patients with a variety of autoimmune diseases, both systemic and organ-specific. They are particularly common in the systemic rheumatic diseases, which include lupus erythematosus (LE), discoid LE, drug-induced LE, mixed connective tissue disease, Sjogren syndrome scleroderma (systemic sclerosis), CREST (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia) syndrome, polymyositis/dermatomyositis, and rheumatoid arthritis. NOTE:

1. The diagnosis of a systemic rheumatic disease is based primarily on the presence of compatible clinical signs and symptoms.

The results of tests for autoantibodies including ANA and specific autoantibodies are ancillary. Additional diagnostic criteria include consistent histopathology or specific radiographic findings. Although individual systemic rheumatic diseases are relatively uncommon, a great many patients present with clinical findings that are compatible with a systemic rheumatic disease ANA screening may be useful for ruling out the disease.

2.Secondary, disease specific auto antibodies maybe ordered for patients who are screen positive as ancillary aids for the diagnosis of specific auto-immune disorders.





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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 10/Dec/2024 08:58AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		
Test Name	Value	Unit	Biological Reference interval

INTERPRETATION:

1.Immunoglobulin is a humoral antibody consisting of two light and two heavy chains in the molecule.

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2.Approximately 80% of serum immunoglobulins is IgG. Its major function is neutralization of toxin in tissues spaces.3.Antibodies of the IgG class are produced in response to most bacteria and viruses. IgG is the only immunogloblin that can cross the placental

barrier and provide passive immune protection for fetus and new born till about 6 month. 4.Increased levels may be seen in SLE, chronic liver diseases, infectious diseases and cystic fibrosis. Monoclonal IgG increases in IgG myeloma. 5.Decreased synthesis of IgG is found in congenital/ acquired immunodeficiencies and in selective subclass deficiency such as bruton type agammaglobinulinemia.

6. Decreased IgG concentrations are seen in protein-losing enteropathies, nephrotic syndrome and in skin burns.

End Of Report ***





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