

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)	
NAME : Mi	r. SONA OBEROI				
AGE/ GENDER : 34	YRS/MALE		PATIENT ID	: 1377358	
COLLECTED BY :			REG. NO./LAB NO.	:012409280019	
REFERRED BY :			REGISTRATION DATE	: 28/Sep/2024 09:43 AM	
	517863		COLLECTION DATE	: 28/Sep/2024 09:45AM	
	S DIAGNOSTIC LAB		REPORTING DATE	: 28/Sep/2024 10:03AM	
	49/1, NICHOLSON ROAD, AMB.	ALA CANTT			
Test Name		Value	Unit	Biological Reference	interval
	SWAST	THYA WE	LLNESS PANEL: 1.5		
	COM	IPI FTF BI (DOD COUNT (CBC)		
RED BLOOD CELLS (RBCS)					
HAEMOGLOBIN (HB) by Calorimetric		13.1	gm/dL	12.0 - 17.0	
RED BLOOD CELL (RBC) CC	OUNT	4.82	Millions/c	2.50 - 5.00	
PACKED CELL VOLUME (PC		40.4	%	40.0 - 54.0	
MEAN CORPUSCULAR VOL		84	fL	80.0 - 100.0	
MEAN CORPUSCULAR HAE	MOGLOBIN (MCH) ATED HEMATOLOGY ANALYZER	27.1	pg	27.0 - 34.0	
MEAN CORPUSCULAR HEN by CALCULATED BY AUTOM.	NOGLOBIN CONC. (MCHC) ATED HEMATOLOGY ANALYZER	32.3	g/dL	32.0 - 36.0	
RED CELL DISTRIBUTION V by CALCULATED BY AUTOM	VIDTH (RDW-CV) ated hematology analyzer	13.2	%	11.00 - 16.00	
RED CELL DISTRIBUTION V by CALCULATED BY AUTOM.	VIDTH (RDW-SD) ated hematology analyzer	41.5	fL	35.0 - 56.0	
MENTZERS INDEX by CALCULATED		17.43	RATIO	BETA THALASSEMIA IRON DEFICIENCY AN	
GREEN & KING INDEX by CALCULATED		22.94	RATIO	BETA THALASSEMIA IRON DEFICIENCY AN	TRAIT:<= 65.0
WHITE BLOOD CELLS (WB	<u>CS)</u>				
TOTAL LEUCOCYTE COUNT by FLOW CYTOMETRY BY SP		6270	/cmm	4000 - 11000	
NUCLEATED RED BLOOD C		NIL		0.00 - 20.00	
NUCLEATED RED BLOOD C by CALCULATED BY AUTOM	ELLS (nRBCS) % ATED HEMATOLOGY ANALYZER	NIL	%	< 10 %	
DIFFERENTIAL LEUCOCYTE					
NEUTROPHILS by flow cytometry by s	F CUBE & MICROSCOPY	49 ^L	%	50 - 70	

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Dr. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. SONA OBEROI AGE/ GENDER : 34 YRS/MALE **PATIENT ID** :1377358 **COLLECTED BY** :012409280019 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 28/Sep/2024 09:43 AM **BARCODE NO.** :01517863 **COLLECTION DATE** : 28/Sep/2024 09:45AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 28/Sep/2024 10:03AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** 42^H LYMPHOCYTES % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 3 % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES % 2 - 12 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE LEUKOCYTES (WBC) COUNT** 3072 ABSOLUTE NEUTROPHIL COUNT /cmm 2000 - 7500 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 2633 /cmm 800 - 4900 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 40 - 440 188 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 376 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 188000 /cmm 150000 - 450000 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.23 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 6.50 - 12.0 12^H fl by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 30000 - 90000 PLATELET LARGE CELL COUNT (P-LCC) 77000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) 40.8 % 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) % 16.5 15.0 - 17.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE



NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 28/Sep/2024 01:34PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		1
Test Name		Value	Unit	Biological Reference interval
	GLY	COSYLATED HA	EMOGLOBIN (HBA1C)	
GLYCOSYLATED HAEN WHOLE BLOOD	NOGLOBIN (HbA1c):	5.5	%	4.0 - 6.4
ESTIMATED AVERAGE		111.15	mg/dL	60.00 - 140.00
	AS PER AMERICAN	DIABETES ASSOCI	ATION (ADA):	
	REFERENCE GROUP		YCOSYLATED HEMOGLOGIB	(HBAIC) in %
	abetic Adults >= 18 years	/	<5.7	
	t Risk (Prediabetes)		5.7 – 6.4	
D	iagnosing Diabetes	_	>= 6.5	
			Age > 19 Years	7.0
Thorapout	ic goals for glycemic control		of Therapy:	< 7.0
inerapeut	ic goals for grycernic control	Action	s Suggested: Age < 19 Years	>8.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

COMMENTS:

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients. 2.Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

3. Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropriate.

4. High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications 5. Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	ERYTH	ROCYTE SEDIME	NTATION RATE (ESI	R)
by RED CELL AGGREN INTERPRETATION: 1. ESR is a non-specifi immune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythy CONDITION WITH LO A low ESR can be see (polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactive 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dext	does not tell the health practition cted by other conditions besides in be used to monitor disease activit ematosus WESR n with conditions that inhibit the hificantly high white blood cell cou- e cell anaemia) also lower the ESI e protein (C-RP) are both markers es not change as rapidly as does CF by as many other factors as is ESR ed, it is typically a result of two ty ye a higher ESR, and menstruation	often indicates the per exactly where the nflammation. For the y and response to t normal sedimentati unt (leucocytosis), a R. of inflammation. RP, either at the start , making it a better pes of proteins, glol and pregnancy can	e inflammation is in the is reason, the ESR is typ herapy in both of the al on of red blood cells, su and some protein abno et of inflammation or as marker of inflammation pulins or fibrinogen. cause temporary eleva	ion associated with infection, cancer and auto- e 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 rmalities. Some changes in red cell shape (such s it resolves.



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REP	ORTING DATE	: 28/Sep/2024 10:57AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
CLIENT ADDRESS Test Name	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT Value	Unit	Biological Reference interval
		Value	//BIOCHEMISTR	

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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SO 9001 : 2008 CERT			EXCELLENCE IN HEALTHCARE	a DIAGNOSTICS
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Test Name		Value	Unit	Biological Reference interval
		LIPID PROFILE :	BASIC	
CHOLESTEROL TOTA	L: SERUM	178.14	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX				BORDERLINE HIGH: 200.0 - 239. HIGH CHOLESTEROL: > OR = 240
TRIGLYCERIDES: SER	RUM PHATE OXIDASE (ENZYMATIC)	242.57 ^H	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199. HIGH: 200.0 - 499.0
		34.9	mg/dL	VERY HIGH: > OR = 500.0 LOW HDL: < 30.0
HDL CHOLESTEROL (by SELECTIVE INHIBIT		34.9	mg/uL	BORDERLINE HIGH HDL: 30.0 -
				60.0
				HIGH HDL: $> OR = 60.0$
LDL CHOLESTEROL: S by CALCULATED, SPE		94.73	mg/dL	OPTIMAL: < 100.0 Above optimal: 100.0 - 129.0
2, 0, 2002, 1, 20, 0, 2				BORDERLINE HIGH: 130.0 - 129.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTE		143.24 ^H	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPE	ECTROPHOTOMETRY			ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189. HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL		48.51 ^H	mg/dL	0.00 - 45.00
by CALCULATED, SPE TOTAL LIPIDS: SERUI by CALCULATED, SPE	M	598.85	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL by CALCULATED, SPE	RATIO: SERUM	5.1 ^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: SER by CALCULATED, SPE		2.71	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

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Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD		6.95 ^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
	LIVE	R FUNCTION	TEST (COMPLETE)	
BILIRUBIN TOTAL: S by diazotization, s	ERUM SPECTROPHOTOMETRY	1.41 ^H	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	CONJUGATED): SERUM	0.24	mg/dL	0.00 - 0.40
	(UNCONJUGATED): SERUM	1.17 ^H	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	18.54	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	27.92	U/L	0.00 - 49.00
AST/ALT RATIO: SER by CALCULATED, SPE		0.66	RATIO	0.00 - 46.00
ALKALINE PHOSPHA by PARA NITROPHEN PROPANOL	TASE: SERUM YL PHOSPHATASE BY AMINO METHYL	82.84	U/L	40.0 - 130.0
GAMMA GLUTAMYL by SZASZ, SPECTROF	. TRANSFERASE (GGT): SERUM PHTOMETRY	15.51	U/L	0.00 - 55.0
TOTAL PROTEINS: SE by BIURET, SPECTRO	ERUM	6.6	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by bromocresol g	REEN	3.91	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPE		2.69	gm/dL	2.30 - 3.50
A : G RATIO: SERUM	l	1.45	RATIO	1.00 - 2.00

Dr. Vinay Chopra

by CALCULATED, SPECTROPHOTOMETRY

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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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
	1.2 1.0



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	KIE	ONEY FUNCTIO	ON TEST (COMPLETE)	
UREA: SERUM		19.49	mg/dL	10.00 - 50.00
-	ATE DEHYDROGENASE (GLDH)	0.01		
CREATININE: SERUN by ENZYMATIC, SPEC		0.91	mg/dL	0.40 - 1.40
BLOOD UREA NITRO)GEN (BUN): SERUM	9.11	mg/dL	7.0 - 25.0
-		10.01	DATIO	10.0 20.0
RATIO: SERUM	OGEN (BUN)/CREATININE	10.01	RATIO	10.0 - 20.0
by CALCULATED, SPE	ECTROPHOTOMETRY			
	RATIO: SERUM ECTROPHOTOMETRY	21.42	RATIO	
URIC ACID: SERUM		5.96	mg/dL	3.60 - 7.70
by URICASE - OXIDAS	SE PEROXIDASE			
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	9.34	mg/dL	8.50 - 10.60
PHOSPHOROUS: SEF		3.88	mg/dL	2.30 - 4.70
	DATE, SPECTROPHOTOMETRY			
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	/F ELECTRODE)	140.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUM		3.98	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV				
CHLORIDE: SERUM by ISE (ION SELECTIV	/F FL FCTRODF)	105.38	mmol/L	90.0 - 110.0
	RULAR FILTERATION RATE			
ESTIMATED GLOME	RULAR FILTERATION RATE	113.4		
(eGFR): SERUM				
by CALCULATED				

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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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST

MBBS, MD (PATHOLOGY)



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	Dr. Vinay Ch MD (Pathology & Chairman & Cor	& Microbiology)	: Yugam Cho MD (Patho Consultant Patho	ology)	
NAME	: Mr. SONA OBEROI				
AGE/ GENDER	: 34 YRS/MALE	PATIENT ID	: 1;	377358	
COLLECTED BY		REG. NO./LAB		12409280019	
REFERRED BY	:	REGISTRATION		3/Sep/2024 09:43 AM	
BARCODE NO.	: 01517863	COLLECTION D	ATE : 28	3/Sep/2024 09:45AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DA	TE : 28	3/Sep/2024 10:57AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT			
Test Name		Value	Unit	Biological Referen	nce interval
	nd starvation. e. creased urea synthesis.				
 Inherited hyperam SIADH (syndrome of Beregnancy. Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin thei Company GLOMERIC 	monemias (urea is virtually absorb of inappropiate antidiuretic harn (0:1) WITH INCREASED CREATINII py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE:	none) due to tubular secretion of u NE: reatine to creatinine). ncrease in creatinine with certain r measurement).	nethodologies,r		when dehydratic
 Inherited hyperam SIADH (syndrome of Beregnancy. Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin thera ESTIMATED GLOMERI CKD STAGE 	monemias (urea is virtually absorb of inappropiate antidiuretic harn (0:1) WITH INCREASED CREATINI py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false ir creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION	ent in blood). none) due to tubular secretion of u NE: reatine to creatinine). ncrease in creatinine with certain r measurement). GFR (mL/min/1.73m2)	nethodologies,r	TED FINDINGS	when dehydratic
 Inherited hyperam SIADH (syndrome of Beregnancy. Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin thei Company GLOMERIC 	monemias (urea is virtually absorb of inappropiate antidiuretic harn (0:1) WITH INCREASED CREATINII py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE:	ent in blood). none) due to tubular secretion of u NE: reatine to creatinine). ncrease in creatinine with certain r measurement). GFR (mL/min/1.73m2) ction >90	nethodologies,r ASSOCIA No p	TED FINDINGS	when dehydratic
 Inherited hyperam SIADH (syndrome of Beregnancy. Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin their ESTIMATED GLOMERI CKD STAGE G1 	monemias (urea is virtually absorb of inappropiate antidiuretic harn (0:1) WITH INCREASED CREATINII py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false ir creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION Normal kidney func- Kidney damage w normal or high Gi	ent in blood). none) due to tubular secretion of u NE: reatine to creatinine). ncrease in creatinine with certain r measurement). GFR (mL/min/1.73m2) ction >90 ith >90 FR	nethodologies,r ASSOCIA No p Presenc	TED FINDINGS	when dehydratic
5. Inherited hyperam 7. SIADH (syndrome of 3. 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 CKD STAGE G1 G2 G3a	monemias (urea is virtually absorb inappropiate antidiuretic harn py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false ir creased BUN/creatinine ratio). apy (interferes with creatinine r UAR FILTERATION RATE: DESCRIPTION Normal kidney func Kidney damage w normal or high Gi	ent in blood). none) due to tubular secretion of u NE: reatine to creatinine). ncrease in creatinine with certain r measurement). GFR (mL/min/1.73m2) ction >90 ith >90 FR 60 -89	nethodologies,r ASSOCIA No p Presenc	TED FINDINGS Toteinuria	when dehydratio
 Inherited hyperam SIADH (syndrome of Beregnancy. Pregnancy. Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin their ESTIMATED GLOMERI G1 G2 	monemias (urea is virtually absorb of inappropiate antidiuretic harn (0:1) WITH INCREASED CREATINII py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false ir creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION Normal kidney func- Kidney damage w normal or high Gi	ent in blood). none) due to tubular secretion of u NE: reatine to creatinine). ncrease in creatinine with certain r measurement). GFR (mL/min/1.73m2) ction >90 ith >90 FR 60 -89 n GFR 60 -89 n GFR 30-59	nethodologies,r ASSOCIA No p Presenc	TED FINDINGS Toteinuria	when dehydratic

G5

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

Kidney failure

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

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NAME	: Mr. SONA OBEROI		
AGE/ GENDER	: 34 YRS/MALE	PATIENT ID	: 1377358
COLLECTED BY	:	REG. NO./LAB NO.	: 012409280019
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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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CLIENT CODE.	: KOS DIAGNOSTI	C LAB		REPORTING DATE				
CLIENT ADDRESS	: 6349/1, NICHOL		AMBALA CANTT		··········			
Test Name			Value	Unit	Biological Reference interval			
IRON: SERUM			IRON 63.3	PROFILE μg/dL	59.0 - 158.0			
by FERROZINE, SPEC	CTROPHOTOMETRY		03.3	µg/aL	59.0 - 158.0			
UNSATURATED IRO	N BINDING CAPACIT	Y (UIBC)	177.47	μg/dL	150.0 - 336.0			
SERUM								
by FERROZINE, SPEC	CTROPHOTOMETERY		240.77	μg/dL	230 - 430			
:SERUM			210.77	μ ₀ , αι	200 100			
by SPECTROPHOTOM								
%TRANSFERRIN SAT		(FERENE)	26.29	%	15.0 - 50.0			
by CALCULATED, SPECTROPHOTOMETERY (FERENE) TRANSFERRIN: SERUM		170.95 ^L	mg/dL	200.0 - 350.0				
by SPECTROPHOTO	METERY (FERENE)		170.70	3.1				
<u>INTERPRETATION:-</u> VARIAE	RIFS A	NEMIA OF CH	RONIC DISEASE	IRON DEFICIENCY ANEMI	A THALASSEMIA α/β TRAIT			
SERUM I			Reduced	Reduced	Normal			
TOTAL IRON BIND	DING CAPACITY:	Decr	eased	Increased	Normal			
% TRANSFERRIN	SATURATION:	Decr	eased	Decreased < 12-15 %	Normal			
	DDITIN	NI 1.1		D				

IRON:

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT

1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia, anemia of chronic disease and thalassemia syndromes. 2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for

Decreased

iron deficiency anemia, is severely contra-indicated in Thalassemia. TOTAL IRON BINDING CAPACITY (TIBC):

SERUM FERRITIN:

1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

Normal to Increased

% TRANSFERRIN SATURATION:

1. Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.



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Normal or Increased





		hopra & Microbiology) onsultant Pathologist	Dr. Yugan MD CEO & Consultant	(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		FERRI	ΓΙΝ	
FERRITIN: SERUM		203.37	ng/mL	21.81 - 274.66

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy. DECREASED:

1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.

KOS Diagnostic Lab (A Unit of KOS Healthcare)

- 2. Hypothyroidism.
 3. Vitamin-C deficiency

INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

1. Hemochromatosis or hemosiderosis.

2. Wilson Disease.

INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

- 1. Transfusion overload
- 2. Excess dietary Iron
- 3. Porphyria Cutanea tada
- 4. Ineffective erythropoiesis

INCREASED FERRITIN WITHOUT IRON OVERLOAD:

- 1. Liver disorders (NASH) or viral hepatitis (B/C)
- 2. Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
- 3. Leukaemia, hodgkin's disease.
- 4. Alcohol excess.

5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma, synthesis of ferritin by tumour cells.

6. Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can thererfore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive

proteins to rule out any inflammatory conditions. 2. Patients with iron deficiency anaemia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.



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NAME	: Mr. SONA OBEROI			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	Value	Unit	Biological Reference interval
	THYR		RINOLOGY	
TRIIODOTHYRONINE by CMIA (CHEMILUMIN	E (T3): SERUM iescent microparticle immunoassay)	0.768	ng/mL	0.35 - 1.93
THYROXINE (T4): SEI	RUM iescent microparticle immunoassay)	7.02	μgm/dL	4.87 - 12.60
by CMIA (CHEMILUMIN 3rd GENERATION, ULT <u>INTERPRETATION</u> :		3.435	µIU/mL	0.35 - 5.50 m. The variation is of the order of 50%.Hence time of th

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

LIMITATIONS:-

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

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

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

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

TRIIODOTHY	(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





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NAME	: Mr. SON	A OBEROI				
AGE/ GENDER	: 34 YRS/M	IALE		PATIENT ID	: 1377	7358
COLLECTED BY	:			REG. NO./LAB NO	. :012	409280019
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CLIENT ADDRESS	: 6349/1, 1	NICHOLSON ROAD,	AMBALA CANTT			
						
Test Name			Value	Un	hit	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	

RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (μιU/mL) 1st Trimester 0.10 - 2.50	
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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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Test Name		Value	Unit	Biological Reference interval
		AMIN D/25 H	AMINS YDROXY VITAMIN D3	
VITAMIN D (25-HYDROXY VITAMI by clia (chemiluminescence imm INTERPRETATION:		26.197 ^L	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
DEFICIENT:		< 20	n	g/mL
INSUFFICIENT:		21 - 29	n	g/mL
PREFFERED RANGE: INTOXICATION:		30 - 100 > 100		g/mL g/mL
tissue and tightly bound by a transp 3.Vitamin D plays a primary role in phosphate reabsorption, skeletal c: 4.Severe deficiency may lead to fail DECREASED: 1.Lack of sunshine exposure. 2.Inadequate intake, malabsorption 3.Depressed Hepatic Vitamin D 25- 4.Secondary to advanced Liver dise 5.Osteoporosis and Secondary Hyp 5.Enzyme Inducing drugs: anti-epile NCREASED: 1. Hypervitaminosis D is Rare, and i severe hypercalcemia and hyperpho CAUTION : Replacement therapy in o hypervitaminosis D	port protein while the maintenance alcium deposition, ure to mineralize n (celiac disease) hydroxylase activitase erparathroidism (N eptic drugs like phe s seen only after p ophatemia. deficient individua	in circulation. of calcium homeo calcium mobiliza newly formed ost ty Mild to Moderate enytoin, phenoba prolonged exposu Is must be monito	ostatis. It promotes calciur ition, mainly regulated by p teoid in bone, resulting in r deficiency) rbital and carbamazepine, re to extremely high doses pred by periodic assessmer	port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH). ickets in children and osteomalacia in adults. that increases Vitamin D metabolism. of Vitamin D. When it occurs, it can result in it of Vitamin D levels in order to prevent <i>iency due to excess of melanin pigment which</i>





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GE/ GENDER :34 YRS/MALE PATIENT ID :1377358 OLLECTED BY :		Dr. Vinay Ch MD (Pathology & Chairman & Con		Dr. Yugam MD CEO & Consultant	(Pathology)	
OLLECTED BY Image: September 2004 09:43 AM EFFRRED BY REGISTRATION DATE :28/Sep/2024 09:43 AM ARCODE NO. :01517863 COLLECTION DATE :28/Sep/2024 09:43 AM ARCODE NO. :01517863 COLLECTION DATE :28/Sep/2024 09:43 AM LIENT CODE. :KOS DIAGNOSTIC LAB REPORTING DATE :28/Sep/2024 11:56AM LIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT : : CUTAMIN B12/COBALAMIN: SERUM NITAMIN B12/COBALAMIN: SERUM biological Reference interval VITAMIN B12/COBALAMIN: SERUM 183 ^L DIGECREASED VITAMIN B12 1.1ngestion of Vitamin C 1.1rgestion of Vitamin C 1.1rgestion of Vitamin A	NAME	: Mr. SONA OBEROI				
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roprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients hav	leal resection, small	l intestinal diseases).				
	5.Vitamin B12 deficie	ency frequently causes macrocyt	ic anemia, glossitis, pe	ripheral neuropathy,	weakness, hyperreflexia, ataxia, loss of	
ne neurologic defects without macrocytic anemia.			avioral changes. These	mannestations may c	becur in any combination; many patients have	

6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.

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.





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



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	Dr. Vinay Cho MD (Pathology & Chairman & Cons	Microbiology)	Dr. Yugam MD EO & Consultant	(Pathology)
AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE.	AE: Mr. SONA OBEROIA/ GENDER: 34 YRS/MALELECTED BY:ERRED BY:CODE NO.: 01517863ENT CODE.: KOS DIAGNOSTIC LABENT ADDRESS: 6349/1, NICHOLSON ROAD, A		T ID /LAB NO. PATION DATE TION DATE ING DATE	: 1377358 : 012409280019 : 28/Sep/2024 09:43 AM : 28/Sep/2024 09:45AM : 28/Sep/2024 11:30AM
Test Name		Value	Unit	Biological Reference interval
PHYSICAL EXAMINATION		CLINICAL PATHO		TON
QUANTITY RECIEVED by DIP STICK/REFLECTA COLOUR by DIP STICK/REFLECTA TRANSPARANCY by DIP STICK/REFLECTA SPECIFIC GRAVITY	NNCE SPECTROPHOTOMETRY NNCE SPECTROPHOTOMETRY NNCE SPECTROPHOTOMETRY	10 PALE YELLOW CLEAR 1.02	ml	PALE YELLOW CLEAR 1.002 - 1.030
PROTEIN by DIP STICK/REFLECTA SUGAR by DIP STICK/REFLECTA PH by DIP STICK/REFLECTA BILIRUBIN by DIP STICK/REFLECTA NITRITE	ANCE SPECTROPHOTOMETRY ANCE SPECTROPHOTOMETRY ANCE SPECTROPHOTOMETRY ANCE SPECTROPHOTOMETRY ANCE SPECTROPHOTOMETRY	ACIDIC Negative Negative 6 Negative Negative		NEGATIVE (-ve) NEGATIVE (-ve) 5.0 - 7.5 NEGATIVE (-ve) NEGATIVE (-ve)
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MICROSCOPIC EXAMINATION



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

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

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Dr. Vinay Chopra

MD (Pathology & Microbiology)

EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

Dr. Yugam Chopra MD (Pathology)

Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. SONA OBEROI AGE/ GENDER : 34 YRS/MALE **PATIENT ID** :1377358 **COLLECTED BY** REG. NO./LAB NO. :012409280019 **REFERRED BY REGISTRATION DATE** : 28/Sep/2024 09:43 AM **BARCODE NO.** :01517863 **COLLECTION DATE** : 28/Sep/2024 09:45AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :28/Sep/2024 11:30AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** NEGATIVE (-ve) **RED BLOOD CELLS (RBCs)** /HPF 0 - 3 by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT PUS CELLS 2-3 /HPF 0 - 5 by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT EPITHELIAL CELLS 1-2 /HPF ABSENT by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT CRYSTALS NEGATIVE (-ve) NEGATIVE (-ve) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT NEGATIVE (-ve) NEGATIVE (-ve) CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT BACTERIA NEGATIVE (-ve) **NEGATIVE** (-ve)

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT OTHERS

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT TRICHOMONAS VAGINALIS (PROTOZOA)

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

*** End Of Report ***

NEGATIVE (-ve)

ABSENT





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

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NEGATIVE (-ve)

ABSENT