



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultan	obiology)		D (Pathology)	
NAME :	Mrs. IPSA				
AGE/ GENDER :	37 YRS/FEMALE		PATIENT ID	: 1676969	
<b>COLLECTED BY</b> :			REG. NO./LAB NO.	: 042411200001	
<b>REFERRED BY</b> :			<b>REGISTRATION DATE</b>	: 20/Nov/2024 11:53 AM	
	A1164445		COLLECTION DATE	: 20/Nov/2024 03:38PM	
	KOS DIAGNOSTIC SHAHBAD		REPORTING DATE	: 20/Nov/2024 03:56PM	
CLIENT ADDRESS :	6349/1, NICHOLSON ROAD, AMBA	ALA CANTT			
Test Name		Value	Unit	<b>Biological Reference interv</b>	val
PED BLOOD CELLS (1			FHY INDIA PACKA OOD COUNT (CBC)	AGE	
HAEMOGLOBIN (HB)	<u>(DCS) COUNT AND INDICES</u>	12.9	gm/dL	12.0 - 16.0	
by CALORIMETRIC			Ŭ		
RED BLOOD CELL (RB	C) COUNT USING, ELECTRICAL IMPEDENCE	5.45 <sup>H</sup>	Millions	s/cmm 3.50 - 5.00	
PACKED CELL VOLUM		42.2	%	37.0 - 50.0	
MEAN CORPUSCULAR		77.5 <sup>L</sup>	fL	80.0 - 100.0	
	HAEMOGLOBIN (MCH) DMATED HEMATOLOGY ANALYZER	23.6 <sup>L</sup>	pg	27.0 - 34.0	
	HEMOGLOBIN CONC. (MCHC) DMATED HEMATOLOGY ANALYZER	30.5 <sup>L</sup>	g/dL	32.0 - 36.0	
	DMATED HEMATOLOGY ANALYZER	15.4	%	11.00 - 16.00	
RED CELL DISTRIBUT	ION WIDTH (RDW-SD)	44.8	fL	35.0 - 56.0	
MENTZERS INDEX by CALCULATED		14.22	RATIO	BETA THALASSEMIA TRAI 13.0 IRON DEFICIENCY ANEMI >13.0	
GREEN & KING INDEX		21.83	RATIO	BETA THALASSEMIA TRAI 65.0 IRON DEFICIENCY ANEMI 65.0	
WHITE BLOOD CELLS		5040		4000 11000	
TOTAL LEUCOCYTE CO	JUNT (TLC) ′ SF CUBE & MICROSCOPY	5640	/cmm	4000 - 11000	
NUCLEATED RED BLO		NIL		0.00 - 20.00	
	OD CELLS (nRBCS) %	NIL	%	< 10 %	





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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





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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	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	45 <sup>L</sup>	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	40	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	10 <sup>H</sup>	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	%	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	2538	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2256	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	564 <sup>H</sup>	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	282	/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	297000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.39 <sup>H</sup>	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	13 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	149000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	50.1 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.2	%	15.0 - 17.0





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







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NAME	: Mrs. IPSA		
	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Patholo		(Pathology)



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





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	MD (Patholog	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologist		r <b>Chopra</b> (Pathology) Pathologist
AME	: Mrs. IPSA			
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LIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
est Name		Value	Unit	<b>Biological Reference interval</b>
stemic lupus eryth	be used to monitor disease ad ematosus <b>W ESR</b>	, , , , , , , , , , , , , , , , , , ,	o therapy in both of the a	bove diseases as well as some others, such as
This test may also stemic lupus eryth DNDTION WITH LO low ESR can be see olycythaemia), sig sickle cells in sick DTE: ESR and C - reactiv Generally, ESR do CRP is not affected If the ESR is elevat Women tend to ha Drugs such as dex	be used to monitor disease ac ematosus <b>W ESR</b> en with conditions that inhibit nificantly high white blood cel le cell anaemia) also lower th re protein (C-RP) are both mar es not change as rapidly as door <b>I by as many other factors as is</b> ed, it is typically a result of tw ave a higher ESR, and menstrue	the normal sedimenta I count (leucocytosis) e ESR. kers of inflammation. es CRP, either at the s ESR, making it a bette ro types of proteins, g ation and pregnancy c	o therapy in both of the a ation of red blood cells, si , and some protein abno tart of inflammation or as er marker of inflammatior lobulins or fibrinogen. an cause temporary eleva	bicallý 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.

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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

MBBS, MD (PATHOLOGY)







	MD (Patho	<b>ay Chopra</b> blogy & Microbiology) & Consultant Pathologist	Dr. Yugan MD CEO & Consultant	(Pathology)
NAME	: Mrs. IPSA			
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BARCODE NO.	: A1164443	CO	DLLECTION DATE	: 20/Nov/2024 03:38PM
CLIENT CODE.	: KOS DIAGNOSTIC SHA	HBAD RI	EPORTING DATE	: 20/Nov/2024 05:24PM
CLIENT ADDRESS	: 6349/1, NICHOLSON I	ROAD, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CI	INICAL CHEMISTI	RY/BIOCHEMIST	'RY
		GLUCOSE FA	ASTING (F)	

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	<b>Biological Reference interval</b>
		I IPIN PRO	OFILE : BASIC	
CHOLESTEROL TO	TAL · SERUM	171.22	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		171.22	ing/ dL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
		110.41		240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)		118.41	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0
				HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERO	L (DIRECT): SERUM ION	49.57	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0
				HIGH HDL: $> OR = 60.0$
LDL CHOLESTEROI by CALCULATED, SPE		97.97	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0
NON HDL CHOLEST	FROI · SERUM	121.65	mg/dL	VERY HIGH: > OR = 190.0 OPTIMAL: < 130.0
by CALCULATED, SPE		121.00		ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTERC		23.68	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER by CALCULATED, SPE	CUM	460.85	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPE	DL RATIO: SERUM	3.45	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
anasa waa				



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Test Name		Value	Unit	<b>Biological Reference interval</b>
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	IDL RATIO: SERUM	2.39 <sup>L</sup>	RATIO	3.00 - 5.00

## by CALCULATED, SPECTROPHOTOMETRY **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.

 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.
 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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Dr. Yugam Chopra

MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** : Mrs. IPSA NAME AGE/ GENDER : 37 YRS/FEMALE **PATIENT ID** :1676969 **COLLECTED BY** :042411200001 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 20/Nov/2024 11:53 AM **BARCODE NO.** :A1164444 **COLLECTION DATE** : 20/Nov/2024 03:38PM CLIENT CODE. : KOS DIAGNOSTIC SHAHBAD **REPORTING DATE** : 20/Nov/2024 05:49PM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Value Unit Test Name **Biological Reference interval** LIVER FUNCTION TEST (COMPLETE) BILIRUBIN TOTAL: SERUM 0.49 mg/dL INFANT: 0.20 - 8.00 by DIAZOTIZATION, SPECTROPHOTOMETRY ADULT: 0.00 - 1.20 0.00 - 0.40 BILIRUBIN DIRECT (CONJUGATED): SERUM 0.12 mg/dL by DIAZO MODIFIED, SPECTROPHOTOMETRY BILIRUBIN INDIRECT (UNCONJUGATED): SERUM 0.37 mg/dL 0.10 - 1.00 by CALCULATED, SPECTROPHOTOMETRY 14.67.00 - 45.00 SGOT/AST: SERUM U/L by IFCC, WITHOUT PYRIDOXAL PHOSPHATE SGPT/ALT: SERUM 11.4 U/L 0.00 - 49.00 by IFCC, WITHOUT PYRIDOXAL PHOSPHATE AST/ALT RATIO: SERUM 1.28 RATIO 0.00 - 46.00 by CALCULATED, SPECTROPHOTOMETRY ALKALINE PHOSPHATASE: SERUM 62.47 U/L 40.0 - 130.0 by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM 10.2 U/L 0.00 - 55.0 by SZASZ, SPECTROPHTOMETRY TOTAL PROTEINS: SERUM 7.52 gm/dL 6.20 - 8.00 by BIURET, SPECTROPHOTOMETRY 4.22 ALBUMIN: SERUM gm/dL 3.50 - 5.50 by BROMOCRESOL GREEN 3.3 2.30 - 3.50 **GLOBULIN: SERUM** gm/dL

Dr. Vinay Chopra

INTERPRETATION

A : G RATIO: SERUM

by CALCULATED, SPECTROPHOTOMETRY

by CALCULATED, SPECTROPHOTOMETRY

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)

1.28



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RATIO

1.00 - 2.00

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Test Name	· · · · ·	Value Unit	Biological Reference interval

Test Name	Value	Unit	<b>Biological Reference interval</b>

# **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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Page 9 of 18





Dr. Yugam Chopra

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	KIDNE	Y FUNCTION 7	FEST (COMPLETE)	
UREA: SERUM		20.77	mg/dL	10.00 - 50.00
•	ATE DEHYDROGENASE (GLDH)	0.07		
CREATININE: SERU		0.95	mg/dL	0.40 - 1.20
BLOOD UREA NITE	COGEN (BUN): SERUM	9.71	mg/dL	7.0 - 25.0
BLOOD UREA NITE RATIO: SERUM	ROGEN (BUN)/CREATININE	10.22	RATIO	10.0 - 20.0
by CALCULATED, SPE				
UREA/CREATININ by CALCULATED, SPE		21.86	RATIO	
URIC ACID: SERUM	[	<b>2.38<sup>L</sup></b>	mg/dL	2.50 - 6.80
by URICASE - OXIDAS CALCIUM: SERUM	E PEROXIDASE	9.85	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE			Ũ	
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY		3.15	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	'E ELECTRODE)	140.4	mmol/L	135.0 - 150.0
POTASSIUM: SERU	Μ	4.33	mmol/L	3.50 - 5.00
CHLORIDE: SERUM	[	105.3	mmol/L	90.0 - 110.0
ESTIMATED GLOM	IERULAR FILTERATION RATE			
ESTIMATED GLOM	ERULAR FILTERATION RATE	79.1		

Dr. Vinay Chopra

ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM

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 PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT

by CALCULATED





		<b>Dr. Vinay Chopra</b> 1D (Pathology & Microt Chairman & Consultant I			gam Chopr MD (Patholog) ultant Pathologis	/)		
NAME	: Mrs. IPSA							
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CLIENT ADDRESS	: 6349/1, NICH	IOLSON ROAD, AMBAL	A CANTT					
Test Name		V	alue	Unit		Biological	Reference i	nterval
<ol> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>INCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;1</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> </ol>	tetracycline, glu <b>0:1) WITH ELEVA</b> (BUN rises dispr superimposed or <b>0:1) WITH DECRE</b> psis. Id starvation.	reatinine production) cocorticoids) <b>TED CREATININE LEVELS</b> oportionately more than orenal disease.		(e.g. obstructive ι	iropathy).			
<ol> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;1</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>Inherited hyperam</li> <li>SIADH (syndrome c</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;1</li> <li>Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin ther</li> </ol>	ass (subnormal c tetracycline, glue 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. d starvation. creased urea syn urea rather than monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates c eleases muscle c who develop rer sis (acetoacetate creased BUN/cre apy (interferes w	reatinine production) cocorticoids) <b>TED CREATININE LEVELS</b> oportionately more that a renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses out is virtually absent in bl ntidiuretic harmone) du <b>ASED CREATININE:</b> onversion of creatine to reatinine). al failure. causes false increase i atinine ratio). vith creatinine measure <b>LRATE:</b>	an creatinine) t of extracellu ood). ue to tubular s o creatinine). in creatinine w ment).	lar fluid). ecretion of urea. vith certain metho	odologies,resul	-	I ratio when o	dehydratio
<ol> <li>B. Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>INCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;1</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>Inherited hyperam</li> <li>SIADH (syndrome c</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;1</li> <li>Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin ther</li> <li>ESTIMATED GLOMERL</li> <li>CKD STAGE</li> </ol>	ass (subnormal c tetracycline, glue 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates c eleases muscle c who develop ren sis (acetoacetate creased BUN/cre apy (interferes w LAR FILTERATION	reatinine production) cocorticoids) <b>TED CREATININE LEVELS</b> oportionately more that a renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses out is virtually absent in bl ntidiuretic harmone) du <b>ASED CREATININE:</b> onversion of creatine to reatinine). al failure. causes false increase i atinine ratio). rith creatinine measure <b>I RATE:</b> <b>DESCRIPTION</b>	an creatinine) t of extracellu ood). ue to tubular s o creatinine). in creatinine v ment). GFR ( mL/r	lar fluid). ecretion of urea. vith certain metho nin/1.73m2 )	odologies,resul ASSOCIATED	FINDINGS	I ratio when o	dehydratio
<ol> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;1</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>Inherited hyperam</li> <li>SIADH (syndrome c</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;1</li> <li>Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin ther</li> <li>ESTIMATED GLOMERL</li> <li>CKD STAGE</li> </ol>	ass (subnormal c tetracycline, glue 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates c eleases muscle c who develop ren sis (acetoacetate creased BUN/cre apy (interferes w LAR FILTERATION	reatinine production) cocorticoids) <b>TED CREATININE LEVELS</b> oportionately more that a renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses out is virtually absent in bl ntidiuretic harmone) du <b>ASED CREATININE:</b> onversion of creatine to reatinine). al failure. causes false increase i atinine ratio). th creatinine measure <b>I RATE:</b> <b>DESCRIPTION</b> nal kidney function	an creatinine) t of extracellu ood). ue to tubular s o creatinine). in creatinine v ment). GFR ( mL/m	lar fluid). ecretion of urea. vith certain metho nin/1.73m2 )	odologies,resul ASSOCIATED No prote	FINDINGS inuria	I ratio when o	dehydratio
<ol> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;1</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>Inherited hyperam</li> <li>SIADH (syndrome c</li> <li>Pregnancy.</li> <li>PCREASED RATIO (&lt;1</li> <li>Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin ther</li> <li>ESTIMATED GLOMERL</li> <li>CKD STAGE</li> </ol>	ass (subnormal of tetracycline, glue 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ren sis (acetoacetate creased BUN/crea apy (interferes w LAR FILTERATION Norn Kio	reatinine production) cocorticoids) <b>TED CREATININE LEVELS</b> oportionately more that a renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses out is virtually absent in bl ntidiuretic harmone) du <b>ASED CREATININE:</b> onversion of creatine to reatinine). al failure. causes false increase i atinine ratio). th creatinine measure <b>I RATE:</b> <b>DESCRIPTION</b> nal kidney function iney damage with	an creatinine) t of extracellu ood). ue to tubular s o creatinine). in creatinine v ment). GFR ( mL/m	lar fluid). ecretion of urea. vith certain metho nin/1.73m2 )	odologies,resul ASSOCIATED	FINDINGS inuria Protein ,	I ratio when o	dehydratio
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 ESTIMATED GLOMERL CKD STAGE	ass (subnormal of tetracycline, glue 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea f inappropiate a 0:1) WITH INCRE oy (accelerates of eleases muscle of who develop ren sis (acetoacetate creased BUN/crea apy (interferes w LAR FILTERATION Norm Norm	reatinine production) cocorticoids) <b>TED CREATININE LEVELS</b> oportionately more that a renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses out is virtually absent in bl ntidiuretic harmone) du <b>ASED CREATININE:</b> onversion of creatine to reatinine). al failure. causes false increase i atinine ratio). th creatinine measure <b>I RATE:</b> <b>DESCRIPTION</b> nal kidney function	an creatinine) t of extracellu ood). ue to tubular s o creatinine). in creatinine v ment). GFR ( mL/r	lar fluid). ecretion of urea. vith certain metho nin/1.73m2 )	odologies,resul <u>ASSOCIATED</u> <u>No prote</u> Presence of	FINDINGS inuria Protein ,	I ratio when o	dehydratio
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DR.VINAY CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY) MBBS , MD (PATHOLOGY)

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST







	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbio Chairman & Consultant Pa	ology) MD	n Chopra ) (Pathology) t Pathologist
NAME	: Mrs. IPSA		
AGE/ GENDER	: 37 YRS/FEMALE	PATIENT ID	: 1676969
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 042411200001
REFERRED BY	:	<b>REGISTRATION DATE</b>	: 20/Nov/2024 11:53 AM
BARCODE NO.	: A1164444	<b>COLLECTION DATE</b>	: 20/Nov/2024 03:38PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	<b>REPORTING DATE</b>	: 20/Nov/2024 05:49PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA	CANTT	
Test Name	Va	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

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



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







	· · · · · · · · · · · · · · · · · · ·	Chopra y & Microbiology) onsultant Pathologist		m <b>Chopra</b> D (Pathology) nt Pathologist	
NAME	: Mrs. IPSA				
AGE/ GENDER	: 37 YRS/FEMALE	Р	ATIENT ID	: 1676969	
COLLECTED BY	:	R	EG. NO./LAB NO.	:042411200001	
REFERRED BY	:	R	EGISTRATION DATE	: 20/Nov/2024 11:53 AM	
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT			
Test Name		Value	Unit	Biological Refer	ence interval
		ENDOCR	INOLOGY		
	1	THYROID FUNCT	ION TEST: TOTAL		
TRIIODOTHYRONI	NE (T3): SERUM IESCENT MICROPARTICLE IMMUN	0.748 DASSAY)	ng/mL	0.35 - 1.93	
THYROXINE (T4): 5 by CMIA (CHEMILUMIN	SERUM iescent microparticle immun	9.19 DASSAY)	µgm/dl	L 4.87 - 12.60	
	ATING HORMONE (TSH): SE IESCENT MICROPARTICLE IMMUN		µIU/mI	L 0.35 - 5.50	
3rd GENERATION, ULT <u>INTERPRETATION</u> :	RASENSITIVE				
day has influence on the trilodothyronine (T3).Fai	measured serum TSH concentrations	. TSH stimulates the prod	uction and secretion of the	pm. The variation is of the order of 50 metabolically active hormones, thyro her underproduction (hypothyroidism	xine (T4)and
CLINICAL CONDITION	Т3		T4	TSH	
Primary Hypothyroidis	m: Reduce		Reduced	Increased (Significantly)	

CLINICAL CONDITION	Т3	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 (e.g.: phenytoin , salicylates).

3. Serum T4 levels 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 hypothyroidism , pregnancy , phenytoin therapy.

TRIIODOTH	YRONINE (T3)	THYROXINE (T4)		THYROID STIMULATING 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	
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00	





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

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST 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



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologi		(Pathology)
NAME	: Mrs. IPSA		
AGE/ GENDER	: 37 YRS/FEMALE	PATIENT ID	: 1676969
COLLECTED BY	:	REG. NO./LAB NO.	: 042411200001
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 20/Nov/2024 11:53 AM
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Test Name		Value Unit		t	Biological Reference interv	
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	
	RECO	MMENDATIONS OF TSH L	EVELS DURING PRE	GNANCY ( 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, iodine 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 goiter & Thyroiditis.

2. Over replacement of thyroid hormone in treatment of hypothyroidism.

3. Autonomously functioning Thyroid adenoma

4. Secondary pituitary or hypothalamic 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





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





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



KOS Diagnostic Lab (A Unit of KOS Healthcare)

	MD (Patho	<b>y Chopra</b> logy & Microbiology) & Consultant Patholog	1	gam Chopra MD (Pathology) tant Pathologist	
NAME	: Mrs. IPSA				
AGE/ GENDER	: 37 YRS/FEMALE		PATIENT ID	: 1676969	
COLLECTED BY			REG. NO./LAB NO.	: 042411200001	
	·				
REFERRED BY	:		REGISTRATION DATE		
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LIENT ADDRESS	: 6349/1, NICHOLSON R	OAD, AMBALA CANT	Т		
Test Name		Value	Unit	Biological Reference in	nterval
UTAMIN D (95 HV)		VITAMIN D/25 H	TAMINS IYDROXY VITAMIN		
by CLIA (CHEMILUMINE	DROXY VITAMIN D3): SE ESCENCE IMMUNOASSAY)	RUM <b>18.6<sup>L</sup></b>	ng/ml	L DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - SUFFICIENCY: 30.0 - 1 TOXICITY: > 100.0	
NTERPRETATION:		20			
	CIENT: CIENT:	< 20 21 - 29		ng/mL ng/mL	
	D RANGE:	30 - 100		ng/mL	
	CATION:	> 100		ng/mL	
issue and tightly bou 3. Vitamin D plays a p bosphate reabsorpti 4. Severe deficiency m <b>DECREASED:</b> 1. Lack of sunshine exi 2. Inadequate intake, 3. Depressed Hepatic 4. Secondary to advan 5. Setoporosis and So 5. Enzyme Inducing dr <b>NCREASED:</b> 1. Hypervitaminosis D Severe hypercalcemia <b>CAUTION:</b> Replaceme hypervitaminosis D	Ind by a transport protein rimary role in the mainten ion, skeletal calcium depos nay lead to failure to miner posure. malabsorption (celiac dise Vitamin D 25- hydroxylase ced Liver disease econdary Hyperparathroid ugs: anti-epileptic drugs lif ) is Rare, and is seen only a and hyperphophatemia. nt therapy in deficient indi	while in circulation. ance of calcium hom sition, calcium mobili ralize newly formed c ase) activity ism (Mild to Modera ke phenytoin, phenot fter prolonged expos viduals must be mon	eostatis. It promotes calc zation, mainly regulated b isteoid in bone, resulting te deficiency) parbital and carbamazepir sure to extremely high dos itored by periodic assessn	ansport form of Vitamin D, being stored i cium absorption, renal calcium absorptio by parathyroid harmone (PTH). in rickets in children and osteomalacia in ne, that increases Vitamin D metabolism. uses of Vitamin D. When it occurs, it can re ment of Vitamin D levels in order to preve- leficiency due to excess of melanin pigment	on and n adults. esult in ent
interefere with Vitami					





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







	Dr. Vinay Chop MD (Pathology & M Chairman & Consult	icrobiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. IPSA			
AGE/ GENDER	: 37 YRS/FEMALE	PATI	ENT ID	: 1676969
COLLECTED BY	:	REG. 1	NO./LAB NO.	: 042411200001
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			KIING DATE	: 20/NOV/2024 05:24PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
VITAMIN B12/COB	IESCENT MICROPARTICLE IMMUNOASSA	242 (Y)	pg/mL	190.0 - 890.0
NTERPRETATION:-				
	SED VITAMIN B12		DECREASED VITAMIN	I B12
INCREAS 1.Ingestion of Vitam	nin C	1.Pregnancy		
INCREAS 1.Ingestion of Vitam 2.Ingestion of Estrog	nin C gen	1.Pregnancy 2.DRUGS:Aspir	in, Anti-convulsants	
INCREAS 1.Ingestion of Vitam 2.Ingestion of Estro 3.Ingestion of Vitam	nin C gen nin A	1.Pregnancy 2.DRUGS:Aspir 3.Ethanol Igest	in, Anti-convulsants ion	
INCREAS 1.Ingestion of Vitam 2.Ingestion of Estro 3.Ingestion of Vitam 4.Hepatocellular in	nin C gen nin A jury	1.Pregnancy 2.DRUGS:Aspir 3.Ethanol Igest 4. Contraceptiv	in, Anti-convulsants ion e Harmones	
1.Ingestion of Vitam 2.Ingestion of Estrog 3.Ingestion of Vitam 4.Hepatocellular in 5.Myeloproliferativ 6.Uremia	nin C gen nin A jury	1.Pregnancy         2.DRUGS:Aspir         3.Ethanol Igest         4. Contraceptiv         5.Haemodialys         6. Multiple My	in, Anti-convulsants ion e Harmones is eloma	

proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.

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)







	<b>Dr. Vinay Ch</b> MD (Pathology & Chairman & Con	Microbiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. IPSA			
AGE/ GENDER	: 37 YRS/FEMALE	PATIEN	T ID	: 1676969
COLLECTED BY	:	REG. NO	)./LAB NO.	: 042411200001
<b>REFERRED BY</b>	:	REGIST	RATION DATE	: 20/Nov/2024 11:53 AM
BARCODE NO.	: A1164446		TION DATE	: 20/Nov/2024 03:41PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD		FING DATE	: 20/Nov/2024 06:16PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		CLINICAL PATH	OLOGY	
	URINE RO	UTINE & MICROSCO	<b>OPIC EXAMINA</b>	ATION
PHYSICAL EXAMI	NATION			
QUANTITY RECIEV		10	ml	
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	PALE YELLOW		PALE YELLOW
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY			
TRANSPARANCY by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	HAZY		CLEAR
SPECIFIC GRAVITY		1.02		1.002 - 1.030
CHEMICAL EXAMI	CTANCE SPECTROPHOTOMETRY			
REACTION		ACIDIC		
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negativo		NEGATIVE (-ve)
	CTANCE SPECTROPHOTOMETRY	Negative		
SUGAR	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
pH		6		5.0 - 7.5
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	0		
NITRITE by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLEC	by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			NEGATIVE (-ve)
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		
BLOOD by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
ASCORBIC ACID		NEGATIVE (-ve)		NEGATIVE (-ve)
MICROSCOPIC EX	CTANCE SPECTROPHOTOMETRY AMINATION			
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	<b>Biological Reference interval</b>

lest Name	value	Unit	Biological Reference interval
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-4	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	15-20	/HPF	ABSENT
CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	+		NEGATIVE (-ve)
OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT		ABSENT

\*\* End Of Report \*\*\*



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