

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

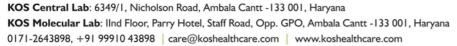


<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar			(Pathology)	
		PATIENT ID	: 1758036	
:		REG. NO./LAB NO.	: 012502150039	
:		<b>REGISTRATION DATE</b>	: 15/Feb/2025 01:57	PM
: 01525567		COLLECTION DATE	: 15/Feb/2025 02:02F	PM
: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMBA		REPORTING DATE	: 15/Feb/2025 02:31F	PM
	Value	Unit	Biological I	Reference interval
CATA CITI		LINECC DANEL, 1	F	
			5	
	LETE BLU	DOD COUNT (CBC)		
	12.8	gm/dI	120-160	
		Ű		
	4.41	Millions	/cmm 3.50 - 5.00	
	38.4	%	37.0 - 50.0	
R VOLUME (MCV)	87	fL	80.0 - 100.	0
	29	pg	27.0 - 34.0	
TOMATED HEMATOLOGY ANALYZER				
	33.3	Ŭ	32.0 - 30.0	
	13.4	%	11.00 - 16.	00
TION WIDTH (RDW-SD)	43.7	fL	35.0 - 56.0	
IOMATED HEMATOLOGY ANALYZER	19.73	RATIO	BETA THAI	LASSEMIA TRAIT: <
			13.0	CIENCY ANEMIA.
			>13.0	CIENCI ANEMIA:
X	26.41	RATIO		LASSEMIA TRAIT:<
				CIENCY ANEMIA: >
			65.0	
	6200		4000 110	00
BY SF CUBE & MICROSCOPY		/ cinffi		
· · · · ·	NIL		0.00 - 20.0	0
OOD CELLS (nRBCS) %	NIL	%	< 10 %	
TOMATED HEMATOLOGY ANALYZER				
	: 6349/1, NICHOLSON ROAD, AMBA SWASTI COMP (RBCS) COUNT AND INDICES ) BC) COUNT CUSING, ELECTRICAL IMPEDENCE ME (PCV) TOMATED HEMATOLOGY ANALYZER R VOLUME (MCV) TOMATED HEMATOLOGY ANALYZER R HAEMOGLOBIN (MCH) TOMATED HEMATOLOGY ANALYZER R HEMOGLOBIN CONC. (MCHC) TOMATED HEMATOLOGY ANALYZER TION WIDTH (RDW-CV) TOMATED HEMATOLOGY ANALYZER TION WIDTH (RDW-CV) TOMATED HEMATOLOGY ANALYZER SX EX	: 22 YRS/FEMALE : : : : : : : : : : : : :	:22 YRS/FEMALE : PATIENT ID : REG. NO./LAB NO. : REGISTRATION DATE : 01525567 COLLECTION DATE : KOS DIAGNOSTIC LAB REPORTING DATE : 6349/1, NICHOLSON ROAD, AMBALA CANTT <b>SWASTHYA WELLINESS PANEL: 1.</b> <b>COMPLETE BLOOD COUNT (CBC)</b> (RECS) COUNT AND INDICES ) 12.8 gm/dL BC) COUNT 4.41 Millions CUSING, ELECTRICAL IMPEDENCE ME (PCV) 38.4 % R HAEMOCLOBIN COUNY 87 fL TOMATED HEMATOLOGY ANALYZER R HAEMOCLOBIN (MCH) 29 pg TOMATED HEMATOLOGY ANALYZER R HAEMOCLOBIN (MCH) 29 ANALYZER R HAEMOCLOBIN (MCH) 29 NG TOMATED HEMATOLOGY ANALYZER R HAEMOCLOBIN (MCH) 29 NG TOMATED HEMATOLOGY ANALYZER R HAEMOCLOBIN (MCH) 29 NG TOMATED HEMATOLOGY ANALYZER TION WIDTH (RDW-CV) 13.4 % TOMATED HEMATOLOGY ANALYZER TON WIDTH (RDW-CV) 26.41 RATIO XX 26.41 RATIO XX 26.41 RATIO XX 26.41 RATIO	::::::::::::::::::::::::::::::::::::





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









Dr. Vinay Chopra Dr. M MD (Pathology & Microbiology) Chairman & Consultant Pathologist CEO & Con PREET KAUR

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

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NAME	: Miss. MANPREET KAUR		
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Value	Unit	<b>Biological Reference interval</b>
64	%	50 - 70
30	%	20 - 40
1		1 - 6
5		2 - 12
0	%	0 - 1
4090	/cmm	2000 - 7500
1917	/cmm	800 - 4900
64	/cmm	40 - 440
	/cmm	80 - 880
		0 - 110
	/cmm	0.0 - 999.0
MARKERS.		
235000	/cmm	150000 - 450000
		0.10 - 0.36
11	fL	6.50 - 12.0
78000	/cmm	30000 - 90000
33.3	%	11.0 - 45.0
16.3	%	15.0 - 17.0
	64 30 1 5 0 4090 1917 64 320 0 0 0 0 <b>MARKERS.</b> 235000 0.26 11 78000 33.3	64       %         30       %         30       %         1       %         5       %         0       %         4090       /cmm         1917       /cmm         64       /cmm         320       /cmm         0       /cmm         0       /cmm         0       /cmm         0       /cmm         0       /cmm         11       fL         78000       /cmm         33.3       %





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Test Name	Value	Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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B NO. : 01	ogy) ogist 58036
B NO. : 01	
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	0500150000
	2502150039
<b>ON DATE</b> : 15/	/Feb/2025 01:57 PM
<b>DATE</b> : 15/	/Feb/2025 02:02PM
	/Feb/2025 04:10PM
Unit	Biological Reference interva
% mg/dL	4.0 - 6.4
	in %
>8.	.0
e < 19 Years	
	DATE : 15/ Unit IN (HBA1C)

**KOS Diagnostic Lab** 

(A Unit of KOS Healthcare)

## COMMENTS:

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 CODE.	: KOS DIAGNOSTIC LAB	REPORTI	NG DATE	: 15/Feb/2025 02:45PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
ERYTHROCYTE SE by RED CELL AGGRE	GATION BY CAPILLARY PHOTOMET	7 RY		hr 0 - 20





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			k Microbiology) sultant Pathologist		(Pathology) Pathologist
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CLIENT ADDRESS	: 6349/1, NICH	OLSON ROAD,	AMBALA CANTT		
Test Name			Value	Unit	Biological Reference interval
		CLINIC	CAL CHEMIST	<b>RY/BIOCHEMIST</b>	'RY
			GLUCOSE	FASTING (F)	
GLUCOSE FASTING	G (F): PLASMA SE - PEROXIDASE (GO		100.6 <sup>H</sup>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

**IN ACCRDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:** 1. A fasting plasma glucose level below 100 mg/dl is considered normal. 2. 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. 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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SU 9001 : 2008 CERT	IFIED LAB		EXCELLENCE IN HEALTHCARE	a DIAGNOSTICS
		Chopra • & Microbiology) onsultant Pathologi		(Pathology)
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Test Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PR	OFILE : BASIC	
CHOLESTEROL TO by CHOLESTEROL O		175.68	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSE	ERUM PHATE OXIDASE (ENZYMATIC)	85.76	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 10N	51.68	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERO		106.85	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLES'		124	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTER(		17.15	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEF by CALCULATED, SPE	RUM	437.12	mg/dL	350.00 - 700.00
CHOLESTEROL/HI by CALCULATED, SPE	DL RATIO: SERUM	3.4	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0



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Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		2.07	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	IDL RATIO: SERUM	1.66 <sup>L</sup>	RATIO	3.00 - 5.00

<u>INTERPRETATION:</u> 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. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Miss. MANPREET KAUR AGE/ GENDER : 22 YRS/FEMALE **PATIENT ID** :1758036 **COLLECTED BY** REG. NO./LAB NO. :012502150039 : **REFERRED BY REGISTRATION DATE** : 15/Feb/2025 01:57 PM : **BARCODE NO.** :01525567 **COLLECTION DATE** :15/Feb/202502:02PM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :15/Feb/202503:50PM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** 

LIVER	FUNCTION TEST (CO	MPLETE)	
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.42	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.11	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by calculated, spectrophotometry	0.31	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	19.3	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	25.1	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by calculated, spectrophotometry	0.77	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	71.87	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by szasz, spectrophtometry	18.32	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.82	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.17	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.65	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by calculated, spectrophotometry	1.57	RATIO	1.00 - 2.00

#### **INTERPRETATION**

**NOTE:** To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range. USE: Differential diagnosis of diseases of hepatobiliary system and pancreas.

# INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)



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	Dr. Vinay Cho		m Chopra

### 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	<b>Biological Reference interval</b>
	KIDNE	Y FUNCTION	TEST (COMPLETE)	
UREA: SERUM		18.07	mg/dL	10.00 - 50.00
by UREASE - GLUTAN CREATININE: SERI	MATE DEHYDROGENASE (GLDH)	0.84	ma (dI	0.40 - 1.20
by ENZYMATIC, SPEC		0.84	mg/dL	0.40 - 1.20
	OGEN (BUN): SERUM	8.44	mg/dL	7.0 - 25.0
by CALCULATED, SPE	CCTROPHOTOMETRY ROGEN (BUN)/CREATININE	10.05	RATIO	10.0 - 20.0
RATIO: SERUM	COULIN (DOIN)/ CREATININE	10.05	RAHO	10.0 - 20.0
by CALCULATED, SPE				
UREA/CREATININ by CALCULATED, SPE		21.51	RATIO	
URIC ACID: SERUM	[	4.84	mg/dL	2.50 - 6.80
by URICASE - OXIDAS	E PEROXIDASE	0.54		9.50, 10.00
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.54	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE		2.68	mg/dL	2.30 - 4.70
by PHOSPHOMOLYBE <u>ELECTROLYTES</u>	DATE, SPECTROPHOTOMETRY			
SODIUM: SERUM		142.1	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV	'E ELECTRODE)	142.1	IIIII01/ L	133.0 - 130.0
POTASSIUM: SERU		4.23	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV CHLORIDE: SERUM		106.57	mmol/L	90.0 - 110.0
by ISE (ION SELECTIV	'E ELECTRODE)	100.07		00.0 110.0
ESTIMATED GLOM	IERULAR FILTERATION RATE			
	ERULAR FILTERATION RATE	100.7		
(eGFR): SERUM by CALCULATED				
INTERPRETATION				

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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT			
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7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia	kia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine produ tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately r	LEVELS:			
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r. 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther	(e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately r superimposed on renal disease. <b>D:1) WITH DECREASED BUN :</b> Disis. d starvation. creased urea synthesis. urea rather than creatinine diffi- monemias (urea is virtually abse- f inappropiate antidiuretic harm <b>D:1) WITH INCREASED CREATINII</b> Dy (accelerates conversion of cre- eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r LAR FILTERATION RATE: <u>DESCRIPTION</u> <u>Normal kidney func-</u> Kidney damage wi	action)         ELEVELS:         hore than creatinine) (e.         uses out of extracellular         ent in blood).         hone) due to tubular sec         JE:         eatine to creatinine).         crease in creatinine with         heasurement).         GFR (mL/minimed to be available of the system)         the system	g. obstructive urop fluid). retion of urea.	ogies,resulting in norma SSOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydrat
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r- 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1 G2	(e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately r superimposed on renal disease. <b>D:1) WITH DECREASED BUN :</b> Disis. d starvation. creased urea synthesis. urea rather than creatinine diffi- monemias (urea is virtually abse- f inappropiate antidiuretic harm <b>D:1) WITH INCREASED CREATINII</b> Dy (accelerates conversion of cre- eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r LAR FILTERATION RATE: <u>DESCRIPTION</u> <u>Normal kidney func- Kidney damage wi- normal or high Gf</u>	action)         ELEVELS:         hore than creatinine) (e.         uses out of extracellular         ent in blood).         hone) due to tubular sec         JE:         eatine to creatinine).         crease in creatinine with         heasurement).         Crease in creatinine with         GFR (mL/minimed)         th         >900         R	g. obstructive urop fluid). retion of urea.	athy). ogies,resulting in norma SSOCIATED FINDINGS No proteinuria	al ratio when dehydrat
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r- 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERU CKD STAGE G1 G2 G3a	(e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately r superimposed on renal disease. <b>D:1) WITH DECREASED BUN :</b> Disis. d starvation. creased urea synthesis. urea rather than creatinine diffi- monemias (urea is virtually abso- f inappropiate antidiuretic harm <b>D:1) WITH INCREASED CREATINII</b> Dy (accelerates conversion of cre- eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r <b>LAR FILTERATION RATE:</b> DESCRIPTION Normal kidney func- Kidney damage wi normal or high GF Mild decrease in G	action)         ELEVELS:         nore than creatinine) (e.         uses out of extracellular         ent in blood).         none) due to tubular sec         JE:         eatine to creatinine).         crease in creatinine wit         neasurement).         Characteristic (mL/min)         th         >900         R         FR       60 - 80	g. obstructive urop fluid). retion of urea.	ogies,resulting in norma SSOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydrat
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r- 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1 G2	(e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>D:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately r superimposed on renal disease. <b>D:1) WITH DECREASED BUN :</b> Disis. d starvation. creased urea synthesis. urea rather than creatinine diffi- monemias (urea is virtually abse- f inappropiate antidiuretic harm <b>D:1) WITH INCREASED CREATINII</b> Dy (accelerates conversion of cre- eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r LAR FILTERATION RATE: <u>DESCRIPTION</u> <u>Normal kidney func- Kidney damage wi- normal or high Gf</u>	action) <b>ELEVELS:</b> nore than creatinine) (e.         uses out of extracellular         ent in blood).         none) due to tubular sec <b>JE:</b> eatine to creatinine).         crease in creatinine wit         neasurement). <b>GFR (mL/min</b> tion       >90         th       >90         R       60 - &         n GFR       30-5	g. obstructive urop fluid). retion of urea. h certain methodol	ogies,resulting in norma SSOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydrat





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









	Dr. Vinay Chopr MD (Pathology & Micr Chairman & Consultar	robiology) MI	m Chopra D (Pathology) nt Pathologist
NAME	: Miss. MANPREET KAUR		
AGE/ GENDER	: 22 YRS/FEMALE	PATIENT ID	: 1758036
COLLECTED BY	:	REG. NO./LAB NO.	: 012502150039
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 15/Feb/2025 01:57 PM
BARCODE NO.	: 01525567	COLLECTION DATE	: 15/Feb/2025 02:02PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 15/Feb/2025 03:50PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CANTT	
Test Name		Value Unit	<b>Biological Reference interval</b>

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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MBBS, MD (PATHOLOGY)







	<b>Dr. Vinay Chop</b> MD (Pathology & M Chairman & Consult	icrobiology)		(Pathology)
NAME	: Miss. MANPREET KAUR			
AGE/ GENDER	: 22 YRS/FEMALE		PATIENT ID	: 1758036
COLLECTED BY	:		REG. NO./LAB NO.	:012502150039
<b>REFERRED BY</b>	:		<b>REGISTRATION DATE</b>	: 15/Feb/2025 01:57 PM
BARCODE NO.	: 01525567		<b>COLLECTION DATE</b>	: 15/Feb/2025 02:02PM
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT		
Test Name		Value	Unit	<b>Biological Refe</b>
		IRON	PROFILE	
IRON: SERUM by FERROZINE, SPEC	TROPHOTOMETRY	66.32	μg/dL	37.0 - 145.0
•	ON BINDING CAPACITY (UIBC)	259.98	μg/dL	150.0 - 336.0

Test Name	Value	Unit	<b>Biological Reference interval</b>
	IRON PRO	FILE	
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	66.32	μg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY	259.98	μg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETERY	326.3	μg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	20.32	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE) INTERPRETATION:-	231.67	mg/dL	200.0 - 350.0
VARIABLES ANEMIA OF CHRO	NIC DISEASE IRO	N DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT

VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased
IDON.			

IRON:

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.
 It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.
 **TOTAL IRON BINDING CAPACITY (TIBC):** It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT





		hopra & Microbiology) nsultant Pathologist	Dr. Yugam Cl MD (Pat CEO & Consultant Patl	hology)	
NAME	: Miss. MANPREET KAUR				
AGE/ GENDER	: 22 YRS/FEMALE	PATIE	NT ID :	1758036	
COLLECTED BY	:	REG. N	IO./LAB NO. :	012502150039	
REFERRED BY	:	REGIS	TRATION DATE :	15/Feb/2025 01:57 PM	
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT			
Test Name		Value	Unit	Biological Reference i	nterval
		ENDOCRINO	LOGY		
	T	HYROID FUNCTION	TEST: TOTAL		
TRIIODOTHYRONI	NE (T3): SERUM IESCENT MICROPARTICLE IMMUNO.	1.021 ASSAY)	ng/mL	0.35 - 1.93	
THYROXINE (T4): S	SERUM iescent microparticle immuno.	7.92 ASSAY)	µgm/dL	4.87 - 12.60	
	ATING HORMONE (TSH): SER		µIU/mL	0.35 - 5.50	
3rd GENERATION, ULT	RASENSITIVE				
INTERPRETATION:					
day has influence on the triiodothyronine (T3).Fai		TSH stimulates the production	and secretion of the metab	e variation is of the order of 50%.Hence blically active hormones, thyroxine (T4 derproduction (hypothyroidism) or	
CLINICAL CONDITION	T3	T4		TSH	

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 (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 (TS	
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)







	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbi Chairman & Consultant P		(Pathology)	
NAME	: Miss. MANPREET KAUR			
AGE/ GENDER	: 22 YRS/FEMALE	PATIENT ID	: 1758036	
<b>COLLECTED BY</b>	:	<b>REG. NO./LAB NO.</b>	: 012502150039	
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 15/Feb/2025 01:57 PM	
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA	A CANTT		

Test Name		Value Unit		t	Biological Reference interval	
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





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



		<b>Chopra</b> gy & Microbiology) Consultant Pathologi	MD	n Chopra 9 (Pathology) t Pathologist	
NAME	: Miss. MANPREET KAUR				
AGE/ GENDER	: 22 YRS/FEMALE		PATIENT ID	: 1758036	
COLLECTED BY	:		REG. NO./LAB NO.	: 012502150039	
REFERRED BY	:		REGISTRATION DATE	: 15/Feb/2025 01:5'	
BARCODE NO.	: 01525567		COLLECTION DATE	: 15/Feb/2025 02:02	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 15/Feb/2025 03:38	BPM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	AD, AMBALA CANT	Г		
Test Name		Value	Unit	Biological	Reference interval
		VE	TAMINS		
	VI		I AMIN'S IYDROXY VITAMIN D	13	
	DROXY VITAMIN D3): SER SCENCE IMMUNOASSAY)		ng/mL	DEFICIEN INSUFFIC SUFFICIE	CY: < 20.0 IENCY: 20.0 - 30.0 NCY: 30.0 - 100.0
INTERPRETATION:				TOXICITY	: > 100.0
DEFIC	IENT:	< 20	r	ng/mL	
INSUFF		21 - 29		ng/mL	
PREFFERE				ng/mL	
conversion of 7- dihyc 2.25-OHVitamin D re- tissue and tiahtly bou 3.Vitamin D plays a pr phosphate reabsorptii 4.Severe deficiency m <b>DECREASED:</b> 1.Lack of sunshine exr 2.Inadequate intake, 1 3.Depressed Hepatic N 4.Secondary to advanc 5.Osteoporosis and Se 6.Enzyme Inducing dru <b>INCREASED:</b> 1. Hypervitaminosis D severe hypercalcemia <b>CAUTION:</b> Replacemer hypervitaminosis D	malabsorption (celiac diseas /itamin D 25- hvdroxylase ac ced Liver disease condary Hyperparathroidisr ugs: anti-epileptic drugs like is Rare, and is seen only aft and hyperphophatemia. It therapy in deficient indivio individuals as compare to whi	n D3 in the skin upon evoir and transport inle in circulation. ice of calcium home ion, calcium mobiliz ize newly formed os civity n (Mild to Moderat phenytoin, phenob er prolonged expose duals must be monit	n plants, Vitamin D2), or che n Ultraviolet exposure. form of Vitamin D and trans eostatis. It promotes calciu vation, mainly regulated by steoid in bone, resulting in e deficiency) arbital and carbamazepine, ure to extremely high doses tored by periodic assessme	Decalciferol (from anim sport form of Vitamin D, m absorption, renal calc parathyroid harmone (F rickets in children and c that increases Vitamin s of Vitamin D. When it c nt of Vitamin D levels in	being stored in adipose cium absorption and DTH). Isteomalacia in adults. D metabolism. D cccurs, it can result in order to prevent

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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		hopra & Microbiology) onsultant Pathologis		(Pathology)
IAME	: Miss. MANPREET KAUR			
AGE/ GENDER	: 22 YRS/FEMALE		PATIENT ID	: 1758036
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	), AMBALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
TTAMIN B19/COD		199.88	ng/mI	190.0 - 830
by CMIA (CHEMILUMIN	BALAMIN: SERUM IESCENT MICROPARTICLE IMMUNO,		pg/mL	130.0 - 830
by CMIA (CHEMILUMIN NTERPRETATION:- INCREAS	IESCENT MICROPARTICLE IMMUNO, SED VITAMIN B12		DECREASED VITAMII	
by CMIA (CHEMILUMIN <u>NTERPRETATION:-</u> INCREAS 1.Ingestion of Vitan	IESCENT MICROPARTICLE IMMUNO, SED VITAMIN B12 nin C	ASSAY)	DECREASED VITAMII	N B12
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS 1.Ingestion of Vitan 2.Ingestion of Estro	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 hin C gen	ASSAY) 1.Pregn 2.DRUG	DECREASED VITAMII ancy S:Aspirin, Anti-convulsants	N B12
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS 1.Ingestion of Vitan 2.Ingestion of Estro 3.Ingestion of Vitan	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 hin C gen hin A	ASSAY) 1.Pregn 2.DRUG 3.Ethan	DECREASED VITAMII ancy S:Aspirin, Anti-convulsants ol Igestion	N B12
by CMIA (CHEMILUMIN <u>INTERPRETATION:-</u> INCREAS 1.Ingestion of Vitan 2.Ingestion of Vitan 4.Hepatocellular in	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 hin C gen hin A jury	ASSAY) 1.Pregn 2.DRUG 3.Ethan 4. Contr	DECREASED VITAMII ancy S:Aspirin, Anti-convulsants ol Igestion aceptive Harmones	N B12
by CMIA (CHEMILUMIN <u>INTERPRETATION:-</u> <u>INCREAS</u> 1.Ingestion of Vitan 2.Ingestion of Vitan 4.Hepatocellular in 5.Myeloproliferativ 6.Uremia	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 hin C gen hin A jury	ASSAY) 1.Pregn 2.DRUG 3.Ethan 4. Contr 5.Haem 6. Multi	DECREASED VITAMII ancy S:Aspirin, Anti-convulsants ol Igestion aceptive Harmones odialysis ple Myeloma	N B12



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

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	Dr. Vinay Cho MD (Pathology & Chairman & Const	Microbiology)	Dr. Yugam MD 50 & Consultant	(Pathology)	
NAME : Miss. MAN	IPREET KAUR				
AGE/ GENDER : 22 YRS/FE	MALE	PATIENT	ID	: 1758036	
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CLIENT CODE. : KOS DIAGN		REPORTING DATE		: 15/Feb/2025 02:21PM	
CLIENT ADDRESS : 6349/1, NI	ICHOLSON ROAD, A	IMBALA CAN I I			
Test Name		Value	Unit	Biological Reference interval	
		CLINICAL PATHO	LOGY		
	URINE ROU	UTINE & MICROSCOI	PIC EXAMINA	ATION	
PHYSICAL EXAMINATION					
QUANTITY RECIEVED		10	ml		
by DIP STICK/REFLECTANCE SPECTR COLOUR	OPHOTOMETRY	PALE YELLOW		PALE YELLOW	
by DIP STICK/REFLECTANCE SPECTR TRANSPARANCY	OPHOTOMETRY	CLEAR		CLEAR	
by DIP STICK/REFLECTANCE SPECTR	OPHOTOMETRY	ULEAK			
SPECIFIC GRAVITY by DIP STICK/REFLECTANCE SPECTR	OPHOTOMETRY	1.02		1.002 - 1.030	
CHEMICAL EXAMINATION	of high of mental				
REACTION		ACIDIC			
by DIP STICK/REFLECTANCE SPECTR PROTEIN	OPHOTOMETRY	Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTR	OPHOTOMETRY	-			
SUGAR by DIP STICK/REFLECTANCE SPECTR	OPHOTOMETRY	Negative		NEGATIVE (-ve)	
pH by DIP STICK/REFLECTANCE SPECTR		5.5		5.0 - 7.5	
BILIRUBIN		Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTR NITRITE	OPHOTOMETRY	Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTR	OPHOTOMETRY.				
UROBILINOGEN by DIP STICK/REFLECTANCE SPECTR	OPHOTOMETRY	Normal	EU/dL	0.2 - 1.0	
KETONE BODIES		Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTR BLOOD	UFAU I UIVIE I KY	Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTR ASCORBIC ACID	OPHOTOMETRY			NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTR	OPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-Ve)	
MICROSCOPIC EXAMINATION					
RED BLOOD CELLS (RBCs)		NEGATIVE (-ve)	/HPF	0 - 3	



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thopsa

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

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

NAME	: Miss. MANPREET KAUR			
AGE/ GENDER	: 22 YRS/FEMALE	]	PATIENT ID	: 1758036
COLLECTED BY	:	]	REG. NO./LAB NO.	: 012502150039
<b>REFERRED BY</b>	:	]	REGISTRATION DATE	: 15/Feb/2025 01:57 PM
BARCODE NO.	: 01525567		COLLECTION DATE	: 15/Feb/2025 02:02PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	1	REPORTING DATE	: 15/Feb/2025 02:21PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AN	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
by MICROSCOPY ON O	CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON (	CENTRIFUGED URINARY SEDIMENT	1-2	/HPF	0 - 5
FDITHFILM CELL	2	2-1	/HDE	ABSENT

2-4	/HPF	ABSENT
NEGATIVE (-ve)		NEGATIVE (-ve)
ABSENT		ABSENT
	NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve)	NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve)

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



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