A PIONEER DIAGNOSTIC CENTRE

【 0171-2532620, 8222896961 🛛 🖾 pkrjainhealthcare@gmail.com

NAME	: Miss. KIRANPREET KAUR				
AGE/ GENDER	: 15 YRS/FEMALE		PATIENT ID	: 1747441	
COLLECTED BY	:		REG. NO./LAB NO.	: 122502060012	
REFERRED BY	:		<b>REGISTRATION DATE</b>	: 06/Feb/2025 11:45 AM	
BARCODE NO.	: 12506863		COLLECTION DATE	: 06/Feb/2025 12:04PM	
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITU	TE	<b>REPORTING DATE</b>	:06/Feb/202502:15PM	
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBAL	A CITY - HA	ARYANA		
Test Name		Value	Unit	<b>Biological Reference</b>	interval
	SWAST	HYA WE	ELLNESS PANEL: 1.4	ł	
	СОМР	LETE BI	OOD COUNT (CBC)		
RED BLOOD CELLS	(RBCS) COUNT AND INDICES		()		
HAEMOGLOBIN (H		13.2	gm/dL	12.0 - 16.0	
RED BLOOD CELL (	RBC) COUNT	4.89	Millions/	cmm 3.50 - 5.00	
PACKED CELL VOLU		39.3	%	35.0 - 49.0	
MEAN CORPUSCUL	AR VOLUME (MCV) utomated hematology analyzer	80.5	KR fl	80.0 - 100.0	
	AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	27	pg	27.0 - 34.0	
	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	33.6	g/dL	32.0 - 36.0	
	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	13.3	%	11.00 - 16.00	
	UTION WIDTH (RDW-SD) utomated hematology analyzer	40.9	fL	35.0 - 56.0	
MENTZERS INDEX by CALCULATED		16.46	RATIO	BETA THALASSEMIA 13.0 IRON DEFICIENCY AI >13.0	
GREEN & KING INE by CALCULATED	DEX	21.9	RATIO	BETA THALASSEMIA 65.0 IRON DEFICIENCY AI 65.0	
WHITE BLOOD CE	LLS (WBCS)				
TOTAL LEUCOCYTE	COUNT (TLC) / by sf cube & microscopy	5430	/cmm	4000 - 11000	
	<u>UCOCYTE COUNT (DLC)</u>				
NEUTROPHILS	' BY SF CUBE & MICROSCOPY	47 <sup>L</sup>	%	50 - 70	

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

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Test Name		Value	Unit	Biological Reference interval
LYMPHOCYTES by FLOW CYTOMETRY	Y BY SF CUBE & MICROSCOPY	41 <sup>H</sup>	%	20 - 40
EOSINOPHILS	Y BY SF CUBE & MICROSCOPY	5	%	1 - 6
MONOCYTES by flow cytometry	Y BY SF CUBE & MICROSCOPY	7	%	2 - 12
	Y BY SF CUBE & MICROSCOPY CYTES (WBC) COUNT	0	%	0 - 1
ABSOLUTE NEUTR	OPHIL COUNT ( by sf cube & microscopy	2552	/cmm	2000 - 7500
ABSOLUTE LYMPH	OCYTE COUNT Y BY SF CUBE & MICROSCOPY	2226 <sup>L</sup>	CR /cmm	800 - 4900
ABSOLUTE EOSINC by FLOW CYTOMETRY	PHIL COUNT y by sf cube & microscopy	272	/cmm	40 - 440
	Y BY SF CUBE & MICROSCOPY	380	/cmm	80 - 880
,	Y BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
<u>PLATELETS AND O</u>	THER PLATELET PREDICTIVE	<u>MARKERS.</u>		
PLATELET COUNT by hydro dynamic f	(PLT) FOCUSING, ELECTRICAL IMPEDENCE	325000	/cmm	150000 - 450000
PLATELETCRIT (PC by HYDRO DYNAMIC F	CT) FOCUSING, ELECTRICAL IMPEDENCE	0.33	%	0.10 - 0.36
	OCUSING, ELECTRICAL IMPEDENCE	10	fL	6.50 - 12.0
by HYDRO DYNAMIC F	CELL COUNT (P-LCC)	96000 <sup>H</sup>	/cmm	30000 - 90000
by HYDRO DYNAMIC F	CELL RATIO (P-LCR) COCUSING, ELECTRICAL IMPEDENCE	29.5	%	11.0 - 45.0
by HYDRO DYNAMIC F	BUTION WIDTH (PDW) FOCUSING, ELECTRICAL IMPEDENCE	15.9	%	15.0 - 17.0
NOTE: TEST CONDU	CTED ON EDTA WHOLE BLOOD			



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CLIENT CODE.	: P.K.R JAIN HEALTHCARE INS	TITUTE <b>REPOI</b>	RTING DATE	:06/Feb/202504:42PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AN	MBALA CITY - HARYANA		
Test Name		Value	Unit	Biological Reference interval
CI VCOSVI ΑΤΈΡ ΠΑΈ	GLY MOGLOBIN (HbA1c):	5.1	LUBIN (HBAIC) %	4.0 - 6.4
		COSYLATED HAEMOG		40.04
	MANCE LIQUID CHROMATOGRAPHY)			
ESTIMATED AVERAG	E PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY)	99.67	mg/dL	60.00 - 140.00
INTERPRETATION:				
	AS PER AMERICAN DIAE	BETES ASSOCIATION (ADA):		
RE	FERENCE GROUP		EMOGLOGIB (HBAIC) in S	%
	etic Adults >= 18 years		<5.7	
	Risk (Prediabetes)		<mark>5.7 – 6</mark> .4	
Dia	gnosing Diabetes		>= 6.5	
			> 19 Years	
Thorapoutic	goals for glycemic control	Goals of Therapy:	< 7.0	
merapeutic	goals for grycernic control	Actions Suggested:	>8.0	
		Goal of therapy:	< 19 Years <7.5	
COMMENTS:		Guar ur trierapy:	<7.5	

#### 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.





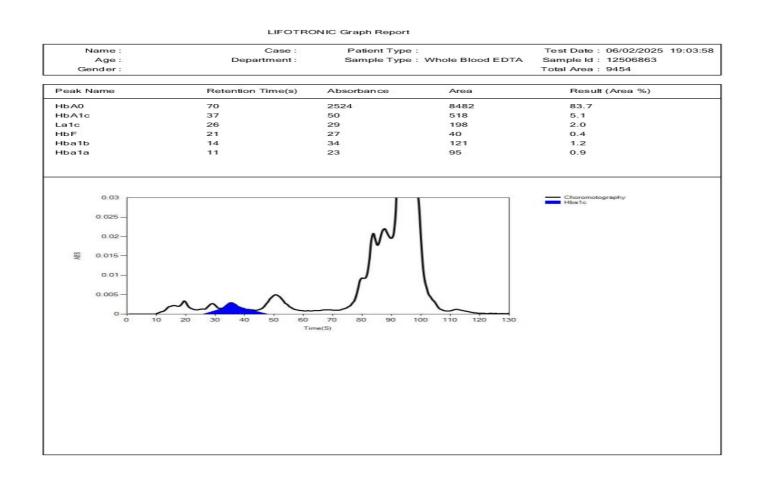
DR.VINAY CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY	HARYANA	
Test Name	Value	Unit	Biological Reference interval
	DIMENTATION RATE (ESR) <b>21<sup>H</sup></b>	mm/1st	hr 0 - 20
by RED CELL AGGRE	GATION BY CAPILLARY PHOTOMETRY		
by RED CELL AGGREG 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	GATION BY CAPILLARY PHOTOMETRY ic test because an elevated result often indica does not tell the health practitioner exactly w cted by other conditions besides inflammation be used to monitor disease activity and respo	ites the presence of inflammati here the inflammation is in the n. For this reason, the ESR is typ	on associated with infection, cancer and auto body or what is causing it. bically used in conjunction with other test suc
by RED CELL AGGREG INTERPRETATION: 1. ESR is a non-specifimmune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythe CONDITION WITH LOI A low ESR can be see (polycythaemia), sigr	GATION BY CAPILLARY PHOTOMETRY ic test because an elevated result often indica does not tell the health practitioner exactly w cted by other conditions besides inflammation be used to monitor disease activity and respo	ites the presence of inflammati where the inflammation is in the n. For this reason, the ESR is typ nse to therapy in both of the al	on associated with infection, cancer and auto body or what is causing it. bically used in conjunction with other test suc bove diseases as well as some others, such as uch as a high red blood cell count

5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.

6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it





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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, A	MBALA CITY - HARY	ANA	
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CT INI	сат спемісті	RY/BIOCHEMIST	<b>'DV</b>
	CLINI			.K1
		GLUCOSE F.	ASTING (F)	
GLUCOSE FASTING by GLUCOSE OXIDAS	G (F): PLASMA e - peroxidase (god-pod)	71.57	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0
INTERPRETATION	H AMERICAN DIABETES ASSOCIA			
	lucose level below 100 mg/dl is			

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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Test Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PR	OFILE : BASIC	
CHOLESTEROL TO' by CHOLESTEROL O		201.66 <sup>H</sup>	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSF	ERUM PHATE OXIDASE (ENZYMATIC)	365.81 <sup>H</sup>	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERO by SELECTIVE INHIBIT	L (DIRECT): SERUM TON	35.79	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERO		92.71	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLES' by Calculated, spe		165.87 <sup>H</sup>	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTER		73.16 <sup>H</sup>	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEF	RUM	769.13 <sup>H</sup>	mg/dL	350.00 - 700.00
CHOLESTEROL/HI by CALCULATED, SPE	DL RATIO: SERUM	5.63 <sup>H</sup>	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT

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Test Name	Value	Unit	Biological Reference interval
LDL/HDL RATIO: SERUM by Calculated, spectrophotometry	2.59	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	10.22 <sup>H</sup>	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
	LIVER	FUNCTIC	ON TEST (COMPLETE)	
BILIRUBIN TOTAL: by DIAZOTIZATION, SF		0.55	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.12	mg/dL	0.00 - 0.40
	CT (UNCONJUGATED): SERUM	0.43	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	RIDOXAL PHOSPHATE	21.87	U/L	7.00 - 45.00
SGPT/ALT: SERUM	RIDOXAL PHOSPHATE	17.56	U/L	0.00 - 49.00
AST/ALT RATIO: SI	ERUM	1. <mark>25</mark>	RATIO	0.00 - 46.00
ALKALINE PHOSPH		135.12	U/L	50.00 - 370.00
GAMMA GLUTAMY	L TRANSFERASE (GGT): SERUM	16.69	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.36	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G	REEN	4.16	gm/dL	3.50 - 5.50
GLOBULIN: SERUM	I	2.2 <sup>L</sup>	gm/dL	2.30 - 3.50

A : G RATIO: SERUM 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)

1.89





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RATIO

1.00 - 2.00

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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).

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6



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	i remerion repr (e		
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	17.39	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	0.48	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	8.13	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	16.94	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	36.23	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	3.32	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY	9.54	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM by phosphomolybdate, spectrophotometry	3.26	mg/dL	2.30 - 4.70
<u>ELECTROLYTES</u>			
SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	141.8	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.39	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	106.35	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RATE			
ESTIMATED GLOMERULAR FILTERATION RATE	143.4		

(eGFR): SERUM

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.

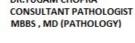
2. Catabolic states with increased tissue breakdown.

3. GI haemorrhage.



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

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST





A PIONEER DIAGNOSTIC CENTRE

🔽 0171-2532620, 8222896961 🛛 🖾 pkrjainhealthcare@gmail.com

	: Miss. KIRANPREET KAUR		
AGE/ GENDER	: 15 YRS/FEMALE	PATIENT ID	: 1747441
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<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 06/Feb/2025 11:45 AM
BARCODE NO.	: 12506863	COLLECTION DATE	: 06/Feb/2025 12:04PM
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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CIT	ГҮ - HARYANA	
	<b>T</b> 7 <b>T</b>		
Test Name	Valu	ue Unit	<b>Biological Reference interval</b>
4. High protein intake 5. Impaired renal fun	ction plus		U
4. High protein intake 5. Impaired renal fun 6. Excess protein inta	ction plus ke or production or tissue breakdown (e.g.		<b>Biological Reference interval</b> osis, Cushing's syndrome, high protein diet,
4. High protein intake 5. Impaired renal fun 6. Excess protein inta burns, surgery, cache	ction plus ke or production or tissue breakdown (e.g.		U
<ol> <li>High protein intake</li> <li>Impaired renal fun</li> <li>Excess protein inta burns, surgery, cache</li> <li>Urine reabsorption</li> <li>Reduced muscle m</li> </ol>	tion plus ke or production or tissue breakdown (e.g. xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine production)		U
<ol> <li>High protein intake</li> <li>Impaired renal fun</li> <li>Excess protein inta burns, surgery, cache</li> <li>Urine reabsorption</li> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> </ol>	e. ction plus ke or production or tissue breakdown (e.g. xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine production) tetracycline, glucocorticoids)		C C
4. High protein intake 5. Impaired renal fun 6. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2	e. ction plus ke or production or tissue breakdown (e.g. xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine production) tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LEVELS:	infection, GI bleeding, thyrotoxic	osis, Cushing's syndrome, high protein diet,
<ol> <li>High protein intake</li> <li>Impaired renal fun</li> <li>Excess protein inta burns, surgery, cache</li> <li>Urine reabsorption</li> <li>Reduced muscle m</li> <li>Certain drugs (e.g. INCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> </ol>	e. ction plus ke or production or tissue breakdown (e.g. xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine production) tetracycline, glucocorticoids)	infection, GI bleeding, thyrotoxic	osis, Cushing's syndrome, high protein diet,

1. Acute tubular necrosis.

2. Low protein diet and starvation.

3. Severe liver disease.

4. Other causes of decreased urea synthesis.

5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).

6. Inherited hyperammonemias (urea is virtually absent in blood).

7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.

8. Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

1. Phenacimide therapy (accelerates conversion of creatine to creatinine).

2. Rhabdomyolysis (releases muscle creatinine).

3. Muscular patients who develop renal failure.

#### **INAPPROPIATE RATIO:**

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).

2. Cephalosporin therapy (interferes with creatinine measurement). ESTIMATED GLOMERULAR FILTERATION RATE:

CKD STAGE	DESCRIPTION	GFR ( mL/min/1.73m2 )	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with	>90	Presence of Protein ,
	normal or high GFR		Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	



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

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





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0171-2532620, 8222896961 🛛 🖾 pkrjainhealthcare@gmail.com

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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA				

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

COMMENTS:

1. 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. 2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012

3. 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 eGFR with Cystatin C for confirmation of CKD

4. eGFR category G1 OR G2 does not fullfill 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)

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

lest name	value	Unit	Biological Reference inte
	IRON PR	OFILE	
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	75.06	µg/dL	50.0 - 170.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY	313.55	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) SERUM by SPECTROPHOTOMETERY	388.61	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	19.31	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)	275.91	mg/dL	200.0 - 350.0
INTERPRETATION:-			

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):

1. 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.





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

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





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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AN	MBALA CITY - HAR	ZYANA		
Test Name		Value	Unit	<b>Biological Reference interval</b>	
	LACTA	<b>FE DEHYDRO</b>	GENASE (LDH): SER	UM	
by BASED ON SCE, SI INTERPRETATION:-	OGENASE (LDH): SERUM PECTROPHOTOMETRY	447.7	U/L	225.0 - 450.0	
1.Lactate dehydrogei erythrocytes.	hase (LDH) activity is present in a	ll cells of the body	with highest concentration	ons in heart, liver, muscle, kidney, lung, and	
2. The test can be use	d for monitoring changes in tumo c to be of use in the diagnosis of		emotherapy, although, lac	tate dehydrogenase elevations in patients with	
INCREASED (MARKED 1.Megaloblastic aner					
2.Untreated pernicio					
3.Hodgkins disease.					
4.Abdominal and lun 5.Severe shock.	g cancers.				
6.Hypoxia.					
INCREASED (MODERA					
1.Myocardial infarct	ion (MI).				

2.Pulmonary infarction and pulmonary embolism.

3.Leukemia.

4.Hemolytic anemia.

5.Infectious mononucleosis.

6.Progressive muscular dystrophy (especially in the early and middle stages of the disease)

7.Liver disease and renal disease.

#### NOTE:-

1. In liver disease, elevations of LDH are not as great as the increases in aspartate amino transferase (AST) and alanine aminotransferase (ALT). 2. Serum LDH may be falsely elevated in otherwise healthy individuals which can be due to mechanical destrunction of RBCs. Therefore, Possiblity of mechanical errors (Transportation or vigorous shaking) should always be ruled out.



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

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





NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

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CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUT	ГЕ	<b>REPORTING DATE</b>	:06/Feb/202502:15PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBAL	A CITY - HA	ARYANA	
Test Name		Value	Unit	Biological Reference interval
	THYRO	DID FUNC	CTION TEST: TOTAL	
		1.35	ng/mL	
TRIIODOTHYRONIN by CMIA (CHEMILUMIN	NE (T3): SERUM IESCENT MICROPARTICLE IMMUNOASSAY)	1.55	iig/ iiiL	0.35 - 1.93
by CMIA (CHEMILUMIN THYROXINE (T4): S	ESCENT MICROPARTICLE IMMUNOASSAY)	7.15	μgm/dL	0.35 - 1.93 4.87 - 13.20
by CMIA (CHEMILUMIN THYROXINE (T4): S by CMIA (CHEMILUMIN THYROID STIMULA	ESCENT MICROPARTICLE IMMUNOASSAY)		C C	
by CMIA (CHEMILUMIN THYROXINE (T4): S by CMIA (CHEMILUMIN THYROID STIMULA	SERUM SERUM SERUM SESCENT MICROPARTICLE IMMUNOASSAY) ATING HORMONE (TSH): SERUM SESCENT MICROPARTICLE IMMUNOASSAY)	7.15	μgm/dL	4.87 - 13.20

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 (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)





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Test Name			Value	Unit	t	Biolog	gical 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		
	RECOM	MENDATIONS OF TSH LE	EVELS DURING PREC	GNANCY ( µIU/mL)			
	1st Trimester			0.10 - 2.50			
	2nd Trimester			0.20 - 3.00			
	3rd Trimester			0.30 - 4.10			ĺ

#### **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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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AME	BALA CITY - HARYANA		
Test Name		Value	Unit	Biological Reference interval
		PROLACTIN		
		6.18	ng/mL	3 - 25
	M ESCENT MICROPARTICLE IMMUNOASS/		0	
INTERPRETATION: 1.Prolactin is secreted 2.The major chemical 3.Physiologic al functi physiologic stimuli su newborn infant. INCREASED (HYPERPR 1.Prolactin-secreting 2.Functional and orga 3.Primary hypothyroi 4.Section compressio 5.Chest wall lesions a	ESCENT MICROPARTICLE IMMUNOASS d by the anterior pituitary gland ar l controlling prolactin secretion is on of prolactin is the stimulation of the as sleep, exercise, nipple stimu <b>OLACTEMIA):</b> pituitary adenoma (prolactinoma, anic disease of the hypothalamus. dism. n of the pituitary stalk.	AY) nd controlled by the hypo dopamine, which inhibits of milk production. In no lation, sexual intercourse	thalamus. prolactin secreti rmal individuals, e, hypoglycemia,	the prolactin level rises in response to postpartum period, and also is elevated in t
by CMIA (CHEMILUMINI INTERPRETATION: 1. Prolactin is secreted 2. The major chemical 3. Physiological functi by siologic stimuli su newborn infant. INCREASED (HYPERPR 1. Prolactin-secreting 2. Functional and orga 3. Primary hypothyroi 4. Section compressio 5. Chest wall lesions a 6. Ectopic tumors. 7. DRUGS:- Anti-Dopan receptors, or seroton Opiates, High doses of 51GNIFICANCE: 1. In loss of libido, gal. 2. Loss of libido, impo from decreased muso 3. In males, prolactin 5. Clear symptoms and	ESCENT MICROPARTICLE IMMUNOASS d by the anterior pituitary gland ar l controlling prolactin secretion is ion of prolactin is the stimulation of ch as sleep, exercise, nipple stimu <b>OLACTEMIA):</b> pituitary adenoma (prolactinoma, anic disease of the hypothalamus. dism. n of the pituitary stalk. and renal failure. minergic drugs like antipsychotic d in reuptake (anti-depressants of a of estrogen or progesterone, antico actorrhea, oligomHyperprolactine tence, infertility, and hypogonadis cle mass and osteoporosis. <i>evels</i> >13 ng/mL are indicative of hy o levels >27 ng/mL in the absence of d signs of hyperprolactinemia are of	AY) Ind controlled by the hypo dopamine, which inhibits of milk production. In no lation, sexual intercourse which is 5 times more free rugs, antinausea/antieme Il classes, ergot derivative onvulsants (valporic acid) mia often results enorrhe im in males. Postmenopau perprolactinemia. pregnancy and postpartur often absent in patients w	thalamus. prolactin secreti rmal individuals, e, hypoglycemia, equent in females etic drugs, Drugs f es, some illegal d , anti-tuberculou ea or amenorrhea usal and premeno m lactation are invith serum prolac	the prolactin level rises in response to postpartum period, and also is elevated in t is than males). that affect CNS serotonin metabolism, seroto rugs such as cannabis), Antihypertensive dru is medications (Isoniazid). a, and infertility in premenopausal females. opausal women, as well as men, can also suf dicative of hyperprolactinemia.





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1	<b>Fest Name</b>	Value	Unit	<b>Biological Reference interval</b>

#### **INSULIN FASTING (F)**

INSULIN FASTING (F)	13.69	µIU/ml	2.0 - 25.0
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)			

#### **INTERPRETATION:-**

1. Insulin is a hormone produced by the beta cells of the pancreas. It regulates the uptake and utilization of glucose and is also involved in protein synthesis and triglyceride storage.

2.Type 1 diabets (insulin-dependent diabetes) is caused by insulin deficiency due to destruction of insulin producing pancreatic islets (beta) cells.

3.Type 2 diabetes (noninsulin dependent diabetes) is characterized by resistance to the action of insulin (insulin resistance).

4. The test is useful for management of diabetes mellitus and for diagnoses of insulinomas, when used in conjunction with proinsulin and C-peptide measurements.

1.No standard referance range has yet been established for INSULIN POST-PRANDIAL (PP) in indian population, therefore same could not be provided along with test. However various studies done on several populations mention that the range of INSULIN PP can vary somewhere from 5-79 mIU/L which can be used for clinical purpose.

2. This assay has 100% cross-reactivity with recombinant human insulin (Novolin R and Novolin N). It does not recognize other commonly used analogues of injectable insulin (ie, insulin lispro, insulin aspart, and insulin glargine).

#### INTERPRETATIVE GUIDE:

1. During prolonged fasting, when the patient's glucose level is reduced to <40 mg/dL, elevated insulin level plus elevated levels of proinsulin and C-peptide suggest insulinomaS.

2. Insulin levels generally decline in patients with type 1 diabetes mellitus.

3.In the early stage of type 2 diabetes, insulin levels are either normal or elevated. In the late stage of type 2 diabetes, insulin levels decline. 4.In normal individuals, insulin levels parallel blood glucose levels.

5.Patients on insulin therapy may develop anti-insulin antibodies. These antibodies may interfere in the assay system, causing inaccurate results. In such individuals, measurement of free insulin FINS / Insulin, Free, Serum should be performed.





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

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



A PIONEER DIAGNOSTIC CENTRE

【 0171-2532620, 8222896961 🛛 🖾 pkrjainhealthcare@gmail.com

NAME	: Miss. KIRANPREET KAUR		
AGE/ GENDER	: 15 YRS/FEMALE	PATIENT ID	: 1747441
COLLECTED BY	:	REG. NO./LAB NO.	: 122502060012
REFERRED BY	:	<b>REGISTRATION DATE</b>	: 06/Feb/2025 11:45 AM
BARCODE NO.	: 12506863	<b>COLLECTION DATE</b>	:06/Feb/202512:04PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	<b>REPORTING DATE</b>	: 06/Feb/2025 05:24PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - 1	HARYANA	
Test Name	Value	Unit	Biological Reference interva
	TESTOS	FERONE: TOTAL	
INTERPRETATION: 1. Testosterone is sec 2. In males it is secret testosterone is in the	COTAL: SERUM 0.37 ESCENT MICROPARTICLE IMMUNOASSAY) reted in females by the ovary and formed indired by the testes. It circulates in blood bound la	ng/mL ectly from androstenedione ir rgely to sex hormone binding	



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**NOT VALID FOR MEDICO LEGAL PURPOSE** 



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CLIENT CODE.	: P.K.R JAIN HEALTHCARE INS	TUTE <b>REPORTING DATE</b>		:06/Feb/202502:15PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AN	IBALA CITY - HARYANA		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		CLINICAL PATHO	LOGY	
	URINE RO	UTINE & MICROSCO	PIC EXAMINA	ATION
PHYSICAL EXAMIN	NATION			
QUANTITY RECIEV	ED TANCE SPECTROPHOTOMETRY	20	ml	
COLOUR	TANCE SPECTRUPHUTUMETRY	PALE YELLOW		PALE YELLOW
•	TANCE SPECTROPHOTOMETRY			
TRANSPARANCY by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	CLEAR		CLEAR
SPECIFIC GRAVITY		1.02 PKR		1.002 - 1.030
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY NATION			
REACTION		ACIDIC		
by DIP STICK/REFLEC PROTEIN	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY			
SUGAR	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
pH		5.5		5.0 - 7.5
by DIP STICK/REFLEC BILIRUBIN	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
NITRITE	TANCE SPECTROPHOTOMETRY.	NEGATIVE (-ve)		NEGATIVE (-ve)
UROBILINOGEN		NOT DETECTED	EU/dL	0.2 - 1.0
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY			
BLOOD by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
ASCORBIC ACID		NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY AMINATION			
	(RBCs)	NEGATIVE (-ve)	/HPF	0 - 3



: Miss. KIRANPREET KAUR

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440 Dated 17.5.2012 u/s 80 G OF INCOME TAX ACT. PAN NO. AAAAP1600. **REPORT ATTRACTS THE CONDITIONS PRINTED OVERLEAF (P.T.O.)** 



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT

NAME

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Test Name	Value	Unit	Biological Reference interval
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	5-7	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	4-6	/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)		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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