









Patient Mrs. Monika

**UHIDNo/IPNO** 100197211

**Age/Gender** 45 Years/Female

Bed No/Ward OPD
Referred By Dr. Self

Lab No/ManualNo 2424077/

**CollectionDate** 03/04/2025 9:03AM

**Receiving Date** 03/04/2025 9:42AM **Report Date** 03/04/2025 2:53PM

Report Status Final

Sample Quality

Test Name Result Unit Bio. Ref. Range Method Sample

Biochemistry

Thomson Press Heart Package (Female)

SERUM CREATININE Serum

Creatinine 0.70 mg/dL 0.52 - 1.04 Enzymatic method

#### Interpretation:-

Serum creatinine and urinary creatinine excretion is a function of lean body mass in normal persons and shows little or no response to dietary changes. The serum creatinine concentration is higher in men than in women. Since urinary creatinine is excreted mainly by glomerular filtration, with only small amounts due to tubular secretion, serum creatinine and a 24-hour urine creatinine excretion can be used to estimate the glomerular filtration rate. Serum creatinine is increased in acute or chronic renal failure, urinary tract obstruction, reduced renal blood flow, shock, dehydration, and rhabdomyolysis. Causes of low serum creatinine concentration include debilitation and decreased muscle mass. common in the elderly, in the bedridden, and in patients with advanced malignancy.

URIC ACID (SERUM) Serum

Uric Acid 4.2 mg/dL 2.5 - 6.2 Uricase / Peroxidase (Colorimetric)

#### Interpretation:-

Uric acid is the end product of purine metabolism. Elevationsof uric acid occur in renal failure, prerenal azotemia, gout, lead poisoning, excessive cell destruction (e.g., following chemotherapy), hemolytic anemia, and congestive heart failure and after myocardial infarction. Uric acid is also increased in some endocrine disorders, acidosis, toxemia of pregnancy, hereditary gout, and glycogen storage disease type I. A low uric acidconcentration may be found following treatment by some drugs (e.g., low-doseaspirin), with low dietary intake of purines, in the presence of renal tubulardefects, and in xanthinuria.

GLUCOSE (PP) Serum

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**Sample Quality** 

Glucose PP 106.00 mg/dL 70.00 - 140.00 GOD/POD colorimetric

#### Interpretation:-

Glucose is a primary cellular energy source. Fasting plasma glucose concentrations and tolerance to a dose of glucose are used to establish the diagnosis of diabetes mellitus and disorders of carbohydrate metabolism. Glucose measurements are used to monitor therapy in diabetics and in patients with dehydration, coma, hypoglycemia, insulinoma, acidosis, and ketoacidosis.

LIPID PROFILE SERUM Serum

Cholesterol Total		177.00	mg/dL	< 200.00	Enzymatic (CHE/CHO/POD)
Triglycerides		99.00	mg/dL	< 150.00	Reflectance spectrophotometry/Enzym atic(lipase /GK/GPO/POD) without correction for free glycerol
HDL Cholesterol	Н	61.00	mg/dL	40.00 - 60.00	Homogenous Enzymatic
Cholesterol LDL (Direct)		92.54	mg/dL	< 130.00	Reflactance Spectrophotometry
VLDL Cholesterol		19.8	mg/dL	< 40	Calculated
Cholesterol HDL / LDL Ratio	Н	0.66	Ratio	0.30 - 0.40	Calculated

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#### NCEP Guidelines:

Lipid	Desirable	Borderline High	High	Very High
Total Cholesterol	< 200 < 100	200-239 130-159	> 240 160-189	> 190
HDL Cholesterol Triglycerides	> 60 < 150	< 40 ( Risk factor ) 150-199	200-499	> 500

BLOOD UREA Serum

Blood Urea 25.0 mg/dL 15.0 - 36.0 Urease,Kinetic,GLDH

Interpretation:-

The major pathway of nitrogen excretion is in the form of urea that is synthesized in the liver, released into the blood, and cleared by the kidneys. A high serum urea nitrogen occurs in glomerulonephritis, shock, urinary tract obstruction, pyelonephritis, and other causes of acute and chronic renal failure. Severe congestive heart failure, hyperalimentation, diabetic ketoacidosis, dehydration, and bleeding from the gastrointestinal tract elevate urea nitrogen. Low urea nitrogen often occurs in normal pregnancy, with decreased protein intake, in acute liver failure, and with intravenous fluid administration.

GLUCOSE (FASTING). PLASMA(FLUORIDE)

Glucose F 92.00 mg/dL 74.00 - 106.00 GOD/POD colorimetric

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\*\*End Of Report\*\*

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**Sample Quality** 

**Test Name** Bio. Ref. Range Result Unit Method Sample

		Thomson Pres	siochemistry s Heart Packas	ge (Female)		
LIVER FUNCTION TEST (LFT) SERUM		1110111001111100	o rrouro r uoriug	50 (2 0)		Serum
Bilirubin Total		0.40	mg/dL	0.20 - 1.30	Azobilirubin/drphylline	
Bilirubin Direct		0.10	mg/dL	0.00 - 0.30	Dual wavelength spectrophotometeric	
Bilirubin Indirect		0.30	mg/dL	0.00 - 1.10	Dual wavelength spectrophotometeric	
AST/SGOT		24.00	U/L	14.00 - 36.00	Enzymatic method	
ALT/SGPT		22.0	U/L	0.0 - 35.0	Kinetic with pyridoxal 5 phosphate	
Gamma GT		16.00	U/L	12.00 - 43.00	L-Gamma-glutamyl-4- nitroanalide	
Alkaline Phosphatase		78.0	U/L	42.0 - 98.0	PNP-Standardize	
Lactic Dehydrogenase (Serum)		191.00	U/L	120.00 - 246.00	pyurate to lactate kinetic method	
Protein Total		8.00	g/dL	6.30 - 8.20	Biuret Method	
Albumin		4.50	g/dL	3.50 - 5.00	BCG Endpoint	
Globulin		3.50	g/dL	3.00 - 3.70	Calculated	
A/G Ratio	L	1.29	Ratio	1.50 - 2.50	Calculated	

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## Interpretation:-

Total bilirubin in serum and plasma is the sum of unconjugated bilirubin (Bu), mono- and di-glucuronide conjugated bilirubin (Bc)?, and delta bilirubin (DELB), a bilirubin fraction covalently bound to albumin. With the exception of anicteric jaundice, total serum bilirubin is invariably increased in jaundice. Causes of jaundice are prehepatic, resulting from various hemolytic diseases; hepatic, resulting from hepatocellular injury or obstruction; and posthepatic, resulting from obstruction of the hepatic or common bile ducts.

Jaundice has been classified as unconjugated and conjugated hyperbilirubinemia. Increased plasma-unconjugated bilirubin is commonly seen in hemolytic disorders, Gilbert's syndrome, Crigler-Najjar syndrome, neonatal jaundice, and ineffective erythropoiesis and in the presence of drugs competing for glucuronide. Increased plasma-conjugated bilirubin occurs with hepatobiliary disorders, including intrahepatic and extrahepatic biliary tree obstruction, liver cell damage, Dubin-Johnson syndrome, and Rotor syndrome. Neonatal bilirubin, the sum of Bu and Bc, is increased in erythroblastosis fetalis (hemolytic disease of the newborn), which causes jaundice in the first two days of life. Other causes of neonatal jaundice include physiologic jaundice, hematoma/hemorrhage, hypothyroidism, and obstructive jaundice.

Aspartate aminotransferase is present in high activity in heart, skeletal muscle, and liver. Increased serum AST activity commonly follows myocardial infarction, pulmonary emboli, skeletal muscle trauma, alcoholic cirrhosis, viral hepatitis, and druginduced hepatitis.

Alanine aminotransferase is present in high activity in liver, skeletal muscle, heart, and kidney. Serum ALT increases rapidly in liver cell necrosis, hepatitis, hepatic cirrhosis, liver tumors, obstructive jaundice, Reye's syndrome, extensive trauma to skeletal muscle, myositis, myocarditis, and myocardial infarction.

Alkaline phosphatase is present mainly in bone, liver, kidney, intestine, placenta, and lung. Serum alkaline phosphatase may be elevated in increased bone metabolism, for example, in adolescents and during the healing of a fracture; primary and secondary hyperparathyroidism; Paget's disease of bone; carcinoma metastatic to bone; osteogenic sarcoma; and Hodgkin's disease if bones are invaded. Hepatobiliary diseases involving cholestasis, inflammation, or cirrhosis increase alkaline phosphatase activity; alkaline phosphatase activity may be increased in renal infarction and failure and in the complications of pregnancy. Low alkaline phosphatase activity may occasionally be seen in hypothyroidism.

Serum proteins transport drugs and metabolites and maintain plasma osmotic pressure. Most serum proteins are synthesized in the liver, with the exception of gamma globulins. One of the most important serum proteins produced in the liver is albumin. Total serum protein concentration can be used for evaluation of nutritional status. Causes of high total serum protein concentration include dehydration, Waldenstrom's macroglobulinemia, multiple myeloma, hyperglobulinemia, granulomatous diseases, and some tropical diseases. Total protein concentration is occasionally increased in collagen diseases, lupus erythematosus, and other instances of chronic infection or inflammation. Causes of low total serum protein concentration include pregnancy, excessive intravenous fluid administration, cirrhosis or other liver diseases, chronic alcoholism, heart failure, nephrotic syndrome, glomerulonephritis, neoplasia, protein-losing enteropathies, malabsorption, and severe malnutrition.

\*\*End Of Report\*\*

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Age/Gender 45 Years/Female **Receiving Date** 03/04/2025 9:42AM **Bed No/Ward** OPD **Report Date** 03/04/2025 1:11PM

**Report Status** Referred By Dr. Self Final

**Sample Quality** 

**Test Name** Result Unit Bio. Ref. Range Method Sample

**Clinical Pathology** 

Thomson Press Heart Package (Female)

**URINE ROUTINE** Urine

**Physical Examination:** 

Pale Yellow Pale Yellow Colour Visual inspection Visual inspection **Appearance** Hazy Clear -Slightly Hazy

**Chemical Examination:** 

**Blood Urine** Peroxidase activity of Negative Negative hemoglobin

Reflectance Bilirubin: Negative Negative

photometer/Fouchet's . method

Urobilinogen Normal Normal Reflectance photometer/schwartz

method

Ketone Negative Negative Reflectance

photometer/Rothera's method

Protein Negative Reflectance Negative

photometer/Sulfosalicylic method

Nitrite: Negative Negative Reflectance

photometer/conv. of nitrate

to nitrite

Urine Glucose Reflectance Negative Negative

photometer/Benedict's method

Leucocyte Negative Negative Reflectance

photometer/Enzymatic reaction

pH:-6.0 4.3 - 7.3Reflectance

photometer/Double

indicator

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1.030 1.010 - 1.030 Reflectance Specific Gravity:

photometer/Reagent strip-

ion exchange **Microscopic Examination:** 

Pus Cells 2-3/HPF 0 - 5 Direct microscopy on centrifuged

sediment

Urine Epithelial Cells 2-3/HPF Direct microscopy on centrifuged sediment

0 - 5

RBC: 0-1/HPF Not Detected Direct microscopy on centrifuged sediment

Not Detected Direct microscopy on centrifuged Casts:

sediment

Urine Bacteria Not Detected Direct microscopy on centrifuged

sediment

Crystals: Not Detected Direct microscopy on centrifuged

sediment

Mucus thread seen.

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Sample Quality

# Interpretation:-

Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders.

**Protein:** Elevated proteins can be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary tract infections and acute illness with fever. Protein reported in urine as Negative(<15 mg/dl), 1+(>=30 mg/dl), 2+(>=100 mg/dl) & 3+(>=500 mg/dl).

**Glucose:** Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain medications. Glucose reported in urine as Negative (<25 mg/dl), 1+(>=50 mg/dl), 2+(>=100 mg/dl), 3+(>=300 mg/dl), 4+(>=1000 mg/dl).

Ketones: Uncontrolled diabetes mellitus can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.

Blood: Occult blood can occur in urine as intact erythrocytes or hemoglobin, which can occur in various urological, nephrological and bleeding disorders.

Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.

Nitrite: Many bacteria give positive results when their number is high. Positive nitrite test suggestive of 105 or more organism in 1 ml of urine specimen.

pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.

Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetis insipidus.

Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine.

Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia.

STOOL OCCULT BLOOD Stool

Stool Occult Blood NEGATIVE Negative Guaiac Method

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Sample Quality

## Interpretation:-

Fecal occult blood identifies the blood(hemoglobin)present in feces as low as 5mg/dl. This test is useful in the detection of bleeding caused by Gastrointestional disorders such as colitis,polyps,diverticulitis,colorectal cancer and hook worm infection.

- 1. Stool samples collected during menstrual bleeding, constipation induced bleeding, bleeding hemorrhoids or when rectal medication is used may cause positive results.
- 2. Medications like aspirin,indomethacin,phenylbutazone,reserpine,corticosteroids and nonsteroidal anti-inflammatory drugs induce gastrointestinal bleeding may cause false positive reactions and should be avoided during and prior to the test.
- 3. Diet containing exogenous peroxidase and food items like red meat, Raw broccoli, cauliflower, radishes and turnips may induce false positive results and should be avoided for 2days before during the test
- 4.Dosages of vitamin c more than 250mgper day may cause a false negative result.
- 5.Because bleeding may be intermittent it is preferable to collect specimens from different bowel movements, preferably consecutives ones.

This test is designed for preliminary screening and does not replace other diagnostic procedures.

Negative result obtained cannot be considered conclusive as the blood in stool is not homogeneously distributed and bleeding is intermittent.

\*\*End Of Report\*\*

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Sample Quality

Test Name		Result	Unit	Bio. Ref. Range	Method Sa	mple
		TTI .	Haematology			
		Thomson	Press Heart Pack	age (Female)		EDTA DI LI
HAEMOGRAM BLOOD						EDTA Blood
Haemoglobin	L	11.5	g/dL	12.0 - 15.0	SLS Method	
TLC	Н	11.2	- 10^3/μL	4.0 - 10.0	Flow Cytometry	
Differential Leukocyte Count						
Neutrophils		65.8	%	40 - 80	Calculated/Light microsopy on leishman stain	
Absolute Neutrophil count	Н	7.37	10^3/μL	2.00 - 7.00	Fluorescence flowcytometry	
Lymphocytes		20.6	%	20 - 40	Calculated/Light microsopy on leishman stain	
Absolute Lymphocyte Count		2.31	10^3/μL	1.00 - 3.00	Fluorescence flowcytometry	
Monocytes		8.2	%	2 - 10	Calculated/Light microsopy on leishman stain	
Absolute Monocyte Count		0.92	10^3/μL	0.20 - 1.00	Fluorescence flowcytometry	
Eosinophils		4.8	%	1 - 6	Calculated/Light microsopy on leishman stain	
Absolute Eosinophil Count	Н	0.54	10^3/μL	0.02 - 0.50	Fluorescence flowcytometry	
Basophils		0.6	%	0 - 2	Calculated/Light microsopy on leishman stain	
Absolute Basophil Count		0.07	10^3/μL	0.02 - 0.10	Fluorescence flowcytometry	
RBC COUNT	Н	5.06	10^6/µL	3.80 - 4.80	H.focusing impedance	
MCV	L	77.3	fl	82.0 - 97.0	Calculated	

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Hematocrit/PCV			39.1	%	36.0 - 46.0	Derived from RBC pulse hieght detection
MCH		L	22.7	pg	27.0 - 32.0	Calculated
MCHC		L	29.4	g/dL	31.5 - 34.5	Calculated
RDW		Н	15.6	%	11.6 - 14.0	Calculated
Platelet count		Н	430	10^3/μL	150 - 410	H.focusing impedance
Erythrocyte Sedir	mentation Rate (ESR)	н	51	mm/hr	0 - 12	Modified westergren Method

## Interpretation:-

The cell morphology is well preserved for 24 hrs. However after 24-48 hrs. a progressive increase in MCV and HCT is observed leading to a decrease in MCHC.

A direct smear is recommended for an accurate diffrential count and for examination of RBC morphology.

Abnormal increases or decreases in cell counts as revealed in a complete blood count may indicate that you have an underlying medical condition that calls for further evaluation.

Erythrocyte sedimentation rate (ESR) is a non-specific phenomena and is clinically useful in the diagnosis and monitoring of disorders associated with an increased production of acute phase reactants.

The ESR is increased in pregnency from about the 3rd month and returns to nomral by the 4th week post partum. ESR is influened by age, sex, menstrual cycle and drugs (eg. corticosteroids, contraceptives).

It is especially low (0-1mm) in polycythaemia, hypofibrinogenemia or congestive cardiac failure and when there are abnormalities of the red cells such as poikilocytosis, spherocytosis or sickle cells.

\*\*End Of Report\*\*

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Attending Consultant Pathology









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**Sample Quality** 

#### **Ultrasound**

Thomson Press Heart Package (Female)

**USG WHOLE ABDOMEN** 









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Referred By Dr. Self Report Status Final Sample Quality

#### **ULTRASOUND WHOLE ABDOMEN:**

#### **FINDINGS:**

Liver is normal in size (~ 14.2 cm) and **shows diffusely increased parenchymal echogenicity** – **s/o grade II fatty changes.** No focal lesions seen. Portal vein and intrahepatic biliary radicals are normal.

Gall bladder is normal in outline & wall thickness. No calculi/sludge/ pericholecystic fluid seen. Common bile duct is normal in caliber.

Pancreas is normal in size, shape & has uniform echogenicity.

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Spleen is normal in size (~9.0cm) and echopattern. No focal lesions seen.

Right kidney approx. 9.7x3.5cm.

Left kidney approx. 10.1x4.0cm.

Both kidneys are normal in position, size, shape and contour. Cortical echogenicity is normal, CMD is maintained. No calculi / hydronephrosis seen. A cortical cyst of size approx. 33x26mm is seen at lower pole of left kidney.

Urinary bladder is well distended with smooth wall outline and normal wall thickness. Lumen is clear.

Uterus is anteverted and normal in size, shape and contour. **A hypoechoic lesion measuring approx. 7.9x7.2mm is seen in anterior myometrium – interstitial fibroid.** Endometrial thickness is 7.9mm.

No adnexal mass lesions seen.

No free fluid is noted.

An anterior abdominal wall defect of approx. size 9.3 mm is seen in umbilical region with herniation of omentum through it- s/o Umbilical hernia.

Please correlate clinically.

\*\*End Of Report\*\*

Dr. Suprabhat Chandra Subhash

Suprement

Senior Resident









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**Sample Quality** 

## X-Ray

Thomson Press Heart Package (Female)

## X-RAY CHEST PA

Investigation: X-Ray - Chest PA View

Bilateral broncho vascular markings are prominent.

CP angles and domes of the diaphragm are normal.

Cardiac size and configuration is normal.

Trachea is central; no mediastinal shift is seen.

Please correlate clinically.

\*\*End Of Report\*\*

Dr. Nitin Kumar

Consultant









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 9:07AM

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**Sample Quality** 

Test Name Result Unit Bio. Ref. Range Method Sample

CytoPathology

Thomson Press Heart Package (Female)

\*PAP SMEAR

Manual Lab No Cyto-372/2025 Specimen Pap smear

Gross Appearance Received 2 slide alcohol fixed smear

Microscopy

Satisfactory for evaluation.

Smear shows predominantly intermediate and superficial mature squamous epithelial cells, few parabasal cells in a background of marked neutrophilic inflammation.

Endocervical cells - seen.

Interpretation Negative for intraepithelial lesion and malignancy (NILM).

Inflammatory smear.

(\*) Not in NABL Scope \*\*End Of Report\*\*

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Dr. Asif Baliyan MD, DNB, DipRCPath Consultant Pathologist











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## Immuno-Haematology

Thomson Press Heart Package (Female)

BLOOD GROUPING EDTA Blood

ABO Group "B" Tube Agglutination Method

Rh Type Positive

Interpretation:-

Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

Disclaimer: Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same.

The test is performed by both forward as well as reverse grouping methods.

\*\*End Of Report\*\*

Dr. Amrita Anand MD PATHOLOGY

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