

**Client**

**Jeevan Jyoti HLM**

Pathkind Diagnostics Pvt. Ltd.

162, Lowther Road, Bai Ka Bagh, Prayagraj

**Processed By**

**Pathkind Diagnostics Pvt. Ltd.**

162, Lowther Road, Bai Ka Bagh, Prayagraj

Uttar Pradesh-211003

<b>Name</b> : Mrs. KUMUD JHA REG-309777	Billing Date	: 10/09/2022 10:25:28
Age : 38 Yrs	Sample Collected on	: 10/09/2022 10:32:30
Sex : Female	Sample Received on	: 10/09/2022 11:49:29
P. ID No. : P1212100000365	Report Released on	: 10/09/2022 12:42:15
<b>Accession No</b> : 121222020372	Barcode No.	: 15457278
Referring Doctor : DEVENDRA TRIPATHI	Ref no.	:
Referred By :		

**Report Status - Preliminary Report**

Test Name	Result	Biological Ref. Interval	Unit
<b>HAEMATOLOGY</b>			
<b>Complete Blood Count (CBC)</b>			
<b>Haemoglobin (Hb)</b> Sample: Whole Blood EDTA Method: Photometric measurement	12.4	12.0 - 15.0	gm/dL
<b>Total WBC Count / TLC</b> Sample: Whole Blood EDTA Method: Impedance	4.4	4.0 - 10.0	thou/ $\mu$ L
<b>RBC Count</b> Sample: Whole Blood EDTA Method: Impedance	<b>5.2 H</b>	3.8 - 4.8	million/ $\mu$ L
<b>PCV / Hematocrit</b> Sample: Whole Blood EDTA Method: Impedance	39.6	36.0 - 46.0	%
<b>MCV</b> Sample: Whole Blood EDTA Method: Calculated	<b>76.6 L</b>	83.0 - 101.0	fL
<b>MCH</b> Sample: Whole Blood EDTA Method: Calculated	<b>24.0 L</b>	27.0 - 32.0	pg
<b>MCHC</b> Sample: Whole Blood EDTA Method: Calculated	<b>31.4 L</b>	31.5 - 34.5	g/dL
<b>RDW (Red Cell Distribution Width)</b> Sample: Whole Blood EDTA Method: Calculated	12.2	11.9 - 15.5	%
<b>DLC (Differential Leucocyte Count)</b> Method: Flowcytometry/Microscopy			
<b>Neutrophils</b> Sample: Whole Blood EDTA Method: VCS Technology & Microscopy	70	40 - 80	%
<b>Lymphocytes</b> Sample: Whole Blood EDTA Method: VCS Technology & Microscopy	21	20 - 40	%

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Test Name	Result	Biological Ref. Interval	Unit
<b>Eosinophils</b> <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology &amp; Microscopy</i>	05	01 - 06	%
<b>Monocytes</b> <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology &amp; Microscopy</i>	04	02 - 10	%
<b>Basophils</b> <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology &amp; Microscopy</i>	00	00 - 02	%
<b>Absolute Neutrophil Count</b> <i>Sample: Whole Blood EDTA</i>	3080	2000 - 7000	/ $\mu$ L
<b>Absolute Lymphocyte Count</b> <i>Sample: Whole Blood EDTA</i>	<b>924 L</b>	1000 - 3000	/ $\mu$ L
<b>Absolute Eosinophil Count</b> <i>Sample: Whole Blood EDTA</i>	220	20 - 500	/ $\mu$ L
<b>Absolute Monocyte Count</b> <i>Sample: Whole Blood EDTA</i>	<b>176 L</b>	200 - 1000	/ $\mu$ L
<b>Absolute Basophil Count</b> <i>Sample: Whole Blood EDTA</i>	<b>00 L</b>	20 - 100	/ $\mu$ L
<b>DLC Performed By</b> <i>Sample: Whole Blood EDTA</i>	EDTA Smear		
<b>Platelet Count</b> <i>Sample: Whole Blood EDTA</i> <i>Method: Impedance</i>	229	150 - 410	thou/ $\mu$ L
<b>MPV (Mean Platelet Volume)</b> <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i>	<b>12.1 H</b>	6.8 - 10.9	fL
<b>Erythrocyte Sedimentation Rate (ESR)</b> <i>Sample: Whole Blood EDTA</i> <i>Method: Modified Westergren Method</i>	<b>14 H</b>	<12	mm 1st Hour

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Test Name	Result	Biological Ref. Interval	Unit
<b>Blood Group</b>			
<b>Blood Grouping</b> <i>Sample: Whole Blood EDTA</i>	" O "		
<b>Rh (D) Typing</b> <i>Sample: Whole Blood EDTA</i>	Positive		
<b>BIOCHEMISTRY</b>			
<b>HbA1C (Glycosylated Hemoglobin)</b>			
<b>HbA1c</b> <i>Sample: Whole Blood EDTA</i> <i>Method: Turbidimetric inhibition immunoassay</i>	<b>8.8 H</b>	Non Diabetic : < 5.7 % Prediabetic Range : 5.7 - 6.4 % Diabetic Range : >= 6.5 % Goal of Therapy : <7.0 % Action suggested : >8.0 %	%
<b>Mean Plasma Glucose</b> <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i>	<b>205.9 H</b>	<116.0	mg/dL
<b>Fasting Plasma Glucose</b> <i>Sample: Fluoride Plasma - F</i>	<b>165 H</b>	74 - 106	mg/dl
<b>Thyroid Profile Total</b>			
<b>Total T3 (Triiodothyronine)</b> <i>Sample: Serum</i> <i>Method: ECLIA</i>	<b>2.68 H</b>	0.80 - 2.00	ng/mL
<b>Total T4 (Thyroxine)</b> <i>Sample: Serum</i> <i>Method: ECLIA</i>	<b>14.60 H</b>	5.10 - 14.10	µg/dL
<b>TSH 3rd Generation</b> <i>Sample: Serum</i> <i>Method: ECLIA</i>	<b>0.008 L</b>	0.270 - 4.200	µIU/mL

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Test Name	Result	Biological Ref. Interval	Unit
<b>Liver Function Test (LFT)</b>			
<b>Bilirubin Total</b> Sample: Serum Method: Spectrophotometry	0.7	<1.1	mg/dL
<b>Bilirubin Direct</b> Sample: Serum Method: Spectrophotometry	0.2	<0.2	mg/dL
<b>Serum Bilirubin (Indirect)</b> Sample: Serum Method: Calculated	0.50	<0.90	mg/dL
<b>SGOT / AST</b> Sample: Serum Method: Spectrophotometry	28	<31	U/L
<b>SGPT / ALT</b> Sample: Serum Method: Spectrophotometry	<b>36 H</b>	<33	U/L
<b>AST / ALT Ratio</b> Sample: Serum Method: Calculated	0.78		
<b>Alkaline Phosphatase (ALP)</b> Sample: Serum Method: Spectrophotometry	88	<98	U/L
<b>Total Protein</b> Sample: Serum Method: Spectrophotometry	7.5	6.4 - 8.3	g/dL
<b>Albumin</b> Sample: Serum Method: Spectrophotometry	4.8	4.0 - 4.9	g/dL
<b>Globulin</b> Sample: Serum Method: Calculated	2.7	1.9 - 3.7	g/dL
<b>Albumin/Globulin (A/G) Ratio</b> Sample: Serum Method: Calculated	1.8	1.0 - 2.1	g/dL

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**Report Status - Preliminary Report**

Test Name	Result	Biological Ref. Interval	Unit
<b>Lipid Profile</b>			
<b>Total Cholesterol</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	197	No risk : < 200 Moderate risk : 200-239 High risk : =240	mg/dL
<b>Triglycerides</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	140	Desirable : < 150 Borderline High : 150 - 199 High : 200 - 499 Very High : >= 500	mg/dL
<b>LDL Cholesterol (Calculated)</b> <i>Sample: Serum</i> <i>Method: Calculated</i>	<b>132 H</b>	Optimal : <100 Near Optimal : 100 - 129 Borderline High : 130 - 160 High : 161 - 189 Very High : >=190	mg/dL
<b>HDL Cholesterol</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	<b>37 L</b>	Low : < 40 Optimal : 40 - 60 High : > 60	mg/dl
<b>Non HDL Cholesterol</b> <i>Sample: Serum</i>	<b>160 H</b>	< 130	mg/dL
<b>VLDL Cholesterol</b> <i>Sample: Serum</i> <i>Method: Calculated</i>	28	Desirable 10 - 35	mg/dL
<b>Total Cholesterol / HDL Ratio</b> <i>Sample: Serum</i> <i>Method: Calculated</i>	<b>5.32 H</b>	Low Risk : 3.3 - 4.4 Average Risk : 4.5 - 7.0 Moderate Risk : 7.1 - 11.0 High Risk : > 11.0	
<b>LDL / HDL Ratio</b> <i>Sample: Serum</i> <i>Method: Calculated</i>	<b>3.6 H</b>	0.5 - 3.0  Low Risk : 0.5 - 3.0 Moderate Risk : 3.1 - 6.0 High Risk : > 6.0	

**Kidney Profile (KFT)****Blood Urea**

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Test Name	Result	Biological Ref. Interval	Unit
<b>Blood Urea Nitrogen (BUN)</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry-Urease / GLDH</i>	8.55	7.00 - 18.69	mg/dL
<b>Urea</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	18.3	17.00 - 43.00	mg/dL
<b>Creatinine</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	0.56	0.50 - 1.10	mg/dL
<b>BUN Creatinine Ratio</b> <i>Sample: Serum</i> <i>Method: Calculated</i>	15	10 - 20	
<b>Calcium</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	<b>10.5 H</b>	8.6 - 10.0	mg/dL
<b>Uric Acid</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	<b>8.0 H</b>	2.4 - 5.7	mg/dL
<b>Total Protein</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	7.5	6.4 - 8.3	g/dL
<b>Albumin</b> <i>Sample: Serum</i> <i>Method: Spectrophotometry</i>	4.8	4.0 - 4.9	g/dL
<b>Globulin</b> <i>Sample: Serum</i> <i>Method: Calculated</i>	2.7	1.9 - 3.7	g/dL
<b>Albumin/Globulin (A/G) Ratio</b> <i>Sample: Serum</i> <i>Method: Calculated</i>	1.8	1.0 - 2.1	g/dL

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**Report Status - Preliminary Report**

Test Name	Result	Biological Ref. Interval	Unit
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**CLINICAL PATHOLOGY**

**Urine Routine & Microscopic Examination**

Method: Reflectance Photometry

**Physical Examination**

**Colour**

Sample: Urine

Method: Physical Examination

amber

Pale Yellow

**Appearance**

Sample: Urine

Method: Physical Examination

Clear

Clear

**Specific Gravity**

Sample: Urine

Method: pKa change of pretreated polyelectrolytes

1.020

1.003 - 1.035

**pH**

Sample: Urine

Method: Double indicator principle

5.0

4.7 - 7.5

**Chemical Examination**

**Glucose**

Sample: Urine

Method: Glucose oxidase/peroxidase

Not Detected

Not Detected

**Protein**

Sample: Urine

Method: Protein-error-of-indicators principle

Trace

Not Detected

**Ketones**

Sample: Urine

Method: Sodium nitroprusside reaction

Not Detected

Not Detected

**Blood**

Sample: Urine

Method: Peroxidase

Not Detected

Not Detected

**Bilirubin**

Sample: Urine

Method: Diazo reaction

Not Detected

Not Detected

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**Report Status - Preliminary Report**

Test Name	Result	Biological Ref. Interval	Unit
<b>Urobilinogen</b> <i>Sample: Urine</i> <i>Method: Ehrlich's reaction</i>	Normal	Normal	
<b>Nitrite</b> <i>Sample: Urine</i> <i>Method: Nitrite Test</i>	Not Detected	Not Detected	
<b>Microscopic Examination</b> <i>Method: Microscopy</i>			
<b>Pus Cells</b> <i>Sample: Urine</i>	2 - 3	0 - 5	/hpf
<b>RBC</b> <i>Sample: Urine</i>	Not Detected	Not Detected	/hpf
<b>Epithelial Cells</b> <i>Sample: Urine</i>	<b>5 - 7</b>	0 - 5	/hpf
<b>Casts</b> <i>Sample: Urine</i>	Not Detected	Not Detected	/hpf
<b>Crystals</b> <i>Sample: Urine</i>	Not Detected	Not Detected	/hpf
<b>Bacteria</b> <i>Sample: Urine</i>	Not Detected	Not Detected	/hpf
<b>Remarks</b> <i>Sample: Urine</i>			

**Remarks** : Microscopic Examination is performed on urine sediment**BIOCHEMISTRY****Electrolytes (Na/K/Cl)**

<b>Sodium</b> <i>Sample: Serum</i> <i>Method: ISE</i>	141	136 - 145	mmol/L
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Test Name	Result	Biological Ref. Interval	Unit
<b>Potassium</b> <i>Sample: Serum</i> <i>Method: ISE</i>	<b>5.2 H</b>	3.5 - 5.1	mmol/L
<b>Chloride</b> <i>Sample: Serum</i> <i>Method: ISE</i>	<b>110 H</b>	97 - 107	mmol/L

**Complete Blood Count (CBC)**Clinical Significance :

CBC comprises of estimation of the cellular components of blood including RBCs, WBCs and Platelets. Mean corpuscular volume (MCV) is a measure of the size of the average RBC, MCH is a measure of the hemoglobin content of the average RBC and MCHC is the hemoglobin concentration per RBC. The red cell distribution width (RDW) is a measure of the degree of variation in RBC size (anisocytosis) and is helpful in distinguishing between some anemias. CBC examination is used as a screening tool to confirm a hematologic disorder, to establish or rule out a diagnosis, to detect an unsuspected hematologic disorder, or to monitor effects of radiation or chemotherapy. Abnormal results may be due to a primary disorder of the cell-producing organs or an underlying disease. Results should be interpreted in conjunction with the patient's clinical picture and appropriate additional testing performed.

**Erythrocyte Sedimentation Rate (ESR)**Clinical Significance :

The erythrocyte sedimentation rate (ESR) is a simple but non-specific test that helps to detect inflammation associated with conditions such as infections, cancers, and autoimmune diseases.

**HbA1C (Glycosylated Hemoglobin)**Clinical Significance :

Hemoglobin A1c (HbA1c) level reflects the mean glucose concentration over the previous period (approximately 8-12 weeks) and provides a much better indication of long-term glycemic control than blood and urinary glucose determinations. American Diabetes Association (ADA) include the use of HbA1c to diagnose diabetes, using a cutpoint of 6.5%. The ADA recommends measurement of HbA1c 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to assess whether a patient's metabolic control has remained continuously within the target range. Falsely low HbA1c results may be seen in conditions that shorten erythrocyte life span. and may

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not reflect glycemc control in these cases accurately.

**Total T3 (Triiodothyronine)**Clinical Significance :

Thyroid hormones, T3 and T4, which are secreted by the thyroid gland, regulate a number of developmental, metabolic, and neural activities throughout the body. The thyroid gland synthesizes 2 hormones - T3 and T4. T3 production in the thyroid gland constitutes approximately 20% of the total circulating T3, 80% being produced by peripheral conversion from T4. T3 is more potent biologically. Total T3 comprises of Free T3 and bound T3. Bound T3 remains bound to carrier proteins like thyroid-binding globulin, prealbumin, and albumin). Only the free forms are metabolically active. In hyperthyroidism, both T4 and T3 levels are usually elevated, but in some rare cases, only T3 elevation is also seen. In hypothyroidism T4 and T3 levels are both low. T3 levels are frequently low in sick or hospitalized euthyroid patients.

**Total T4 (Thyroxine)**Clinical Significance :

Total T4 is synthesized in the thyroid gland. About 0.05% of circulating T4 is in the free or biologically active form. The remainder is bound to thyroxine-binding globulin (TBG), prealbumin, and albumin. High levels of T4 (and FT4) causes hyperthyroidism and low levels lead to hypothyroidism.

**TSH 3rd Generation**Clinical Significance :

TSH levels are elevated in primary hypothyroidism and low in primary hyperthyroidism. Evaluation of TSH is useful in the differential diagnosis of primary from secondary and tertiary hypothyroidism. In primary hypothyroidism, TSH levels are elevated, while in secondary and tertiary hypothyroidism, TSH levels are low or normal. High TSH level in the presence of normal FT4 is called subclinical hypothyroidism and low TSH with normal FT4 is called subclinical hyperthyroidism. Sick, hospitalized patients may have falsely low or transiently elevated TSH. Significant diurnal variation is also seen in TSH levels.

**Guidelines for TSH levels in pregnancy, as per American Thyroid Association, are as follows:**

PREGNANCY TRIMESTER	BIOLOGICAL REFERENCE INTERVAL	UNIT
FIRST TRIMESTER	0.100 - 2.500	μIU/mL
SECOND TRIMESTER	0.200 - 3.000	μIU/mL
THIRD TRIMESTER	0.300 - 3.000	μIU/mL

**Bilirubin Total**

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Referred By	:				

**Report Status - Preliminary Report**

Test Name	Result	Biological Ref. Interval	Unit
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Clinical Significance :

"Total Bilirubin is one of the most commonly used tests to assess liver function. A number of inherited and acquired diseases affect bilirubin production, metabolism, storage and excretion and causes hyperbilirubinemia resulting in jaundice. Hyperbilirubinemia may be due to increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Unconjugated hyperbilirubinemia is seen in newborn and known as physiological jaundice. Elevated unconjugated bilirubin in the neonatal period may result in brain damage (kernicterus). Crigler-Najjar syndromes type I and type II are also associated with elevated levels of indirect bilirubin. Both conjugated and unconjugated bilirubin are increased in hepatitis and space-occupying lesions of the liver; and obstructive lesions such as carcinoma of the head of the pancreas, common bile duct, or ampulla of Vater."

**Bilirubin Direct**Clinical Significance :

"Direct bilirubin is a measurement of conjugated bilirubin. Jaundice can occur as a result of increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Inherited disorders in which direct bilirubin levels are increased are seen in Dubin-Johnson syndrome and Rotor syndrome, idiopathic neonatal hepatitis and biliary atresia. The most commonly occurring form of jaundice of the newborn called physiological jaundice is due to increase in levels of indirect bilirubin. Both conjugated and unconjugated bilirubin are increased in hepatocellular diseases such as hepatitis and space-occupying lesions of the liver, obstructive lesions such as carcinoma of the head of the pancreas, common bile duct, or ampulla of Vater."

**SGOT / AST**Clinical Significance :

"Elevated aspartate aminotransferase (AST) values are seen most commonly in parenchymal liver diseases. Values can be elevated from 10 to 100 times the normal range, though commonly 20 to 50 times elevations are seen. AST levels are raised in infectious hepatitis and other inflammatory conditions affecting the liver along with ALT, though ALT levels are higher. The ALT:AST ratio which is normally <1 is reversed in these conditions and becomes >1. AST levels are usually raised before clinical signs and symptoms of disease appear. AST and ALT also rise in primary or metastatic carcinoma of the liver, with AST usually being higher than ALT. Elevated AST values may also be seen in disorders affecting the heart, skeletal muscle and kidney, such as myocardial infarction, muscular dystrophy, dermatomyositis, acute pancreatitis and crushed muscle injuries."

**SGPT / ALT**

121222020372 Mrs. KUMUD JHA REG-30977

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**Client****Jeevan Jyoti HLM**

Pathkind Diagnostics Pvt. Ltd.

162, Lowther Road, Bai Ka Bagh, Prayagraj

**Processed By****Pathkind Diagnostics Pvt. Ltd.**

162, Lowther Road, Bai Ka Bagh, Prayagraj

Uttar Pradesh-211003

<b>Name</b> : Mrs. KUMUD JHA REG-309777	Billing Date	:	10/09/2022 10:25:28
Age : 38 Yrs	Sample Collected on	:	10/09/2022 10:32:30
Sex : Female	Sample Received on	:	10/09/2022 11:49:29
P. ID No. : P1212100000365	Report Released on	:	10/09/2022 12:42:15
<b>Accession No</b> : 121222020372	Barcode No.	:	15457277, 15457279, 15457287, 15457278
Referring Doctor : DEVENDRA TRIPATHI	Ref no.	:	
Referred By :			

**Report Status - Preliminary Report**

Test Name	Result	Biological Ref. Interval	Unit
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**Clinical Significance :**

Elevated alanine aminotransferase (ALT) values are seen in parenchymal liver diseases characterized by a destruction of hepatocytes. Values are at least 10 times higher the normal range and may reach up to 100 times the upper reference limit. Commonly, values are seen to be 20 - 50 times higher than normal. In infectious hepatitis and other inflammatory conditions affecting the liver, ALT levels rise more than aspartate aminotransferase (AST), and the ALT/AST ratio, which is normally <1, is reversed and becomes >1. ALT levels usually rise before clinical signs and symptoms of disease appear.

**Alkaline Phosphatase (ALP)****Clinical Significance :**

Alkaline Phosphatase levels can be elevated in both liver related as well as bone related conditions. ALP levels are raised (more than 3 fold) in extrahepatic biliary obstruction (eg, by stone or by cancer of the head of the pancreas) than in intrahepatic obstruction, and is directly proportional to the level of obstruction. Levels may rise up to 10 to 12 times the upper limit of normal range and returns to normal on surgical removal of the obstruction. ALP levels rise together with GGT levels and If both GGT and ALP are elevated, a liver source of the ALP is likely. Among bone diseases, ALP levels rise in Paget disease (up to 25 fold), osteomalacia, rickets, primary and secondary hyperparathyroidism and osteogenic bone cancer. Elevated ALP is seen in children following accelerated bone growth. Also, a 2 to 3fold elevation may be observed in women in the third trimester of pregnancy, although the interval is very wide and levels may not exceed the upper limit of the reference interval in some cases.

**Total Protein****Clinical Significance :**

High levels of Serum Total Protein is seen in increased acute phase reactants in inflammation, late-stage liver disease, infections, multiple myeloma and other malignant paraproteinemias. Hypoproteinemia is seen in hypogammaglobulinemia, nephrotic syndrome and protein-losing enteropathy.

**Albumin****Clinical Significance :**

"Hypoalbuminemia can be caused by impaired synthesis due to liver disease (primary) or due to diminished protein intake (secondary), increased catabolism due to tissue damage and inflammation; malabsorption of amino acids; and increased renal excretion (eg, nephrotic syndrome). Hyperalbuminemia is seen in dehydration."

**Lipid Profile**

121222020372 Mrs. KUMUD JHA REG-30977

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**Client**

**Jeevan Jyoti HLM**

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162, Lowther Road, Bai Ka Bagh, Prayagraj

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**Proposed LDL-C goals in very high risk and extreme risk group patients by the Lipid Association of India.**

Very High Risk group(VHRG)	Extreme Risk group	
	Category A	Category B
LDL-C goal of <50 mg/dl	LDL-C goal of <50 mg/dl (recommended) LDL-C goal of ≤30 mg/dl (optional)	LDL-C goal of ≤30 mg/dl
High-risk conditions Any one of following:	CAD with ≥1 of following:	CAD with ≥1 of following:
1. ASCVD (CAD/PAD/TIA or stroke) 2. Homozygous familial 3. hypercholesterolemia 4. Diabetes with ≥2 major ASCVD risk factors*/target organ damage	1. Diabetes without target organ damage/≤1 major 2. ASCVD risk factors 3. Familial hypercholesterolemia 4. ≥3 major ASCVD risk factors 5. CKD stage 3B and 4 6. ≥2 major ASCVD risk factors with ≥1 moderate 7. non-conventional risk factor# 8. Lp(a) ≥50 mg/dl 9. Coronary calcium score ≥300 HU 10. Extreme of a single risk factor 11. PAD 12. H/o TIA or stroke 13. Non-stenotic carotid plaque	1. Diabetes + polyvascular disease/≥2 2. major ASCVD risk factors*/target organ 3. damage 4. Recurrent ACS (within 12 months) 5. despite on LDL-C goal 6. Homozygous familial 7. Hypercholesterolemia

The LDL-C goal of ≤30 mg/dl must be pursued after detailed risk-benefit discussion between physician and patient.

Clinical judgment to be used in decision making if the patient has disease/risk factors not covered in the table, eg. peripheral arterial disease or cerebrovascular disease.

\*Major ASCVD risk factors: 1. Age- male ≥45 years, female ≥55 years, 2. Family h/o premature CAD- male <55 years, female <65 years, 3. Smoking/tobacco use, 4. Systemic hypertension, 5.Low HDL (males <40 mg/dl and females <50 mg/dl).

#Moderate non-conventional risk factors: 1. Coronary calcium score 100–299 HU, 2. Increased carotid intima-media thickness, 3. Lp(a) ≥20–49 mg/dl, 4. Impaired fasting glucose, 5. Increased waist circumference, 6. Apolipoprotein B ≥ 110 mg/dl, 7. hsCRP ≥2 mg/L.

121222020372 Mrs. KUMUD JHA REG-30977



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Pathkind Diagnostics Pvt. Ltd.

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**Report Status - Preliminary Report**

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**Uric Acid**Clinical Significance :

Uric acid is the final product of purine metabolism. Serum uric acid levels are raised in case of increased purine synthesis, inherited metabolic disorder, excess dietary purine intake, increased nucleic acid turnover, malignancy and cytotoxic drugs. Decreased levels are seen in chronic renal failure, severe hepatocellular disease with reduced purine synthesis, defective renal tubular reabsorption, overtreatment of hyperuricemia with allopurinol, as well as some cancer therapies.

**Urine Routine & Microscopic Examination**Clinical Significance :

Urine routine examination and microscopy comprises of a set of screening tests that can detect some common diseases like urinary tract infections, kidney disorders, liver problems, diabetes or other metabolic conditions. Physical characteristics (colour and appearance), chemical composition (glucose, protein, ketone, blood, bilirubin and urobilinogen) and microscopic content ( pus cells, epithelial cells, RBCs, casts and crystals) are analyzed and reported.

**\*\* End of Report\*\*****Dr. Ankit Singh**

MBBS, MD (Pathologist)

Lab Head

121222020372 Mrs. KUMUD JHA REG-30977

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HEART	Lipid Profile	Lipid Profile with Direct LDL	Lipid Profile with Direct LDL
DIABETES	FBS, HbA1c	FBS, HbA1c, Microalbumin	FBS, HbA1c, Microalbumin
KIDNEY	BUN, Creatinine, Bun/Creatinine Ratio, Electrolytes, Uric Acid, Urine R/E	BUN, Creatinine, BUN/Creatinine Ratio, Electrolytes, Uric Acid, Urine R/E	BUN, Creatinine, BUN/Creatinine Ratio, Electrolytes, Uric Acid, Urine R/E
BONES	Vitamin D, Calcium	Vitamin D, Calcium, Phosphorus	Vitamin D, Calcium, Phosphorus, Rheumatoid Factor
THYROID	T3, T4, TSH	T3, T4, TSH	FT3, FT4, TSH
NERVES	Vitamin B12	Vitamin B12	Vitamin B12
LIVER	Bilirubin (Total, Direct, Indirect), SGOT, SGPT, ALP, Protein, Albumin, Globulin, A:G Ratio, HBsAg	Bilirubin (Total, Direct, Indirect), SGOT, SGPT, ALP, GGT, LDH, Protein, Albumin, Globulin, A:G Ratio, HBsAg	Bilirubin (Total, Direct, Indirect), SGOT, SGPT, ALP, GGT, LDH, Protein, Albumin, Globulin, A:G Ratio, HBsAg
ANAEMIA	Iron, TIBC, UIBC, % Saturation	Iron, TIBC, UIBC, % Saturation, Ferritin	Iron, TIBC, UIBC, % Saturation, Ferritin, Folic Acid
INFECTION	CBC, ESR	CBC, ESR	CBC, ESR

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