

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

| | | | |
|------------------|---|---------------------|-----------------------|
| Name | : Mrs. YASHIKA KUMARI REG-317921 ECHS OPD | Billing Date | : 01/04/2023 11:16:54 |
| Age | : 30 Yrs | Sample Collected on | : 01/04/2023 11:23:15 |
| Sex | : Female | Sample Received on | : 01/04/2023 11:40:20 |
| P. ID No. | : P1212100010802 | Report Released on | : 01/04/2023 14:34:43 |
| Accession No | : 12122308 | Barcode No. | : 1212069171 |
| Referring Doctor | : SELF | Ref no. | : |
| Referred By | : | | |

Report Status - Preliminary Report

| Test Name | Result | Biological Ref. Interval | Unit |
|--|--------|--------------------------|------------------|
| HAEMATOLOGY | | | |
| Complete Blood Count (CBC) | | | |
| Haemoglobin (Hb) <i>Sample: Whole Blood EDTA Method: Photometric measurement</i> | 13.1 | 12.0 - 15.0 | gm/dL |
| Total WBC Count / TLC <i>Sample: Whole Blood EDTA Method: Impedance</i> | 7.0 | 4.0 - 10.0 | thou/ μ L |
| RBC Count <i>Sample: Whole Blood EDTA Method: Impedance</i> | 4.5 | 3.8 - 4.8 | million/ μ L |
| PCV / Hematocrit <i>Sample: Whole Blood EDTA Method: Impedance</i> | 39.6 | 36.0 - 46.0 | % |
| MCV <i>Sample: Whole Blood EDTA Method: Calculated</i> | 88.5 | 83.0 - 101.0 | fL |
| MCH <i>Sample: Whole Blood EDTA Method: Calculated</i> | 29.3 | 27.0 - 32.0 | pg |
| MCHC <i>Sample: Whole Blood EDTA Method: Calculated</i> | 33.1 | 31.5 - 34.5 | g/dL |
| RDW (Red Cell Distribution Width) <i>Sample: Whole Blood EDTA Method: Calculated</i> | 11.3 L | 11.9 - 15.5 | % |
| DLC (Differential Leucocyte Count) <i>Method: Flowcytometry/Microscopy</i> | | | |
| Neutrophils <i>Sample: Whole Blood EDTA Method: VCS Technology & Microscopy</i> | 72 | 40 - 80 | % |
| Lymphocytes <i>Sample: Whole Blood EDTA Method: VCS Technology & Microscopy</i> | 24 | 20 - 40 | % |

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| Eosinophils <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology & Microscopy</i> | 02 | 01 - 06 | % |
| Monocytes <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology & Microscopy</i> | 02 | 02 - 10 | % |
| Basophils <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology & Microscopy</i> | 00 | 00 - 02 | % |
| Absolute Neutrophil Count <i>Sample: Whole Blood EDTA</i> | 5040 | 2000 - 7000 | / μ L |
| Absolute Lymphocyte Count <i>Sample: Whole Blood EDTA</i> | 1680 | 1000 - 3000 | / μ L |
| Absolute Eosinophil Count <i>Sample: Whole Blood EDTA</i> | 140 | 20 - 500 | / μ L |
| Absolute Monocyte Count <i>Sample: Whole Blood EDTA</i> | 140 L | 200 - 1000 | / μ L |
| Absolute Basophil Count <i>Sample: Whole Blood EDTA</i> | 00 L | 20 - 100 | / μ L |
| DLC Performed By <i>Sample: Whole Blood EDTA</i> | EDTA Smear | | |
| Platelet Count <i>Sample: Whole Blood EDTA</i> <i>Method: Impedance</i> | 175 | 150 - 410 | thou/ μ L |
| MPV (Mean Platelet Volume) <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i> | 8.0 | 6.8 - 10.9 | fL |
| Erythrocyte Sedimentation Rate (ESR) <i>Sample: Whole Blood EDTA</i> <i>Method: Modified Westergren Method</i> | 14 H | <12 | mm 1st Hour |

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| Blood Group | | | |
| Blood Grouping <i>Sample: Whole Blood EDTA</i> | "B" | | |
| Rh (D) Typing <i>Sample: Whole Blood EDTA</i> | POSITIVE | | |
| BIOCHEMISTRY | | | |
| HbA1C (Glycosylated Hemoglobin) | | | |
| HbA1c <i>Sample: Whole Blood EDTA</i> <i>Method: Turbidimetric inhibition immunoassay</i> | 5.2 | Non Diabetic : < 5.7 % Prediabetic Range : 5.7 - 6.4 % Diabetic Range : >= 6.5 % Goal of Therapy : <7.0 % Action suggested : >8.0 % | % |
| Mean Plasma Glucose <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i> | 102.5 | <116.0 | mg/dL |
| Fasting Plasma Glucose <i>Sample: Fluoride Plasma - F</i> | 87 | 74 - 106 | mg/dl |
| Thyroid Profile Total | | | |
| Total T3 (Triiodothyronine) <i>Sample: Serum</i> <i>Method: ECLIA</i> | 0.98 | 0.80 - 2.00 | ng/mL |
| Total T4 (Thyroxine) <i>Sample: Serum</i> <i>Method: ECLIA</i> | 8.70 | 5.10 - 14.10 | µg/dL |
| TSH 3rd Generation <i>Sample: Serum</i> <i>Method: ECLIA</i> | 3.840 | 0.270 - 4.200 | µIU/mL |

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| Liver Function Test (LFT) | | | |
| Bilirubin Total Sample: Serum Method: Spectrophotometry | 0.6 | <1.1 | mg/dL |
| Bilirubin Direct Sample: Serum Method: Spectrophotometry | 0.2 | <0.2 | mg/dL |
| Serum Bilirubin (Indirect) Sample: Serum Method: Calculated | 0.40 | <0.90 | mg/dL |
| SGOT / AST Sample: Serum Method: Spectrophotometry | 36 H | <31 | U/L |
| SGPT / ALT Sample: Serum Method: Spectrophotometry | 42 H | <33 | U/L |
| AST / ALT Ratio Sample: Serum Method: Calculated | 0.86 | | |
| Alkaline Phosphatase (ALP) Sample: Serum Method: Spectrophotometry | 105 H | <98 | U/L |
| Total Protein Sample: Serum Method: Spectrophotometry | 7.3 | 6.4 - 8.3 | g/dL |
| Albumin Sample: Serum Method: Spectrophotometry | 4.6 | 4.0 - 4.9 | g/dL |
| Globulin Sample: Serum Method: Calculated | 2.7 | 1.9 - 3.7 | g/dL |
| Albumin/Globulin (A/G) Ratio Sample: Serum Method: Calculated | 1.7 | 1.0 - 2.1 | g/dL |

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| Lipid Profile | | | |
| Total Cholesterol <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 151 | No risk : < 200 Moderate risk : 200-239 High risk : =240 | mg/dL |
| Triglycerides <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 90 | Desirable : < 150 Borderline High : 150 - 199 High : 200 - 499 Very High : >= 500 | mg/dL |
| LDL Cholesterol (Calculated) <i>Sample: Serum</i> <i>Method: Calculated</i> | 92 | Optimal : <100 Near Optimal : 100 - 129 Borderline High : 130 - 160 High : 161 - 189 Very High : >=190 | mg/dL |
| HDL Cholesterol <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 41 | Low : < 40 Optimal : 40 - 60 High : > 60 | mg/dl |
| Non HDL Cholesterol <i>Sample: Serum</i> | 110 | < 130 | mg/dL |
| VLDL Cholesterol <i>Sample: Serum</i> <i>Method: Calculated</i> | 18.0 | Desirable 10 - 35 | mg/dL |
| Total Cholesterol / HDL Ratio <i>Sample: Serum</i> <i>Method: Calculated</i> | 3.68 | Low Risk : 3.3 - 4.4 Average Risk : 4.5 - 7.0 Moderate Risk : 7.1 - 11.0 High Risk : > 11.0 | |
| LDL / HDL Ratio <i>Sample: Serum</i> <i>Method: Calculated</i> | 2.2 | 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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| Blood Urea Nitrogen (BUN) <i>Sample: Serum</i> <i>Method: Spectrophotometry-Urease / GLDH</i> | 6.86 L | 7.00 - 18.69 | mg/dL |
| Urea <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 14.68 L | 17.00 - 43.00 | mg/dL |
| Creatinine <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 0.58 | 0.50 - 1.10 | mg/dL |
| BUN Creatinine Ratio <i>Sample: Serum</i> <i>Method: Calculated</i> | 12 | 10 - 20 | |
| Calcium <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 9.1 | 8.6 - 10.0 | mg/dL |
| Uric Acid <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 3.3 | 2.4 - 5.7 | mg/dL |
| Total Protein <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 7.3 | 6.4 - 8.3 | g/dL |
| Albumin <i>Sample: Serum</i> <i>Method: Spectrophotometry</i> | 4.6 | 4.0 - 4.9 | g/dL |
| Globulin <i>Sample: Serum</i> <i>Method: Calculated</i> | 2.7 | 1.9 - 3.7 | g/dL |
| Albumin/Globulin (A/G) Ratio <i>Sample: Serum</i> <i>Method: Calculated</i> | 1.7 | 1.0 - 2.1 | g/dL |
| Electrolytes (Na/K/Cl) | | | |
| Sodium <i>Sample: Serum</i> <i>Method: ISE</i> | 141 | 136 - 145 | mmol/L |

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| Potassium <i>Sample: Serum</i> <i>Method: ISE</i> | 4.3 | 3.5 - 5.1 | mmol/L |
| Chloride <i>Sample: Serum</i> <i>Method: ISE</i> | 112 H | 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 GenerationClinical 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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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

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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

12122308 Mrs. YASHIKA KUMARI REG-317921 ECHS

Page No: 10 of 12



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

| | |
|---|---|
| Name : Mrs. YASHIKA KUMARI REG-317921 ECHS OPD | Billing Date : 01/04/2023 11:16:54 |
| Age : 30 Yrs | Sample Collected on : 01/04/2023 11:23:15 |
| Sex : Female | Sample Received on : 01/04/2023 11:40:20 |
| P. ID No. : P1212100010802 | Report Released on : 01/04/2023 14:34:43 |
| Accession No : 12122308 | Barcode No. : 1212069170, 1212069172, 1212069171 |
| Referring Doctor : SELF | Ref no. : |
| Referred By : | |

Report Status - Preliminary Report

| Test Name | Result | Biological Ref. Interval | Unit |
|-----------|--------|--------------------------|------|
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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.

12122308 Mrs. YASHIKA KUMARI REG-317921 ECHS



Client

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| Referred By : | | | |

Report Status - Preliminary Report

| Test Name | Result | Biological Ref. Interval | Unit |
|-----------|--------|--------------------------|------|
|-----------|--------|--------------------------|------|

Uric AcidClinical 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.

** End of Report**

**Dr. Ankit Singh**

MBBS, MD (Pathologist)

Lab Head

12122308 Mrs. YASHIKA KUMARI REG-317921 ECHS

Page No: 12 of 12

जांच सही तो इलाज सही

from the promoters of 