

Client
Gurugram
 Pathkind Diagnostics Pvt. Ltd.
 Plot No. 55-56, Udhog Vihar Ph-IV, Gurugram - 122015

Processed By
Pathkind Diagnostics Pvt. Ltd.
 Plot No. 55-56, Udhog Vihar Ph-IV, Gurugram - 122015

Name : Mr. PL20	Billing Date : 07/07/2023 12:27:56
Age : 35 Yrs	Sample Collected on : 10/07/2023 10:01:31
Sex : Male	Sample Received on : 10/07/2023 11:02:13
P. ID No. : P1000100012856	Report Released on : 20/07/2023 18:57:42
Accession No : 10002304912	Barcode No. : 10002304912-02
Referring Doctor : Self	
Referred By :	Ref no. :

Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
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HEALTHKIND COMPLETE

HAEMATOLOGY

Complete Blood Count (CBC)

Haemoglobin (Hb) <i>Sample: Whole Blood EDTA</i> <i>Method: Photometric measurement</i>	13.0	13.0 - 17.0	gm/dL
Total WBC Count / TLC <i>Sample: Whole Blood EDTA</i> <i>Method: Impedance</i>	5.0	4.0 - 10.0	thou/ μ L
RBC Count <i>Sample: Whole Blood EDTA</i> <i>Method: Impedance</i>	4.9	4.5 - 5.5	million/ μ L
PCV / Hematocrit <i>Sample: Whole Blood EDTA</i> <i>Method: Impedance</i>	42.6	40.0 - 50.0	%
MCV <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i>	85.6	83.0 - 101.0	fL
MCH <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i>	30.4	27.0 - 32.0	pg
MCHC <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i>	32.6	31.5 - 34.5	g/dL
RDW (Red Cell Distribution Width) <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i>	12.7	11.8 - 15.6	%
DLC (Differential Leucocyte Count) <i>Method: Flowcytometry/Microscopy</i>			
Neutrophils <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology & Microscopy</i>	60	40 - 80	%



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Lymphocytes <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology & Microscopy</i>	30	20 - 40	%
Eosinophils <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology & Microscopy</i>	05	01 - 06	%
Monocytes <i>Sample: Whole Blood EDTA</i> <i>Method: VCS Technology & Microscopy</i>	05	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>	3000	2000 - 7000	/μL
Absolute Lymphocyte Count <i>Sample: Whole Blood EDTA</i>	1500	1000 - 3000	/μL
Absolute Eosinophil Count <i>Sample: Whole Blood EDTA</i>	250	20 - 500	/μL
Absolute Monocyte Count <i>Sample: Whole Blood EDTA</i>	250	200 - 1000	/μL
Absolute Basophil Count <i>Sample: Whole Blood EDTA</i>	00 L	20 - 100	/μL
Platelet Count <i>Sample: Whole Blood EDTA</i> <i>Method: Impedance</i>	210	150 - 410	thou/μL
MPV (Mean Platelet Volume) <i>Sample: Whole Blood EDTA</i> <i>Method: Calculated</i>	8.9	6.8 - 10.9	fL
Erythrocyte Sedimentation Rate (ESR) <i>Sample: Whole Blood EDTA</i> <i>Method: Modified Westergren Method</i>	05	<10	mm 1st Hour



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BIOCHEMISTRY			
Fasting Plasma Glucose <i>Sample: Fluoride Plasma - F</i> <i>Method: Hexokinase</i>	102 H	74 - 99	mg/dL
Glucose Random <i>Sample: Fluoride Plasma - R</i> <i>Method: Hexokinase</i>	132	70 - 140	mg/dL
HbA1C (Glycosylated Hemoglobin)			
HbA1c <i>Sample: Whole Blood EDTA</i> <i>Method: High Performance Liquid Chromatography (HPLC)</i>	4.8	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>	91.1	<116.0	mg/dL
Lipid Profile			
Total Cholesterol <i>Sample: Serum</i> <i>Method: Spectrophotometry-Esterase/CO/Peroxidase</i>	205 H	Desirable Level : < 200 Borderline : 200 - 239 High Risk : >= 240	mg/dL
Triglycerides <i>Sample: Serum</i> <i>Method: Spectrophotometry-Enzymatic</i>	135	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>	45	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-Esterase/CO/Peroxidase</i>	65 H	Low : < 40 Optimal : 40 - 60	mg/dL



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Non HDL Cholesterol <i>Sample: Serum</i>	140 H	High : > 60 < 130	mg/dL
VLDL Cholesterol <i>Sample: Serum Method: Calculated</i>	27.0	Desirable 10 - 35	mg/dL
Total Cholesterol / HDL Ratio <i>Sample: Serum Method: Calculated</i>	3.15 L	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 Method: Calculated</i>	0.7	0.5 - 3.0 Low Risk : 0.5 - 3.0 Moderate Risk : 3.1 - 6.0 High Risk : > 6.0	



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<u>Liver Function Test (LFT)</u>			
Bilirubin Total <i>Sample: Serum</i> <i>Method: Spectrophotometry-Diazo</i>	1.3 H	0.0 - 1.2	mg/dL
Bilirubin Direct <i>Sample: Serum</i> <i>Method: Spectrophotometry-Diazo</i>	0.3 H	0.0 - 0.2	mg/dL
Serum Bilirubin (Indirect) <i>Sample: Serum</i> <i>Method: Calculated</i>	1.00 H	0.00 - 0.90	mg/dL
SGOT / AST <i>Sample: Serum</i> <i>Method: Spectrophotometry-IFCC Without Pyridoxal PO4</i>	45 H	0 - 33	U/L
SGPT / ALT <i>Sample: Serum</i> <i>Method: Spectrophotometry-IFCC Without Pyridoxal PO4</i>	35	0 - 41	U/L
AST / ALT Ratio <i>Sample: Serum</i> <i>Method: Calculated</i>	1.29		
Alkaline Phosphatase (ALP) <i>Sample: Serum</i> <i>Method: IFCC</i>	105	40 - 129	U/L
Total Protein <i>Sample: Serum</i> <i>Method: Spectrophotometry Biuret</i>	7.4	6.4 - 8.3	g/dL
Albumin <i>Sample: Serum</i> <i>Method: Spectrophotometry-Bromocresol Purple</i>	4.6	3.5 - 4.8	g/dL
Globulin <i>Sample: Serum</i> <i>Method: Calculated</i>	2.8	1.9 - 3.7	g/dL
Albumin/Globulin (A/G) Ratio <i>Sample: Serum</i> <i>Method: Calculated</i>	1.6	1.0 - 2.1	g/dL



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Gamma-Glutamyl Transferase (GGT) <i>Sample: Serum</i> <i>Method: Spectrophotometry-GGCNA</i>	85 H	0 - 71	U/L
Kidney Function Test			
Blood Urea Nitrogen (BUN) <i>Sample: Serum</i> <i>Method: Spectrophotometry-Urease / GLDH</i>	20.50	8.87 - 20.50	mg/dL
Urea <i>Sample: Serum</i> <i>Method: Calculated</i>	43.87	19.00 - 44.00	mg/dL
Creatinine <i>Sample: Serum</i> <i>Method: Spectrophotometry Alkaline Picrate</i>	1.35 H	0.70 - 1.30	mg/dL
BUN Creatinine Ratio <i>Sample: Serum</i> <i>Method: Calculated</i>	15	10 - 20	
Uric Acid <i>Sample: Serum</i> <i>Method: Uricase-Peroxidase</i>	4.6	3.6 - 8.2	mg/dL
Sodium <i>Sample: Serum</i> <i>Method: ISE</i>	136	136 - 145	mmol/L
Potassium <i>Sample: Serum</i> <i>Method: ISE</i>	3.8	3.5 - 5.1	mmol/L
Chloride <i>Sample: Serum</i> <i>Method: ISE</i>	106	97 - 107	mmol/L
Calcium <i>Sample: Serum</i> <i>Method: Spectrophotometry - OCC</i>	9.6	8.6 - 10.0	mg/dL
Phosphorus <i>Sample: Serum</i> <i>Method: Spectrophotometry-Phosphomolybdate Reduction</i>	3.9	2.6 - 4.5	mg/dL



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SEROLOGY

Hepatitis B Surface Antigen (HBsAg) Rapid Card

Sample: Serum
 Method: Immunochromatography

Reactive

Non Reactive

BIOCHEMISTRY

Iron Studies

Sample: Serum

Method: Method: Spectrophotometry-Ferrozine

Iron

Sample: Serum
 Method: Spectrophotometry-Ferrozine

75

59 - 158

µg/dL

UIBC

Unsaturated Iron Binding Capacity

Sample: Serum
 Method: Spectrophotometry

258

110 - 370

µg/dL

Total Iron Binding Capacity (TIBC)

Sample: Serum
 Method: Calculated

333

228 - 428

µg/dL

% Saturation

Sample: Serum
 Method: Calculated

23

20 - 50

%

Thyroid Profile Total

Total T3 (Triiodothyronine)

Sample: Serum
 Method: ECLIA

2.89 H

0.80 - 2.00

ng/mL

Total T4 (Thyroxine)

Sample: Serum
 Method: ECLIA

4.53 L

5.10 - 14.10

µg/dL

TSH 3rd Generation

Sample: Serum
 Method: ECLIA

45.600 H

0.270 - 4.200

µIU/mL



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Vitamin Profile			
Vitamin D 25 - Hydroxy <i>Sample: Serum</i> <i>Method: ECLIA</i>	35.0	Deficiency < 20 Insufficiency 20 - 30 Sufficiency 30 - 100 Toxicity > 100	ng/mL
Vitamin B12 <i>Sample: Serum</i> <i>Method: ECLIA</i>	265	211 - 946	pg/mL



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CLINICAL PATHOLOGY

Urine Routine & Microscopic Examination

Method: Reflectance Photometry

Physical Examination

Colour

Sample: Urine

Method: Physical Examination

Pale Yellow

Pale Yellow

Appearance

Sample: Urine

Method: Physical Examination

Clear

Clear

Specific Gravity

Sample: Urine

Method: pKa change of pretreated polyelectrolytes

1.010

1.003 - 1.035

pH

Sample: Urine

Method: Double indicator principle

6.0

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

Not Detected

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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Urobilinogen <i>Sample: Urine</i> <i>Method: Ehrlich's reaction</i>	Normal	Normal	
Nitrite <i>Sample: Urine</i> <i>Method: Nitrite Test</i>	Not Detected	Not Detected	
Microscopic Examination <i>Method: Microscopy</i>			
Pus Cells <i>Sample: Urine</i>	2 - 3	0 - 5	/hpf
RBC <i>Sample: Urine</i>	Not Detected	Not Detected	/hpf
Epithelial Cells <i>Sample: Urine</i>	2 - 3	0 - 5	/hpf
Casts <i>Sample: Urine</i>	Not Detected	Not Detected	/hpf
Crystals <i>Sample: Urine</i>	Not Detected	Not Detected	/hpf
Bacteria <i>Sample: Urine</i>	Not Detected	Not Detected	/hpf
Remarks <i>Sample: Urine</i>			

Remarks : Microscopic Examination is performed on urine sediment
Haemoglobin (Hb)

-

10002304912 Mr. PL20



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Hemoglobin is the iron containing protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs. Decrease in Hemoglobin levels results in anaemia and very high Hemoglobin levels results in hemochromatosis.

PCV / Hematocrit

Clinical Significance :

Hemoglobin is the iron containing protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs. Decrease in Hemoglobin levels results in anaemia and very high Hemoglobin levels results in hemochromatosis. Hematocrit or Packed cell volume (PCV) is the proportion of blood volume occupied by red blood cells and is typically about three times the hemoglobin concentration.

Platelet Count

Clinical Significance :

Platelets or thrombocytes are a cellular component of blood whose function is to stop bleeding by clumping or clotting blood vessel injuries. Low platelet count, also known as Thrombocytopenia, can be either due to less production or increased destruction of platelets. High platelet count or Thrombocytosis can be due to unregulated production, secondary to congenital, reactive or neoplastic conditions.

Complete Blood Count (CBC)

Clinical Significance :

CBC comprises of estimation of the cellular componenets 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 cointent 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 :



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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 not reflect glycemic control in these cases accurately.

Total Cholesterol
Clinical Significance :

Serum cholesterol is elevated in hereditary hyperlipoproteinemias and in other metabolic diseases. Moderate-to-markedly elevated values are also seen in cholestatic liver disease. Increased levels are a risk factor for cardiovascular disease. Low levels of cholesterol may be seen in disorders like hyperthyroidism, malabsorption, and deficiencies of apolipoproteins.

Triglycerides
Clinical Significance :

Triglycerides are partly synthesized in the liver and partly derived from the diet. Increased serum triglyceride levels are a risk factor for atherosclerosis. Hyperlipidemia may be inherited or may be due to conditions like biliary obstruction, diabetes mellitus, nephrotic syndrome, renal failure, certain metabolic disorders or drug induced.

HDL Cholesterol
Clinical Significance :

High-density lipoprotein (HDL) is an important tool used to assess risk of developing coronary heart disease. Increased levels are seen in persons with more physical activity. Very high levels are seen in case of metabolic response to medications like hormone replacement therapy. Raised levels are also



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seen in case of chronic intoxication with alcohol, heavy metals or industrial chemicals. Low HDL cholesterol correlates with increased risk for coronary heart disease (CHD). Very low levels are seen in Tangier disease, cholestatic liver disease and in association with decreased hepatocyte function.

Lipid Profile

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 High-risk conditions Any one 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	LDL-C goal of <50 mg/dl (recommended) LDL-C goal of ≤ 30 mg/dl (optional) CAD with ≥ 1 of following: 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	LDL-C goal of ≤ 30 mg/dl CAD with ≥ 1 of following: 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



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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.

Bilirubin Total

Interpretation

Bilirubin is one of the most commonly used tests to assess liver function. Approximately 85% of the total bilirubin produced is derived from hemoglobin, while the remaining 15% is produced from RBC precursors destroyed in the bone marrow and from the catabolism of other heme-containing proteins. After production in peripheral tissues, bilirubin is rapidly taken up by hepatocytes where it is conjugated and then excreted in the bile. A number of inherited and acquired diseases affect one or more of the steps involved in the production, uptake, storage, metabolism, and excretion of bilirubin. In hepatobiliary diseases of various causes, bilirubin uptake, storage, and excretion are impaired to varying degrees.

The most commonly occurring form of unconjugated hyperbilirubinemia is that seen in newborns and referred to as physiological jaundice. Indirect bilirubin is a calculated parameter its range has not been defined for neonatal period (0-14 days).

Bilirubin Direct

Interpretation

Bilirubin is one of the most commonly used tests to assess liver function. Approximately 85% of the total bilirubin produced is derived from hemoglobin, while the remaining 15% is produced from RBC precursors destroyed in the bone marrow and from the catabolism of other heme-containing proteins. After production in peripheral tissues, bilirubin is rapidly taken up by hepatocytes where it is conjugated and then



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excreted in the bile. A number of inherited and acquired diseases affect one or more of the steps involved in the production, uptake, storage, metabolism, and excretion of bilirubin. In hepatobiliary diseases of various causes, bilirubin uptake, storage, and excretion are impaired to varying degrees.

The most commonly occurring form of unconjugated hyperbilirubinemia is that seen in newborns and referred to as physiological jaundice.

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

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



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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."

Blood Urea Nitrogen (BUN)

Clinical Significance :

Increased blood urea nitrogen (BUN) may be due to prerenal causes (cardiac decompensation, water depletion due to decreased intake and excessive loss, increased protein catabolism, and high protein diet), renal causes (acute glomerulonephritis, chronic nephritis, polycystic kidney disease, nephrosclerosis, and tubular necrosis) and postrenal causes (eg, all types of obstruction of the urinary tract, such as stones, enlarged prostate gland, tumors).

Creatinine

Clinical Significance :

Serum creatinine is inversely correlated with glomerular filtration rate (GFR). Increased levels of Serum Creatinine is associated with renal dysfunction.

Uric Acid

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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.

Sodium

Clinical Significance :

Serum Sodium estimation is performed to assess acid-base balance, water balance, water intoxication, and dehydration.

Potassium

Clinical Significance :

Potassium (K⁺) is the major intracellular cation. It regulates neuromuscular excitability, heart contractility, intracellular fluid volume, and hydrogen ion concentration. High levels of serum Potassium is seen in acute renal disease and end-stage renal failure due to decreased excretion. Levels are also high during the diuretic phase of acute tubular necrosis, during administration of non-potassium sparing diuretic therapy, and during states of excess mineralocorticoid or glucocorticoid.

Chloride

Clinical Significance :

"Chloride (Cl⁻) is the major extracellular anion and it has an important role in maintaining proper body water distribution, osmotic pressure, and normal anion-cation balance in the extracellular fluid compartment. Chloride is increased in dehydration, renal tubular acidosis, acute renal failure, metabolic acidosis associated with prolonged diarrhea and loss of sodium bicarbonate, diabetes insipidus, adrenocortical hyperfunction, salicylate intoxication and with excessive infusion of isotonic saline or extremely high dietary intake of salt. Hyperchloremia acidosis may be a sign of severe renal tubular pathology. Chloride is decreased in overhydration, chronic respiratory acidosis, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting, aldosteronism, bromide intoxication, syndrome of inappropriate antidiuretic hormone secretion, and conditions associated with expansion of extracellular fluid volume."

Calcium

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Serum Calcium levels are used to monitor and diagnose a wide range of diseases of bone, kidney, parathyroid gland, or gastrointestinal tract. Calcium levels may also reflect abnormal vitamin D or protein levels. Hypocalcemia or low serum calcium levels is associated with absent or decreased function of the parathyroid glands, impaired vitamin-D synthesis, low dietary intake and chronic renal failure. Hypercalcemia is due to increased mobilization of calcium from the skeletal system or increased intestinal absorption. It is usually seen in case of primary hyperparathyroidism (pHPT) or bone metastasis of carcinoma of the breast, prostate, thyroid gland, or lung.

Phosphorus

Clinical Significance :

Serum phosphorus levels are low in case of shift of phosphate from extracellular to intracellular space, renal phosphate wasting, loss from the gastrointestinal tract, and loss from intracellular stores. Serum Phosphorus levels rise when the kidneys have an inability to excrete phosphate, increased intake or a shift from of phosphate from the tissues into the extracellular fluid.

Hepatitis B Surface Antigen (HBsAg)

Clinical Significance :

Hepatitis B surface antigen (HBsAg) is the first serologic marker appearing in the serum at 6 to 16 weeks following exposure to HBV. In acute infection, HBsAg usually disappears in 1 to 2 months after the onset of symptoms. Persistence of HBsAg for more than 6 months in duration indicates development of either a chronic carrier state or chronic HBV infection.

In case of negative results:

Please note that while rapid test is a sensitive and reliable screening test, it should not be used as a sole criterion for diagnosis. It is recommended to use molecular testing (PCR) for confirmation.

In case of positive results:

The test has been performed on two different rapid technologies. Please note that while rapid test is a sensitive and reliable screening test, it should not be used as a sole criterion for diagnosis. It is recommended to use molecular testing (PCR) for confirmation.

Iron



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Clinical Significance :

Serum Iron is normal or low in iron deficient anaemia, pregnancy, patients taking oral contraceptive medications, in chronic inflammatory and malignancies. Serum Iron is high in hereditary hemochromatosis and in iron overload states.

Total Iron Binding Capacity (TIBC)

Clinical Significance :

Transferrin is the primary plasma iron transport protein but accounts for 25% to 30% saturation with iron. The additional amount of iron that can be bound is the unsaturated iron-binding capacity (UIBC). The total iron-binding capacity (TIBC) can be indirectly determined using the sum of the serum iron and UIBC. TIBC levels are usually low when serum Iron levels are high and vice versa.

Iron Studies

Iron is an essential trace mineral element which forms an important component of hemoglobin, metal compounds and Vitamin A. Deficiency of iron, leads to microcytic hypochromic anemia. The toxic effects of iron are deposition of iron in various organs of the body and hemochromatosis.

Total Iron Binding capacity (TIBC) is a direct measure of the protein Transferrin which transports iron from the gut to storage sites in the bone marrow. In iron deficiency anemia, serum iron is reduced and TIBC increases.

Transferrin Saturation 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.

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



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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 secondary and tertiary hypothyroidism, TSH levels are low or normal. High TSH level in the presence of normal FT4 is 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

Vitamin D 25 - Hydroxy
Clinical Significance :

The 25-hydroxy vitamin D test is used to detect bone weakness or other bone malfunctions or disorders that occur as a result of a vitamin D deficiency. Those who are at high risk of having low levels of vitamin D include people who don't get much exposure to the sun, older adult, people with obesity, babies who are breastfed only, post gastric bypass surgery, Crohn's disease and other intestinal malabsorption conditions. Hypervitaminosis D usually occurs due to over intake of Vitamin D supplementation.

Vitamin B12
Clinical Significance :

Vitamin B12 is necessary for hematopoiesis and normal neuronal function. It requires intrinsic factor (IF) for absorption. Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal



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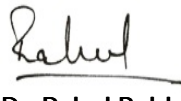
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diseases). Vitamin B12 deficiency results in macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes.

Urine Routine & Microscopic ExaminationClinical 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**

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