Pathkind Labs

Jeevan Jyoti Hospital
Multispeciality Hospital & Infertility Research Center
NABH Accredited Hospital

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	: **Mr. KUMAR SHIVAM**	**REG-323160**	**OPD**	Billing Date	: 12/08/2023 09:43:09
Age	: 30 Yrs			Sample Collected on	: 12/08/2023 12:50:32
Sex	: Male			Sample Received on	: 12/08/2023 12:51:07
P. ID No.	: P1212100017112			Report Released on	: 12/08/2023 13:37:59
Accession No	: **12122306536**			Barcode No.	: 1212051916
Referring Doctor	: SELF				
Referred By	:			Ref no.	:

Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
HAEMATOLOGY			
Complete Blood Count (CBC)			
Haemoglobin (Hb) *Sample: Whole Blood EDTA* *Method: Photometric measurement*	15.4	13.0 - 17.0	gm/dL
Total WBC Count / TLC *Sample: Whole Blood EDTA* *Method: Impedance*	6.3	4.0 - 10.0	thou/µL
RBC Count *Sample: Whole Blood EDTA* *Method: Impedance*	5.2	4.5 - 5.5	million/µL
PCV / Hematocrit *Sample: Whole Blood EDTA* *Method: Impedance*	47.9	40.0 - 50.0	%
MCV *Sample: Whole Blood EDTA* *Method: Calculated*	91.4	83.0 - 101.0	fL
MCH *Sample: Whole Blood EDTA* *Method: Calculated*	29.4	27.0 - 32.0	pg
MCHC *Sample: Whole Blood EDTA* *Method: Calculated*	32.1	31.5 - 34.5	g/dL
RDW (Red Cell Distribution Width) *Sample: Whole Blood EDTA* *Method: Calculated*	14.9	11.8 - 15.6	%
DLC (Differential Leucocyte Count) *Method: Flowcytometry/Microscopy*			
Neutrophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	58	40 - 80	%

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Lymphocytes *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	36	20 - 40	%
Eosinophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	02	01 - 06	%
Monocytes *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	04	02 - 10	%
Basophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	00	00 - 02	%
Absolute Neutrophil Count *Sample: Whole Blood EDTA*	3654	2000 - 7000	/µL
Absolute Lymphocyte Count *Sample: Whole Blood EDTA*	2268	1000 - 3000	/µL
Absolute Eosinophil Count *Sample: Whole Blood EDTA*	126	20 - 500	/µL
Absolute Monocyte Count *Sample: Whole Blood EDTA*	252	200 - 1000	/µL
Absolute Basophil Count *Sample: Whole Blood EDTA*	**00 L**	20 - 100	/µL
DLC Performed By *Sample: Whole Blood EDTA*	EDTA Smear		
Platelet Count *Sample: Whole Blood EDTA* *Method: Impedance*	151	150 - 410	thou/µL
MPV (Mean Platelet Volume) *Sample: Whole Blood EDTA* *Method: Calculated*	**12.7 H**	6.8 - 10.9	fL

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Test Name	Result	Biological Ref. Interval	Unit
Sample: Whole Blood EDTA			
Erythrocyte Sedimentation Rate (ESR) *Sample: Whole Blood EDTA* *Method: Modified Westergren Method*	08	<10	mm 1st Hour
Blood Group			
Blood Grouping *Sample: Whole Blood EDTA* *Method: Column Agglutination*	" A "		
Rh (D) Typing *Sample: Whole Blood EDTA* *Method: Column agglutination*	POSITIVE		
BIOCHEMISTRY			
HbA1C (Glycosylated Hemoglobin)			
HbA1c *Sample: Whole Blood EDTA* *Method: Turbidimetric inhibition immunoassay*	5.5	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 *Sample: Whole Blood EDTA* *Method: Calculated*	111.2	<116.0	mg/dL
Fasting Plasma Glucose *Sample: Fluoride Plasma - F*	94	74 - 106	mg/dl
Glucose Post-Prandial *Sample: Fluoride Plasma - PP* *Method: Hexokinase*	124	70 - 140	mg/dl
Kidney Profile			
Blood Urea			
Blood Urea Nitrogen (BUN) *Sample: Serum* *Method: Spectrophotometry-Urease / GLDH*	9.80	8.87 - 20.50	mg/dL

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Urea *Sample: Serum* *Method: Spectrophotometery*	20.97	17.00 - 43.00	mg/dL
Creatinine *Sample: Serum* *Method: Spectrophotometry*	0.71	0.70 - 1.30	mg/dL
BUN Creatinine Ratio *Sample: Serum* *Method: Calculated*	14	10 - 20	
Uric Acid *Sample: Serum* *Method: Spectrophotometery*	7.0	3.4 - 7.0	mg/dL
Total Protein *Sample: Serum* *Method: Spectrophotometry*	7.4	6.4 - 8.3	g/dL
Albumin *Sample: Serum* *Method: Spectrophotometery*	4.9	4.0 - 4.9	g/dL
Globulin *Sample: Serum* *Method: Calculated*	2.5	1.9 - 3.7	g/dL
Albumin : Globulin Ratio *Sample: Serum* *Method: Calculated*	2.0	1.0 - 2.1	
Sodium *Sample: Serum* *Method: ISE*	140	136 - 145	mmol/L
Potassium *Sample: Serum* *Method: ISE*	4.1	3.5 - 5.1	mmol/L
Chloride *Sample: Serum* *Method: ISE*	**108 H**	97 - 107	mmol/L

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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*	Slightly Hazy	Clear	
Specific Gravity *Sample: Urine* *Method: pKa change of pretreated polyelectrolytes*	1.020	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*	**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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Test Name	Result	Biological Ref. Interval	Unit
Urobilinogen *Sample: Urine* *Method: Ehrlich's reaction*	Normal	Normal	
Nitrite *Sample: Urine* *Method: Nitrite Test*	Not Detected	Not Detected	
Microscopic Examination *Method: Microscopy*			
Pus Cells *Sample: Urine*	2 - 3	0 - 5	/hpf
RBC *Sample: Urine*	Not Detected	Not Detected	/hpf
Epithelial Cells *Sample: Urine*	2 - 3	0 - 5	/hpf
Casts *Sample: Urine*	Not Detected	Not Detected	/hpf
Crystals *Sample: Urine*	Calcium Oxalate	Not Detected	/hpf
Bacteria *Sample: Urine*	Not Detected	Not Detected	/hpf
Remarks *Sample: Urine*			

Remarks : Microscopic Examination is performed on urine sediment

BIOCHEMISTRY

Thyroid Profile Total			
Total T3 (Triiodothyronine) *Sample: Serum* *Method: ECLIA*	1.04	0.80 - 2.00	ng/mL

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Test Name	Result	Biological Ref. Interval	Unit
Total T4 (Thyroxine) *Sample: Serum* *Method: ECLIA*	8.25	5.10 - 14.10	µg/dL
TSH 3rd Generation *Sample: Serum* *Method: ECLIA*	2.300	0.270 - 4.200	µIU/mL
Lipid Profile *Method: Sample: Seurm*			
Total Cholesterol *Sample: Serum* *Method: Spectrophotometery*	190	No risk : < 200 Moderate risk : 200–239 High risk : =240	mg/dL
Triglycerides *Sample: Serum* *Method: Spectrophotometry*	**268 H**	Desirable : < 150 Borderline High : 150 - 199 High : 200 - 499 Very High : >/= 500	mg/dL
LDL Cholesterol (Calculated) *Sample: Serum* *Method: Calculated*	97	Optimal : <100 Near Optimal : 100 - 129 Borderline High : 130 - 160 High : 161 - 189 Very High : >/=190	mg/dL
HDL Cholesterol *Sample: Serum* *Method: Spectrophometry*	**39 L**	Low : < 40 Optimal : 40 - 60 High : > 60	mg/dl
VLDL Cholesterol *Sample: Serum* *Method: Calculated*	**53.6 H**	Desirable 10 - 35	mg/dL
Total Cholesterol / HDL Ratio *Sample: Serum* *Method: Calculated*	**4.87 H**	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 *Sample: Serum* *Method: Calculated*	2.5	0.5 - 3.0	

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Test Name	Result	Biological Ref. Interval	Unit
		Low Risk : 0.5 - 3.0 Moderate Risk : 3.1 - 6.0 High Risk : > 6.0	

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Test Name	Result	Biological Ref. Interval	Unit
Liver Function Test (LFT)			
Bilirubin Total *Sample: Serum* *Method: Spectrophotometry-Diazo*	0.5	0.0 - 1.2	mg/dL
Bilirubin Direct *Sample: Serum* *Method: Spectrophotometry-Diazo*	0.2	0.0 - 0.2	mg/dL
Serum Bilirubin (Indirect) *Sample: Serum* *Method: Calculated*	0.30	0.00 - 0.90	mg/dL
SGOT / AST *Sample: Serum* *Method: Spectrophotometery*	32	<37	U/L
SGPT / ALT *Sample: Serum* *Method: Spectrophotometery*	**46 H**	<41	U/L
AST / ALT Ratio *Sample: Serum* *Method: Calculated*	0.70		
Alkaline Phosphatase (ALP) *Sample: Serum* *Method: Spectrophotometery*	113	<128	U/L
Total Protein *Sample: Serum* *Method: Spectrophotometry*	7.4	6.4 - 8.3	g/dL
Albumin *Sample: Serum* *Method: Spectrophotometery*	4.9	4.0 - 4.9	g/dL
Globulin *Sample: Serum* *Method: Calculated*	2.5	1.9 - 3.7	g/dL
Albumin/Globulin (A/G) Ratio *Sample: Serum* *Method: Calculated*	2.0	1.0 - 2.1	g/dL
Complete Blood Count (CBC)			

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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 :
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.

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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.

Glucose Post-Prandial

COMMENTS / INTERPRETATION:

Any of the following results, confirmed on a subsequent day, can be considered diagnostic for diabetes:
-Fasting plasma or serum glucose > or =126 mg/dL after an 8-hour fast

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-2-Hour plasma or serum glucose > or =200 mg/ dL during a 75-gram oral glucose tolerance test (OGTT)
-Random glucose >200 mg/dL, plus typical symptoms

Patients with "impaired" glucose regulation are those whose fasting serum or plasma glucose fall between 101 and 126 mg/dL, or whose 2-hour value on oral glucose tolerance test fall between 140 and 199 mg/dL. These patients have a markedly increased risk of developing type 2 diabetes and should be counseled for lifestyle changes and followed up with more testing.

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.

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)

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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 hyperthroidism and low levels lead to hypothyroidism.

TSH 3rd Generation

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Clinical Significance :

TSH levels are elevated in primary hyporthyroidism 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.

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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162, Lowther Road, Bai Ka Bagh, Prayagraj

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162, Lowther Road, Bai Ka Bagh, Prayagraj
Uttar Pradesh-211003

Name	: **Mr. KUMAR SHIVAM**	**REG-323160 OPD**	Billing Date	:	12/08/2023 09:43:09
Age	: 30 Yrs		Sample Collected on	:	12/08/2023 12:50:32
Sex	: Male		Sample Received on	:	12/08/2023 12:51:07
P. ID No.	: P1212100017112		Report Released on	:	12/08/2023 13:37:59
Accession No	: **12122306536**		Barcode No.	:	1212051917, 1212051872, 1212051918, 1212051947, 1212051916
Referring Doctor	: SELF				
Referred By	:		Ref no.	:	

Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit

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

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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.n. 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."

** End of Report**

Dr Aparajita singh chauhan

Lab head - Prayagraj (JJH)

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Test Name	Result	Biological Ref. Interval	Unit
HAEMATOLOGY			
Complete Blood Count (CBC)			
Haemoglobin (Hb) *Sample: Whole Blood EDTA* *Method: Photometric measurement*	15.4	13.0 - 17.0	gm/dL
Total WBC Count / TLC *Sample: Whole Blood EDTA* *Method: Impedance*	6.3	4.0 - 10.0	thou/µL
RBC Count *Sample: Whole Blood EDTA* *Method: Impedance*	5.2	4.5 - 5.5	million/µL
PCV / Hematocrit *Sample: Whole Blood EDTA* *Method: Impedance*	47.9	40.0 - 50.0	%
MCV *Sample: Whole Blood EDTA* *Method: Calculated*	91.4	83.0 - 101.0	fL
MCH *Sample: Whole Blood EDTA* *Method: Calculated*	29.4	27.0 - 32.0	pg
MCHC *Sample: Whole Blood EDTA* *Method: Calculated*	32.1	31.5 - 34.5	g/dL
RDW (Red Cell Distribution Width) *Sample: Whole Blood EDTA* *Method: Calculated*	14.9	11.8 - 15.6	%
DLC (Differential Leucocyte Count) *Method: Flowcytometry/Microscopy*			
Neutrophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	58	40 - 80	%

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Lymphocytes *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	36	20 - 40	%
Eosinophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	02	01 - 06	%
Monocytes *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	04	02 - 10	%
Basophils *Sample: Whole Blood EDTA* *Method: VCS Technology & Microscopy*	00	00 - 02	%
Absolute Neutrophil Count *Sample: Whole Blood EDTA*	3654	2000 - 7000	/µL
Absolute Lymphocyte Count *Sample: Whole Blood EDTA*	2268	1000 - 3000	/µL
Absolute Eosinophil Count *Sample: Whole Blood EDTA*	126	20 - 500	/µL
Absolute Monocyte Count *Sample: Whole Blood EDTA*	252	200 - 1000	/µL
Absolute Basophil Count *Sample: Whole Blood EDTA*	**00 L**	20 - 100	/µL
DLC Performed By *Sample: Whole Blood EDTA*	EDTA Smear		
Platelet Count *Sample: Whole Blood EDTA* *Method: Impedance*	151	150 - 410	thou/µL
MPV (Mean Platelet Volume) *Sample: Whole Blood EDTA* *Method: Calculated*	**12.7 H**	6.8 - 10.9	fL

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Test Name	Result	Biological Ref. Interval	Unit
Sample: Whole Blood EDTA			
Erythrocyte Sedimentation Rate (ESR) *Sample: Whole Blood EDTA* *Method: Modified Westergren Method*	08	<10	mm 1st Hour
Blood Group			
Blood Grouping *Sample: Whole Blood EDTA* *Method: Column Agglutination*	" A "		
Rh (D) Typing *Sample: Whole Blood EDTA* *Method: Column agglutination*	POSITIVE		
BIOCHEMISTRY			
HbA1C (Glycosylated Hemoglobin)			
HbA1c *Sample: Whole Blood EDTA* *Method: Turbidimetric inhibition immunoassay*	5.5	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 *Sample: Whole Blood EDTA* *Method: Calculated*	111.2	<116.0	mg/dL
Fasting Plasma Glucose *Sample: Fluoride Plasma - F*	94	74 - 106	mg/dl
Glucose Post-Prandial *Sample: Fluoride Plasma - PP* *Method: Hexokinase*	124	70 - 140	mg/dl
Kidney Profile			
Blood Urea			
Blood Urea Nitrogen (BUN) *Sample: Serum* *Method: Spectrophotometry-Urease / GLDH*	9.80	8.87 - 20.50	mg/dL

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Test Name	Result	Biological Ref. Interval	Unit
Urea *Sample: Serum* *Method: Spectrophotometery*	20.97	17.00 - 43.00	mg/dL
Creatinine *Sample: Serum* *Method: Spectrophotometry*	0.71	0.70 - 1.30	mg/dL
BUN Creatinine Ratio *Sample: Serum* *Method: Calculated*	14	10 - 20	
Uric Acid *Sample: Serum* *Method: Spectrophotometery*	7.0	3.4 - 7.0	mg/dL
Total Protein *Sample: Serum* *Method: Spectrophotometry*	7.4	6.4 - 8.3	g/dL
Albumin *Sample: Serum* *Method: Spectrophotometery*	4.9	4.0 - 4.9	g/dL
Globulin *Sample: Serum* *Method: Calculated*	2.5	1.9 - 3.7	g/dL
Albumin : Globulin Ratio *Sample: Serum* *Method: Calculated*	2.0	1.0 - 2.1	
Sodium *Sample: Serum* *Method: ISE*	140	136 - 145	mmol/L
Potassium *Sample: Serum* *Method: ISE*	4.1	3.5 - 5.1	mmol/L
Chloride *Sample: Serum* *Method: ISE*	**108 H**	97 - 107	mmol/L

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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*	Slightly Hazy	Clear	
Specific Gravity *Sample: Urine* *Method: pKa change of pretreated polyelectrolytes*	1.020	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*	**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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Urobilinogen *Sample: Urine* *Method: Ehrlich's reaction*	Normal	Normal	
Nitrite *Sample: Urine* *Method: Nitrite Test*	Not Detected	Not Detected	
Microscopic Examination *Method: Microscopy*			
Pus Cells *Sample: Urine*	2 - 3	0 - 5	/hpf
RBC *Sample: Urine*	Not Detected	Not Detected	/hpf
Epithelial Cells *Sample: Urine*	2 - 3	0 - 5	/hpf
Casts *Sample: Urine*	Not Detected	Not Detected	/hpf
Crystals *Sample: Urine*	Calcium Oxalate	Not Detected	/hpf
Bacteria *Sample: Urine*	Not Detected	Not Detected	/hpf
Remarks *Sample: Urine*			

Remarks : Microscopic Examination is performed on urine sediment

BIOCHEMISTRY

Thyroid Profile Total			
Total T3 (Triiodothyronine) *Sample: Serum* *Method: ECLIA*	1.04	0.80 - 2.00	ng/mL

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Total T4 (Thyroxine) *Sample: Serum* *Method: ECLIA*	8.25	5.10 - 14.10	µg/dL
TSH 3rd Generation *Sample: Serum* *Method: ECLIA*	2.300	0.270 - 4.200	µIU/mL
Lipid Profile *Method: Sample: Seurm*			
Total Cholesterol *Sample: Serum* *Method: Spectrophotometery*	190	No risk : < 200 Moderate risk : 200–239 High risk : =240	mg/dL
Triglycerides *Sample: Serum* *Method: Spectrophotometry*	**268 H**	Desirable : < 150 Borderline High : 150 - 199 High : 200 - 499 Very High : >/= 500	mg/dL
LDL Cholesterol (Calculated) *Sample: Serum* *Method: Calculated*	97	Optimal : <100 Near Optimal : 100 - 129 Borderline High : 130 - 160 High : 161 - 189 Very High : >/=190	mg/dL
HDL Cholesterol *Sample: Serum* *Method: Spectrophometry*	**39 L**	Low : < 40 Optimal : 40 - 60 High : > 60	mg/dl
VLDL Cholesterol *Sample: Serum* *Method: Calculated*	**53.6 H**	Desirable 10 - 35	mg/dL
Total Cholesterol / HDL Ratio *Sample: Serum* *Method: Calculated*	**4.87 H**	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 *Sample: Serum* *Method: Calculated*	2.5	0.5 - 3.0	

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		Low Risk : 0.5 - 3.0 Moderate Risk : 3.1 - 6.0 High Risk : > 6.0	

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Pathkind Diagnostics Pvt. Ltd.
162, Lowther Road, Bai Ka Bagh, Prayagraj
Uttar Pradesh-211003

Name	: **Mr. KUMAR SHIVAM**	**REG-323160 OPD**	Billing Date	:	12/08/2023 09:43:09
Age	: 30 Yrs		Sample Collected on	:	12/08/2023 12:50:32
Sex	: Male		Sample Received on	:	12/08/2023 12:51:07
P. ID No.	: P1212100017112		Report Released on	:	12/08/2023 13:37:59
Accession No	: **12122306536**		Barcode No.	:	1212051917, 1212051872, 1212051918, 1212051947, 1212051916
Referring Doctor	: SELF				
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Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
Liver Function Test (LFT)			
Bilirubin Total *Sample: Serum* *Method: Spectrophotometry-Diazo*	0.5	0.0 - 1.2	mg/dL
Bilirubin Direct *Sample: Serum* *Method: Spectrophotometry-Diazo*	0.2	0.0 - 0.2	mg/dL
Serum Bilirubin (Indirect) *Sample: Serum* *Method: Calculated*	0.30	0.00 - 0.90	mg/dL
SGOT / AST *Sample: Serum* *Method: Spectrophotometery*	32	<37	U/L
SGPT / ALT *Sample: Serum* *Method: Spectrophotometery*	**46 H**	<41	U/L
AST / ALT Ratio *Sample: Serum* *Method: Calculated*	0.70		
Alkaline Phosphatase (ALP) *Sample: Serum* *Method: Spectrophotometery*	113	<128	U/L
Total Protein *Sample: Serum* *Method: Spectrophotometry*	7.4	6.4 - 8.3	g/dL
Albumin *Sample: Serum* *Method: Spectrophotometery*	4.9	4.0 - 4.9	g/dL
Globulin *Sample: Serum* *Method: Calculated*	2.5	1.9 - 3.7	g/dL
Albumin/Globulin (A/G) Ratio *Sample: Serum* *Method: Calculated*	2.0	1.0 - 2.1	g/dL
Complete Blood Count (CBC)			

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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 :
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.

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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.

Glucose Post-Prandial

COMMENTS / INTERPRETATION:

Any of the following results, confirmed on a subsequent day, can be considered diagnostic for diabetes:
-Fasting plasma or serum glucose > or =126 mg/dL after an 8-hour fast

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-2-Hour plasma or serum glucose > or =200 mg/ dL during a 75-gram oral glucose tolerance test (OGTT)
-Random glucose >200 mg/dL, plus typical symptoms

Patients with "impaired" glucose regulation are those whose fasting serum or plasma glucose fall between 101 and 126 mg/dL, or whose 2-hour value on oral glucose tolerance test fall between 140 and 199 mg/dL. These patients have a markedly increased risk of developing type 2 diabetes and should be counseled for lifestyle changes and followed up with more testing.

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.

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)

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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 hyperthroidism and low levels lead to hypothyroidism.

TSH 3rd Generation

-

Clinical Significance :

TSH levels are elevated in primary hyporthyroidism 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.

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

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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.n. 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."

** End of Report**

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