DIAGNOSTIC REPORT

Patient Ref. No. 775000002237538

SRL Diagnostics

CLIENT CODE : C000138375

CLIENT'S NAME AND ADDRESS :
ACROFEMI HEALTHCARE LTD (MEDIWHEEL)
F-703, LADO SARAI, MEHRAULI
SOUTH WEST DELHI
NEW DELHI 110030
DELHI INDIA
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Tel : 0291-2646000, 2644000, Fax :
CIN - U74899PB1995PLC045956
Email : srl.jodhpur@gmail.com

PATIENT NAME : JEEVAN RAM KARWA PATIENT ID : **JEEVM30016461**

ACCESSION NO : **0061WA002138** AGE : 59 Years SEX : Male ABHA NO :

DRAWN : 28/01/2023 00:00 RECEIVED : 30/01/2023 15:39 REPORTED : 04/02/2023 13:34

REFERRING DOCTOR : DR. BOB PACKAGE CLIENT PATIENT ID :

Test Report Status **Final**	Results		Biological Reference Interval	Units
MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE				
BLOOD COUNTS,EDTA WHOLE BLOOD				
HEMOGLOBIN (HB)	15.0		13.0 - 17.0	g/dL
RED BLOOD CELL (RBC) COUNT	5.36		4.5 - 5.5	mil/µL
WHITE BLOOD CELL (WBC) COUNT	5.35		4.0 - 10.0	thou/µL
PLATELET COUNT	248		150 - 410	thou/µL
RBC AND PLATELET INDICES				
HEMATOCRIT (PCV)	48.9		40 - 50	%
MEAN CORPUSCULAR VOLUME (MCV)	91.2		83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	28.0		27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	**30.7**	**Low**	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW)	**14.3**	**High**	11.6 - 14.0	%
MENTZER INDEX	17.0			
MEAN PLATELET VOLUME (MPV)	**13.0**	**High**	6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT				
NEUTROPHILS	49		40 - 80	%
LYMPHOCYTES	39		20 - 40	%
MONOCYTES	08		2 - 10	%
EOSINOPHILS	04		1 - 6	%
BASOPHILS	00		< 1 - 2	%
MORPHOLOGY				
RBC	RED BLOOD CELLS ARE NORMOCYTIC NORMOCHROMIC			
WBC	WBCS ARE NORMAL IN NUMBER & MORPHOLOGY			
PLATELETS	PLATELETS ARE ADEQUATE IN NUMBER			
ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD				
E.S.R	05		0 - 14	mm at 1 hr
METHOD : WESTERGREN METHOD				
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD				
HBA1C	**9.2**	**High**	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%

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ESTIMATED AVERAGE GLUCOSE(EAG)	**217.3**	**High**	< 116.0	mg/dL
GLUCOSE FASTING,FLUORIDE PLASMA				
FBS (FASTING BLOOD SUGAR)	**181**	**High**	74 - 99	mg/dL
METHOD : SPECTROPHOTOMETRY				
GLUCOSE, POST-PRANDIAL, PLASMA				
PPBS(POST PRANDIAL BLOOD SUGAR)	**322**	**High**	70 - 139	mg/dL
METHOD : SPECTROPHOTOMETRY				
LIPID PROFILE, SERUM				
CHOLESTEROL, TOTAL	**210**	**High**	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : SPECTROPHOTOMETRY				
TRIGLYCERIDES	**602**	**High**	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
METHOD : SPECTROPHOTOMETRY				
HDL CHOLESTEROL	**34**	**Low**	< 40 Low >/=60 High	mg/dL
METHOD : SPECTROPHOTOMETRY				
CHOLESTEROL LDL	56		< 100 Optimal 100 - 129 Near optimal/ above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
NON HDL CHOLESTEROL	**176**	**High**	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
VERY LOW DENSITY LIPOPROTEIN	**120.4**	**High**	</= 30.0	mg/dL
CHOL/HDL RATIO	**6.2**	**High**	3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk	
LDL/HDL RATIO	1.6		0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	

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Comments

NOTE : RESULT RECHECKED FOR SERUM TRIGLYCERIDES.
VLDL IS A CALCULATED VALUE AND THE FORMULA IS INVALID WHEN THE TRIGLYCERIDES VALUE EXCEED 400 MG/DL.

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Interpretation(s)

1) Cholesterol levels help assess the patient risk status and to follow the progress of patient under treatment to lower serum cholesterol concentrations.

2) Serum Triglyceride (TG) are a type of fat and a major source of energy for the body. Both quantity and composition of the diet impact on plasma triglyceride concentrations. Elevations in TG levels are the result of overproduction and impaired clearance. High TG are associated with increased risk for CAD (Coronary artery disease) in patients with other risk factors, such as low HDL-C, some patient groups with elevated apolipoprotein B concentrations, and patients with forms of LDL that may be particularly atherogenic.

3)HDL-C plays a crucial role in the initial step of reverse cholesterol transport, this considered to be the primary atheroprotective function of HDL

4) LDL -C plays a key role in causing and influencing the progression of atherosclerosis and, in particular, coronary sclerosis.The majority of cholesterol stored in atherosclerotic plaques originates from LDL, thus LDL-C value is the most powerful clinical predictor.

5)Non HDL cholesterol: Non-HDL-C measures the cholesterol content of all atherogenic lipoproteins, including LDL hence it is a better marker of risk in both primary and secondary prevention studies. Non-HDL-C also covers, to some extent, the excess ASCVD risk imparted by the sdLDL, which is significantly more atherogenic than the normal large buoyant particles, an elevated non-HDL-C indirectly suggests greater proportion of the small, dense variety of LDL particles

Serum lipid profile is measured for cardiovascular risk prediction.Lipid Association of India recommends LDL-C as primary target and Non HDL-C as co-primary treatment target.

Risk Stratification for ASCVD (Atherosclerotic cardiovascular disease) by Lipid Association of India

Risk Category	
Extreme risk group	A.CAD with > 1 feature of high risk group
	B. CAD with > 1 feature of Very high risk group or recurrent ACS (within 1 year) despite LDL-C < or = 50 mg/dl or polyvascular disease
Very High Risk	1. Established ASCVD 2. Diabetes with 2 major risk factors or evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia
High Risk	1. Three major ASCVD risk factors. 2. Diabetes with 1 major risk factor or no evidence of end organ damage. 3. CKD stage 3B or 4. 4. LDL >190 mg/dl 5. Extreme of a single risk factor. 6. Coronary Artery Calcium - CAC >300 AU. 7. Lipoprotein a >/= 50mg/dl 8. Non stenotic carotid plaque
Moderate Risk	2 major ASCVD risk factors
Low Risk	0-1 major ASCVD risk factors
Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors	
1. Age > or = 45 years in males and > or = 55 years in females	3. Current Cigarette smoking or tobacco use
2. Family history of premature ASCVD	4. High blood pressure
5. Low HDL	

Newer treatment goals and statin initiation thresholds based on the risk categories proposed by LAI in 2020.

Risk Group	**Treatment Goals**		**Consider Drug Therapy**	
	LDL-C (mg/dl)	**Non-HDL (mg/dl)**	**LDL-C (mg/dl)**	**Non-HDL (mg/dl)**

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Extreme Risk Group Category A	<50 (Optional goal < OR = 30)	< 80 (Optional goal <OR = 60)	>OR = 50	>OR = 80
Extreme Risk Group Category B	<OR = 30	<OR = 60	> 30	>60
Very High Risk	<50	<80	>OR= 50	>OR= 80
High Risk	<70	<100	>OR= 70	>OR= 100
Moderate Risk	<100	<130	>OR= 100	>OR= 130
Low Risk	<100	<130	>OR= 130*	>OR= 160

*After an adequate non-pharmacological intervention for at least 3 months.

References: Management of Dyslipidaemia for the Prevention of Stroke: Clinical Practice Recommendations from the Lipid Association of India. Current Vascular Pharmacology, 2022, 20, 134-155.

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	1.00		0.2 - 1.0	mg/dL
METHOD : SPECTROPHOTOMETRY				
BILIRUBIN, DIRECT	0.10		0.0 - 0.2	mg/dL
METHOD : SPECTROPHOTOMETRY				
BILIRUBIN, INDIRECT	0.9		0.1 - 1.0	mg/dL
METHOD : SPECTROPHOTOMETRY				
TOTAL PROTEIN	7.7		6.4 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRY				
ALBUMIN	4.6		3.4 - 5.0	g/dL
METHOD : SPECTROPHOTOMETRY				
GLOBULIN	3.1		2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER				
ALBUMIN/GLOBULIN RATIO	1.5		1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	**50**	**High**	15 - 37	U/L
METHOD : SPECTROPHOTOMETRY				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	**48**	**High**	< 45.0	U/L
METHOD : SPECTROPHOTOMETRY				
ALKALINE PHOSPHATASE	80		30 - 120	U/L
METHOD : SPECTROPHOTOMETRY				
GAMMA GLUTAMYL TRANSFERASE (GGT)	**92**	**High**	15 - 85	U/L
METHOD : SPECTROPHOTOMETRY				
LACTATE DEHYDROGENASE	176		100 - 190	U/L
METHOD : SPECTROPHOTOMETRY				

BLOOD UREA NITROGEN (BUN), SERUM

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BLOOD UREA NITROGEN	6		6 - 20	mg/dL
METHOD : SPECTROPHOTOMETRY				
CREATININE, SERUM				
CREATININE	0.96		0.90 - 1.30	mg/dL
METHOD : SPECTROPHOTOMETRY				
BUN/CREAT RATIO				
BUN/CREAT RATIO	6.25		5.00 - 15.00	
METHOD : SPECTROPHOTOMETRY				
URIC ACID, SERUM				
URIC ACID	5.9		3.5 - 7.2	mg/dL
METHOD : SPECTROPHOTOMETRY				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.7		6.4 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRY				
ALBUMIN, SERUM				
ALBUMIN	4.6		3.4 - 5.0	g/dL
METHOD : SPECTROPHOTOMETRY				
GLOBULIN				
GLOBULIN	3.1		2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM, SERUM	**132**	**Low**	136 - 145	mmol/L
METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY				
POTASSIUM, SERUM	5.0		3.50 - 5.10	mmol/L
METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY				
CHLORIDE, SERUM	**96**	**Low**	98 - 107	mmol/L
METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY				

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Interpretation(s)

Sodium	Potassium	Chloride
Decreased in:CCF,cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy,adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide,carbamazepine,anti depressants (SSRI), antipsychotics.	**Decreased in:** Low potassium intake,prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome,osmotic diuresis (e.g., hyperglycemia),alkalosis, familial periodic paralysis,trauma (transient).Drugs: Adrenergic agents, diuretics.	**Decreased in:** Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism,metabolic alkalosis. Drugs: chronic laxative,corticosteroids, diuretics.
Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea),diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice,oral contraceptives.	**Increased in:** Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration,renal failure, Addison' s disease, RTA type IV, hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium- sparing diuretics,NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole.	**Increased in:** Renal failure, nephrotic syndrome, RTA,dehydration, overtreatment with saline,hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis,hyperadrenocorticism. Drugs: acetazolamide,androgens, hydrochlorothiazide,salicylates.
Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.	**Interferences:** Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.	**Interferences:**Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (Normal serum chloride)

PHYSICAL EXAMINATION, URINE

COLOR	PALE YELLOW	
APPEARANCE	**HAZY**	

CHEMICAL EXAMINATION, URINE

PH	7.0	4.7 - 7.5
SPECIFIC GRAVITY	1.015	1.003 - 1.035
PROTEIN	NOT DETECTED	NOT DETECTED
GLUCOSE	NOT DETECTED	NOT DETECTED
KETONES	NOT DETECTED	NOT DETECTED
BLOOD	NOT DETECTED	NOT DETECTED
BILIRUBIN	NOT DETECTED	NOT DETECTED
UROBILINOGEN	NORMAL	NORMAL
NITRITE	NOT DETECTED	NOT DETECTED
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED

MICROSCOPIC EXAMINATION, URINE

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RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S)	2-3	0-5	/HPF
EPITHELIAL CELLS	2-3	0-5	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	**DETECTED (+++)**	NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	

Interpretation(s)

The following table describes the probable conditions, in which the analytes are present in urine

Presence of	Conditions
Proteins	Inflammation or immune illnesses
Pus (White Blood Cells)	Urinary tract infection, urinary tract or kidney stone, tumors or any kind of kidney impairment
Glucose	Diabetes or kidney disease
Ketones	Diabetic ketoacidosis (DKA), starvation or thirst
Urobilinogen	Liver disease such as hepatitis or cirrhosis
Blood	Renal or genital disorders/trauma
Bilirubin	Liver disease
Erythrocytes	Urological diseases (e.g. kidney and bladder cancer, urolithiasis), urinary tract infection and glomerular diseases
Leukocytes	Urinary tract infection, glomerulonephritis, interstitial nephritis either acute or chronic, polycystic kidney disease, urolithiasis, contamination by genital secretions
Epithelial cells	Urolithiasis, bladder carcinoma or hydronephrosis, ureteric stents or bladder catheters for prolonged periods of time
Granular Casts	Low intratubular pH, high urine osmolality and sodium concentration, interaction with Bence-Jones protein
Hyaline casts	Physical stress, fever, dehydration, acute congestive heart failure, renal diseases
Calcium oxalate	Metabolic stone disease, primary or secondary hyperoxaluria, intravenous infusion of large doses of vitamin C, the use of vasodilator naftidrofuryl oxalate or the gastrointestinal lipase inhibitor orlistat, ingestion of ethylene glycol or of star fruit (Averrhoa carambola) or its juice
Uric acid	arthritis
Bacteria	Urinary infectionwhen present in significant numbers & with pus cells.
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis

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THYROID PANEL, SERUM			
T3	109.40	80.00 - 200.00	ng/dL
T4	7.36	5.10 - 14.10	µg/dL
TSH (ULTRASENSITIVE)	3.720	0.270 - 4.200	µIU/mL

Interpretation(s)

Triiodothyronine T3 , **Thyroxine T4,** and **Thyroid Stimulating Hormone TSH** are thyroid hormones which affect almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate.
Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.
Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism.
In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hyperthyroidism, TSH levels are low.
Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3.Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hyperthyroidism, and measurement of TT4 is more useful in the diagnosis of hypothyroidism.Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active. It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4**.**

Sr. No.	TSH	Total T4	FT4	Total T3	Possible Conditions
1	High	Low	Low	Low	(1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3) Post Thyroidectomy (4) Post Radio-Iodine treatment
2	High	Normal	Normal	Normal	(1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid hormone replacement therapy (3) In cases of Autoimmune/Hashimoto thyroiditis (4). Isolated increase in TSH levels can be due to Subclinical inflammation, drugs like amphetamines, Iodine containing drug and dopamine antagonist e.g. domperidone and other physiological reasons.
3	Normal/Low	Low	Low	Low	(1) Secondary and Tertiary Hypothyroidism
4	Low	High	High	High	(1) Primary Hyperthyroidism (Graves Disease) (2) Multinodular Goitre (3)Toxic Nodular Goitre (4) Thyroiditis (5) Over treatment of thyroid hormone (6) Drug effect e.g. Glucocorticoids, dopamine, T4 replacement therapy (7) First trimester of Pregnancy
5	Low	Normal	Normal	Normal	(1) Subclinical Hyperthyroidism
6	High	High	High	High	(1) TSH secreting pituitary adenoma (2) TRH secreting tumor
7	Low	Low	Low	Low	(1) Central Hypothyroidism (2) Euthyroid sick syndrome (3) Recent treatment for Hyperthyroidism
8	Normal/Low	Normal	Normal	High	(1) T3 thyrotoxicosis (2) Non-Thyroidal illness
9	Low	High	High	Normal	(1) T4 Ingestion (2) Thyroiditis (3) Interfering Anti TPO antibodies

REF: 1. TIETZ Fundamentals of Clinical chemistry 2.Guidlines of the American Thyroid association duriing pregnancy and Postpartum, 2011.
NOTE: It is advisable to detect Free T3,FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.TSH is not affected by variation in thyroid - binding protein. TSH has a diurnal rhythm, with peaks at 2:00 - 4:00 a.m. And troughs at 5:00 - 6:00 p.m. With ultradian variations.

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

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DIAGNOSTIC REPORT

Patient Ref. No. 775000002237538

SRL Diagnostics

CLIENT CODE : C000138375

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CIN - U74899PB1995PLC045956
Email : srl.jodhpur@gmail.com

PATIENT NAME : JEEVAN RAM KARWA PATIENT ID : **JEEVM30016461**

ACCESSION NO : **0061WA002138** AGE : 59 Years SEX : Male ABHA NO :

DRAWN : 28/01/2023 00:00 RECEIVED : 30/01/2023 15:39 REPORTED : 04/02/2023 13:34

REFERRING DOCTOR : DR. BOB PACKAGE CLIENT PATIENT ID :

Test Report Status **Final**	Results	Biological Reference Interval	Units
ABO GROUP	TYPE B		
METHOD : FORWARD/REVERSE			
RH TYPE	POSITIVE		
METHOD : FORWARD/REVERSE			

Interpretation(s)
BLOOD COUNTS,EDTA WHOLE BLOOD-The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.
RBC AND PLATELET INDICES-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait
(<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.
WBC DIFFERENTIAL COUNT-The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to show mild disease.
(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients A.-P. Yang, et al. International Immunopharmacology 84 (2020) 106504
This ratio element is a calculated parameter and out of NABL scope.
ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD-**TEST DESCRIPTION** :-
Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of clear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

ESR is not diagnostic it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition.CRP is superior to ESR because it is more sensitive and reflects a more rapid change.
TEST INTERPRETATION

Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy, Estrogen medication, Aging.
Finding a very accelerated ESR**(>100 mm/hour)** in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).
In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm /hr(95 if anemic). ESR returns to normal 4th week post partum.
Decreased in: Polycythermia vera, Sickle cell anemia

LIMITATIONS

False elevated ESR : Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia
False Decreased : Poikilocytosis,(SickleCells,spherocytes),Microcytosis, Low fibrinogen, Very high WBC counts, Drugs(Quinine,

salicylates)

REFERENCE :
1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition 2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin 3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis,10th edition.
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-**Used For**:

1.Evaluating the long-term control of blood glucose concentrations in diabetic patients.
2.Diagnosing diabetes.
3.Identifying patients at increased risk for diabetes (prediabetes).
The ADA recommends measurement of HbA1c (typically 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 determine whether a patients metabolic control has remained continuously within the target range.
1.eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
2. eAG gives an evaluation of blood glucose levels for the last couple of months.
3. eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c - 46.7

HbA1c Estimation can get affected due to :

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I.Shortened Erythrocyte survival : Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss,hemolytic anemia) will falsely lower HbA1c test results.Fructosamine is recommended in these patients which indicates diabetes control over 15 days.
II.Vitamin C & E are reported to falsely lower test results.(possibly by inhibiting glycation of hemoglobin.
III.Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia,uremia, hyperbilirubinemia, chronic alcoholism,chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods,falsely increasing results.
IV.Interference of hemoglobinopathies in HbA1c estimation is seen in
a.Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
b.Heterozygous state detected (D10 is corrected for HbS & HbC trait.)
c.HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy
GLUCOSE FASTING,FLUORIDE PLASMA-**TEST DESCRIPTION**
Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and sothat no glucose is excreted in the urine.
Increased in
Diabetes mellitus, Cushing' s syndrome (10 – 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.
Decreased in
Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency, hypopituitarism,diffuse liver disease, malignancy (adrenocortical, stomach,fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases(e.g., galactosemia),Drugs- insulin, ethanol, propranolol sulfonylureas,tolbutamide, and other oral hypoglycemic agents.
NOTE:
Hypoglycemia is defined as a glucoseof < 50 mg/dL in men and< 40 mg/dL in women.
While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals.Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.
High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.
GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.Additional test HbA1c
LIVER FUNCTION PROFILE, SERUM-LIVER FUNCTION PROFILE
Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice.Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors &Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.
AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver,liver cancer,kidney failure,hemolytic anemia,pancreatitis,hemochromatosis. AST levels may also increase after a heart attack or strenuous activity.ALT test measures the amount of this enzyme in the blood.ALT is found mainly in the liver, but also in smaller amounts in the kidneys,heart,muscles, and pancreas.It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis,sometimes due to a viral infection,ischemia to the liver,chronic hepatitis,obstruction of bile ducts,cirrhosis.
ALP is a protein found in almost all body tissues.Tissues with higher amounts of ALP include the liver,bile ducts and bone.Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Paget'''''''s disease,Rickets,Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia,Malnutrition,Protein deficiency,Wilson'''''''s disease.GGT is an enzyme found in cell membranes of many tissues mainly in the liver,kidney and pancreas.It is also found in other tissues including intestine,spleen,heart, brain and seminal vesicles.The highest concentration is in the kidney,but the liver is considered the source of normal enzyme activity.Serum GGT has been widely used as an index of liver dysfunction.Elevated serum GGT activity can be found in diseases of the liver,biliary system and pancreas.Conditions that increase serum GGT are obstructive liver disease,high alcohol consumption and use of enzyme-inducing drugs etc.Serum total protein,also known as total protein,is a biochemical test for measuring the total amount of protein in serum.Protein in the plasma is made up of albumin and globulin.Higher-than-normal levels may be due to:Chronic inflammation or infection,including HIV and hepatitis B or C,Multiple myeloma,Waldenstrom'''''''s disease.Lower-than-normal levels may be due to: Agammaglobulinemia,Bleeding (hemorrhage),Burns,Glomerulonephritis,Liver disease, Malabsorption,Malnutrition,Nephrotic syndrome,Protein-losing enteropathy etc.Human serum albumin is the most abundant protein in human blood plasma.It is produced in the liver.Albumin constitutes about half of the blood serum protein.Low blood albumin levels (hypoalbuminemia) can be caused by:Liver disease like cirrhosis of the liver, nephrotic syndrome,protein-losing enteropathy,Burns,hemodilution,increased vascular permeability or decreased lymphatic clearance,malnutrition and wasting etc
BLOOD UREA NITROGEN (BUN), SERUM-Causes of Increased levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)
Causes of decreased level include Liver disease, SIADH.
CREATININE, SERUM-Higher than normal level may be due to:
• Blockage in the urinary tract
• Kidney problems, such as kidney damage or failure, infection, or reduced blood flow
• Loss of body fluid (dehydration)
• Muscle problems, such as breakdown of muscle fibers
• Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to:
• Myasthenia Gravis

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• Muscular dystrophy
URIC ACID, SERUM-**Causes of Increased levels:**-Dietary(High Protein Intake,Prolonged Fasting,Rapid weight loss),Gout,Lesch nyhan syndrome,Type 2 DM,Metabolic syndrome
Causes of decreased levels-Low Zinc intake,OCP,Multiple Sclerosis
TOTAL PROTEIN, SERUM-Serum total protein,also known as total protein, is a biochemical test for measuring the total amount of protein in serum..Protein in the plasma is made up of albumin and globulin

Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom''''''''''''''''''''''''''''''''s disease
Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage),Burns,Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome,Protein-losing enteropathy etc.
ALBUMIN, SERUM-Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance,malnutrition and wasting etc.
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

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

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

****End Of Report****

Please visit www.srlworld.com for related Test Information for this accession

Dr. Itisha Dhiman
Pathologist

DIAGNOSTIC REPORT

Patient Ref. No. 775000002237538

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CONDITIONS OF LABORATORY TESTING & REPORTING

1. It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
2. All tests are performed and reported as per the turnaround time stated in the SRL Directory of Services.
3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.
4. A requested test might not be performed if:
   i. Specimen received is insufficient or inappropriate
   ii. Specimen quality is unsatisfactory
   iii. Incorrect specimen type
   iv. Discrepancy between identification on specimen container label and test requisition form
5. SRL confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.
7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.
8. Test results cannot be used for Medico legal purposes.
9. In case of queries please call customer care (91115 91115) within 48 hours of the report.

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