





CLIENT CODE: C000138356 **CLIENT'S NAME AND ADDRESS:** KRISHNAMUR VANDANA KODANDAPANI

PRIME SQUARE BUILDING, PLOT NO 1, GAIWADI INDUSTRIAL ESTATE, S.V. ROAD, GOREGAON (W)

MUMBAI, 400062 MAHARASHTRA, INDIA

Tel: 9111591115, Fax: 022 - 67801212

CIN - U74899PB1995PLC045956

PATIENT NAME: KRISHNAMUR VANDANA KODANDAPANI PATIENT ID: KRISF1306822

0002WB02139 AGE: 40 Years SEX: Female ABHA NO:

DRAWN: 11/02/2023 08:44 RECEIVED: 11/02/2023 08:46 13/02/2023 18:11 REPORTED:

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status Biological Reference Interval Units Results <u>Final</u>

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

HEMOGLOBIN (HB) METHOD: PHOTOMETRIC MEASUREMENT RED BLOOD CELL (RBC) COUNT METHOD: COULTER PRINCIPLE WHITE BLOOD CELL (WBC) COUNT METHOD: COULTER PRINCIPLE PLATELET COUNT 8.0 4.46 3.8 - 4.8 4.0 - 10.0 4.0 - 10.0 150 - 410	g/dL mil/μL
RED BLOOD CELL (RBC) COUNT METHOD: COULTER PRINCIPLE WHITE BLOOD CELL (WBC) COUNT METHOD: COULTER PRINCIPLE 4.46 3.8 - 4.8 4.0 - 10.0	mil/µL
METHOD : COULTER PRINCIPLE WHITE BLOOD CELL (WBC) COUNT 6.00 4.0 - 10.0 METHOD : COULTER PRINCIPLE	mil/μL
WHITE BLOOD CELL (WBC) COUNT 6.00 4.0 - 10.0 METHOD: COULTER PRINCIPLE	
METHOD : COULTER PRINCIPLE	
	thou/µL
PLATELET COUNT 334 150 - 410	
	thou/µL
METHOD: ELECTRONIC IMPEDENCE & MICROSCOPY	
RBC AND PLATELET INDICES	
HEMATOCRIT (PCV) 27.7 Low 36.0 - 46.0	%
METHOD: CALCULATED PARAMETER	
MEAN CORPUSCULAR VOLUME (MCV) 62.1 Low 83.0 - 101.0	fL
METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM	
MEAN CORPUSCULAR HEMOGLOBIN (MCH) 17.9 Low 27.0 - 32.0	pg
METHOD: CALCULATED PARAMETER	
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD: CALCULATED PARAMETER Low 31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) 20.6 High 11.6 - 14.0	%
METHOD: DERIVED PARAMETER FROM RBC HISTOGRAM	
MENTZER INDEX 13.9	
MEAN PLATELET VOLUME (MPV) 9.0 6.8 - 10.9	fL
METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM	
WBC DIFFERENTIAL COUNT	
NEUTROPHILS 67 40 - 80	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY	
LYMPHOCYTES 24 20 - 40	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY	
MONOCYTES 7 2.0 - 10.0	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY	
EOSINOPHILS 2 1.0 - 6.0	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY	
BASOPHILS 0 0 - 1	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY	
ABSOLUTE NEUTROPHIL COUNT 4.02 2.0 - 7.0	thou/µL











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METHOD : CALCULATED PAR	RAMETER				
ABSOLUTE LYMPHOCY	TE COUNT	1.44		1.0 - 3.0	thou/µL
METHOD : CALCULATED PAR	RAMETER				
ABSOLUTE MONOCYTE	COUNT	0.42		0.2 - 1.0	thou/µL
METHOD : CALCULATED PAR	RAMETER				
ABSOLUTE EOSINOPH	IL COUNT	0.12		0.02 - 0.50	thou/µL
METHOD : CALCULATED PAR	RAMETER				
ABSOLUTE BASOPHIL	COUNT	0	Low	0.02 - 0.10	thou/µL
METHOD : CALCULATED PAR	RAMETER				
NEUTROPHIL LYMPHOO	CYTE RATIO (NLR)	2.8			
METHOD : CALCULATED					
MORPHOLOGY					
RBC		Moderate anisor	ooikilocytos	is. Microcytic hypochromic with	ovalocytes.
METHOD : MICROSCOPIC EX	XAMINATION	·	•	, ,,	•
WBC		Normal morphol	logy		
METHOD : MICROSCOPIC EX	XAMINATION	·	<i>5,</i>		
PLATELETS		Adequate in smo	ear		
METHOD : ELECTRONIC IMP	PEDENCE & MICROSCOPY	·			
* ERYTHROCYTE SEI	DIMENTATION RATE (E	SR),WHOLE			
BLOOD					
E.S.R		19		0 - 20	mm at 1 hr
•	OTOMETRICAL CAPILLARY STOPPE	D FLOW KINETIC ANALYSIS)			
* GLUCOSE FASTING	G,FLUORIDE PLASMA				
FBS (FASTING BLOOD	SUGAR)	110	High	Normal <100 Impaired fasting glucose:100 125 Diabetes mellitus: > = 126 (o more than 1 occassion)	
METIOD - CDECTROPHOTON	AETDY HEVOKINA CE			(ADA guidelines 2021)	
METHOD : SPECTROPHOTON		TA WILOI F			
BLOOD	MOGLOBIN(HBA1C), ED	TA WHOLE			
HBA1C		5.1		Non-diabetic Adult < 5.7 Pre-diabetes 5.7 - 6.4 Diabetes diagnosis: > or = 6.9 Therapeutic goals: < 7.0 Action suggested: > 8.0 (ADA Guideline 2021)	%
METHOD : ION- EXCHANGE	HPLC			•	
ESTIMATED AVERAGE	GLUCOSE(EAG)	99.7		< 116	mg/dL

* GLUCOSE, POST-PRANDIAL, PLASMA











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PPBS(POST PRANDIAL	BLOOD SUGAR)	136		Normal <140 Impaired glucose tolerance:140 to 199 Diabetes mellitus : > = 200 (on more than 1 occassion) ADA guideline 2021	mg/dL
METHOD : SPECTROPHOTOM					
LIPID PROFILE, SER	UM				
CHOLESTEROL, TOTAL		128		Desirable : < 200 Borderline : 200 - 239 High : > / = 240	mg/dL
	IETRY, ENZYMATIC COLORIMETR	IC - CHOLETSEROL OXIDASE, ES	STERASE, PERC		
TRIGLYCERIDES		88		Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
	TETRY, ENZYMATIC ENDPOINT W		_		
HDL CHOLESTEROL		33	Low	At Risk: < 40 Desirable: > or = 60	mg/dL
METHOD : SPECTROPHOTOM	TETRY, HOMOGENEOUS DIRECT	ENZYMATIC COLORIMETRIC		Desirable. > 01 = 00	
CHOLESTEROL LDL		77		Optimal: < 100 Near optimal/above optimal: 129 Borderline high: 130-159 High: 160-189 Very high: = 190	mg/dL 100-
METHOD : CALCULATED PAR	RAMETER				
NON HDL CHOLESTER	DL	95		Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
METHOD : CALCULATED PAR	RAMETER				
VERY LOW DENSITY LI METHOD : CALCULATED PAR		18.0		< or = 30.0	mg/dL
CHOL/HDL RATIO		3.9		Low Risk: 3.3 - 4.4 Average Risk: 4.5 - 7.0 Moderate Risk: 7.1 - 11.0 High Risk: > 11.0	
METHOD : CALCULATED PAR	RAMETER	2.6		- · · · · · - · · - · · - · · · · · · ·	
LDL/HDL RATIO		2.6		Desirable/Low Risk: 0.5 - 3.0 Borderline/Moderate Risk: 3.1 6.0 High Risk: > 6.0	



METHOD: CALCULATED PARAMETER











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Biological Reference Interval Units

Interpretation(s)

Test Report Status

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

Results

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.

SEX: Female

- 3)HDL-C plays a crucial role in the initial step of reverse cholesterol transport, this considered to be the primary atheroprotective function of
- 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 g	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 1	major risk factors or evidence of end organ damage 3.			
	Familial Homozygous Hypercholesterolemi	a			
High Risk	1. Three major ASCVD risk factors. 2. Dia	betes 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 (Ath	Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors				
1. Age $>$ or $=$ 45 year	Age $>$ or $= 45$ years in males and $>$ or $= 55$ years in females 3. Current Cigarette smoking or tobacco use				
2. Family history of p	2. Family history of premature ASCVD 4. High blood pressure				
5. Low HDL	5. Low HDL				
		11 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1			

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

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











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Test Report Status

Low Risk

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<u>Final</u>

<100

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Biological Reference Interval

>OR = 160

KRISF1306822

Units

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Extreme Risk Group	<50 (Optional goal	< 80 (Optional goal	>OR = 50	>OR = 80
Category A	< OR = 30)	<OR = 60)		
Extreme Risk Group	<or 30<="" =="" td=""><td><or 60<="" =="" td=""><td>> 30</td><td>>60</td></or></td></or>	<or 60<="" =="" td=""><td>> 30</td><td>>60</td></or>	> 30	>60
Category B				
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

<130

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.

>OR = 130*

* LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.45	Upto 1.2	mg/dL
METHOD: SPECTROPHOTOMETRY, COLORIMETRIC -DIAZO METHOD			-
BILIRUBIN, DIRECT	0.21	< or = 0.3	mg/dL
METHOD: SPECTROPHOTOMETRY, JENDRASSIK & GROFF - DIAZOTIZ	ATION		
BILIRUBIN, INDIRECT	0.24	0.0 - 0.9	mg/dL
METHOD: CALCULATED PARAMETER			
TOTAL PROTEIN	7.0	6.0 - 8.0	g/dL
METHOD: SPECTROPHOTOMETRY, COLORIMETRIC-BIURET, REAGEN	T BLANK, SERUM BLANK		
ALBUMIN	4.1	3.97 - 4.94	g/dL
METHOD: SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DY	E BINDING		
GLOBULIN	2.9	2.0 - 3.5	g/dL
METHOD: CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	1.4	1.0 - 2.1	RATIO
METHOD: CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	11	Upto 32	U/L
METHOD: SPECTROPHOTOMETRY, WITHOUT PYRIDOXAL PHOSPHATE	ACTIVATION(P5P) - IFCC		
ALANINE AMINOTRANSFERASE (ALT/SGPT)	12	Upto 33	U/L
METHOD: SPECTROPHOTOMETRY, WITHOUT PYRIDOXAL PHOSPHATE	ACTIVATION(P5P) - IFCC		
ALKALINE PHOSPHATASE	96	35 - 104	U/L
METHOD: SPECTROPHOTOMETRY, PNPP, AMP BUFFER - IFCC			
GAMMA GLUTAMYL TRANSFERASE (GGT)	10	< 40	U/L
METHOD: SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC - G-G	LUTAMYL-CARBOXY-NITROANILIDE - 1	FCC	
LACTATE DEHYDROGENASE	92	< 223	U/L
METHOD: SPECTROPHOTOMETRY, LACTATE TO PYRUVATE - UV-IFCC			
* BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN	6	6 - 20	mg/dL





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







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METHOD : SPECTROPHOTOM	IETRY, UREASE -COLORIM	ETRIC				
* CREATININE, SERU	JM					
CREATININE		0.70		0.60 - 1.10	n	ng/dL
METHOD: SPECTROPHOTOM	1ETRY, JAFFE'S ALKALINE I	PICRATE KINETIC - RATE BLANKED - IFCO	C-IDMS STAN	IDARIZED		
BUN/CREAT RATIO						
BUN/CREAT RATIO		8.70		8 - 15		
METHOD : CALCULATED PAR	RAMETER					
* URIC ACID, SERUM	1					
URIC ACID		4.2		2.4 - 5.7	n	ng/dL
METHOD : SPECTROPHOTOM	IETRY, ENZYMATIC COLOR	IMETRIC- URICASE				
* TOTAL PROTEIN, S	SERUM					
TOTAL PROTEIN		7.0		6.0 - 8.0	g	/dL
METHOD : SPECTROPHOTOM	METRY, COLORIMETRIC -BI	URET, REAGENT BLANK, SERUM BLANK				
* ALBUMIN, SERUM						
ALBUMIN		4.1		3.97 - 4.94	g	/dL
METHOD : SPECTROPHOTOM	METRY, BROMOCRESOL GR	EEN(BCG) - DYE BINDING				
GLOBULIN						
GLOBULIN		2.9		2.0 - 3.5	g	/dL
METHOD : CALCULATED PAR	RAMETER					
* ELECTROLYTES (N.	A/K/CL), SERUM					
SODIUM, SERUM		136		136 - 145	n	nmol/L
METHOD : ISE INDIRECT						
POTASSIUM, SERUM		4.40		3.5 - 5.1	n	nmol/L
METHOD : ISE INDIRECT						
CHLORIDE, SERUM		107	High	98 - 106	n	nmol/L
METHOD : ISE INDIRECT						











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Interpretation(s)

Sodium	Potassium	Chloride
Decreased in: CCF, cirrhosis,	Decreased in: Low potassium	Decreased in: Vomiting, diarrhea,
vomiting, diarrhea, excessive	intake,prolonged vomiting or diarrhea,	renal failure combined with salt
sweating, salt-losing	RTA types I and II,	deprivation, over-treatment with
nephropathy,adrenal insufficiency,	hyperaldosteronism, Cushing's	diuretics, chronic respiratory acidosis,
nephrotic syndrome, water	syndrome,osmotic diuresis (e.g.,	diabetic ketoacidosis, excessive
intoxication, SIADH. Drugs:	hyperglycemia),alkalosis, familial	sweating, SIADH, salt-losing
thiazides, diuretics, ACE inhibitors,	periodic paralysis,trauma	nephropathy, porphyria, expansion of
chlorpropamide,carbamazepine,anti	(transient).Drugs: Adrenergic agents,	extracellular fluid volume,
depressants (SSRI), antipsychotics.	diuretics.	adrenalinsufficiency,
		hyperaldosteronism, metabolic
		alkalosis. Drugs: chronic
		laxative,corticosteroids, diuretics.
Increased in: Dehydration	Increased in: Massive hemolysis,	Increased in: Renal failure, nephrotic
(excessivesweating, severe	severe tissue damage, rhabdomyolysis,	syndrome, RTA,dehydration,
vomiting or diarrhea),diabetes	acidosis, dehydration,renal failure,	overtreatment with
mellitus, diabetesinsipidus,	Addison's disease, RTA type IV,	saline,hyperparathyroidism, diabetes
hyperaldosteronism, inadequate	hyperkalemic familial periodic	insipidus, metabolic acidosis from
water intake. Drugs: steroids,	paralysis. Drugs: potassium salts,	diarrhea (Loss of HCO3-), respiratory
licorice,oral contraceptives.	potassium- sparing diuretics,NSAIDs,	alkalosis, hyperadre no corticism.
	beta-blockers, ACE inhibitors, high-	Drugs: acetazolamide, and rogens,
	dose trimethoprim-sulfamethoxazole.	hydrochlorothiazide, salicylates.
Interferences: Severe lipemia or	Interferences: Hemolysis of sample,	Interferences:Test is helpful in
hyperproteinemi, if sodium analysis	delayed separation of serum,	assessing normal and increased anion
involves a dilution step can cause	prolonged fist clenching during blood	gap metabolic acidosis and in
spurious results. The serum sodium	drawing, and prolonged tourniquet	distinguishing hypercalcemia due to
falls about 1.6 mEq/L for each 100	placement. Very high WBC/PLT counts	hyperparathyroidism (high serum
mg/dL increase in blood glucose.	may cause spurious. Plasma potassium	chloride) from that due to malignancy
	levels are normal.	(Normal serum chloride)

PHYSICAL EXAMINATION, URINE

COLOR	PALE YELLOW	
APPEARANCE	CLEAR	
CHEMICAL EXAMINATION, URINE		
PH	6.5	5.00 - 7.50
SPECIFIC GRAVITY	1.015	1.010 - 1.030
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	NOT DETECTED	
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)	1-2	0-5	/HPF
EPITHELIAL CELLS	0-1	0-5	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	NOT DETECTED	NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	

METHOD: URINE ROUTINE & MICROSCOPY EXAMINATION BY INTEGRATED AUTOMATED SYSTEM

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











KRISF1306822

CLIENT CODE: C000138356 **CLIENT'S NAME AND ADDRESS:** KRISHNAMUR VANDANA KODANDAPANI

METHOD: SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY

PRIME SQUARE BUILDING, PLOT NO 1, GAIWADI INDUSTRIAL ESTATE, S.V. ROAD, GOREGAON (W)

MUMBAI, 400062 MAHARASHTRA, INDIA

Tel: 9111591115, Fax: 022 - 67801212 CIN - U74899PB1995PLC045956

PATIENT NAME: KRISHNAMUR VANDANA KODANDAPANI PATIENT ID:

0002WB02139 AGE: 40 Years SEX: Female ABHA NO:

RECEIVED: 11/02/2023 08:46 DRAWN: 11/02/2023 08:44 13/02/2023 18:11 REPORTED:

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status	<u>Final</u>	Results		Biological Reference Interv	al Units
* THYPOTO DANIEL	CEDIIM				
* THYROID PANEL, S	SERUM	95.4		Non-Pregnant Women 80.0 - 200.0 Pregnant Women 1st Trimester:105.0 - 230.0 2nd Trimester:129.0 - 262.0 3rd Trimester:135.0 - 262.0	ng/dL
METHOD : COMPETITIVE EL	ECTROCHEMILUMINESCEN	CE IMMUNOASSAY			
T4		7.41		Non-Pregnant Women 5.10 - 14.10 Pregnant Women 1st Trimester: 7.33 - 14.80 2nd Trimester: 7.93 - 16.10 3rd Trimester: 6.95 - 15.70	µg/dL
METHOD : COMPΕΤΙΤΊVE EL	ECTROCHEMILUMINESCEN	CE IMMUNOASSAY			
TSH (ULTRASENSITIVE	Ē)	4.370	High	Non Pregnant Women 0.27 - 4.20 Pregnant Women 1st Trimester: 0.33 - 4.59 2nd Trimester: 0.35 - 4.10 3rd Trimester: 0.21 - 3.15	μIU/mL











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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 during 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.

PAPANICOLAOU SMEAR

TEST METHOD CONVENTIONAL GYNEC CYTOLOGY

SPECIMEN TYPE TWO UNSTAINED CERVICAL SMEARS RECEIVED

2CW-3976

REPORTING SYSTEM 2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY











KRISF1306822

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SPECIMEN ADEQUACY SMEARS ARE SATISFACTORY FOR EVALUATION.

MICROSCOPY THE SMEAR SHOWS MAINLY SUPERFICIAL SQUAMOUS CELLS, FEW

INTERMEDIATE SQUAMOUS CELLS AND FEW POLYMORPHS.

INTERPRETATION / RESULT NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY

Comments

Suggestions / Guidelines: (REF: THE BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY,2014, 3rd Edition)
PAP RE-TESTING AT 3 YEARS

- 1) Please note papanicolaou smear study is a screening procedure for cervical cancer with inherent false negative results, hence should be interpreted with caution.
- 2) No cytologic evidence of hpv infection in the smears studied.
- 3) Primary screening of papanicolaou smears is carried out by cytotechnologist with 100% rescreening and reporting by surgical pathologist.

PHYSICAL EXAMINATION, STOOL

COLOUR BROWN
CONSISTENCY SEMI FORMED

MUCUS NOT DETECTED NOT DETECTED

VISIBLE BLOOD ABSENT ABSENT ABSENT

ADULT PARASITE NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

CHEMICAL EXAMINATION, STOOL

STOOL PH 6.0

OCCULT BLOOD NOT DETECTED NOT DETECTED

METHOD: MODIFIED GUAIAC METHOD

MICROSCOPIC EXAMINATION, STOOL

PUS CELLS NOT DETECTED /hpf
RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF

METHOD: MICROSCOPIC EXAMINATION

CYSTS NOT DETECTED NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

OVA NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

LARVAE NOT DETECTED NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

TROPHOZOITES NOT DETECTED NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

FAT ABSENT CHARCOT LEYDEN CRYSTALS ABSENT











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Test Report Status <u>Final</u> Results Biological Reference Interval Units

Interpretation(s)

Stool routine analysis is only a screening test for disorders of gastrointentestinal tract like infection, malabsorption, etc. The following table describes the probable conditions, in which the analytes are present in stool.

PRESENCE OF	CONDITION		
Pus cells	Pus in the stool is an indication of infection		
Red Blood cells	Parasitic or bacterial infection or an inflammatory bowel condition such as		
	ulcerative colitis		
Parasites	Infection of the digestive system. Stool examination for ova and parasite detection of parasitic infestation of gastrointestinal tract. Various forms of parasite that can be detected include cyst, trophozoite and larvae. One negative result does not rule out the possibility of parasitic infestation. Intermittent shedding of parasites warrants examinations of multiple specimens tested on consecutive days. Stool specimens for parasitic examination should be collected before initiation of antidiarrheal therapy or antiparasitic therapy. This test does not detect presence of opportunistic parasites like Cyclospora, Cryptosporidia and Isospora species. Examination of Ova and Parasite has been carried out by direct and concentration techniques.		
Mucus	Mucus is a protective layer that lubricates, protects& reduces damage due to bacteria or viruses.		
Charcot-Leyden crystal	Parasitic diseases.		
Ova & cyst	Ova & cyst indicate parasitic infestation of intestine.		
Frank blood	Bleeding in the rectum or colon.		
Occult blood	Occult blood indicates upper GI bleeding.		
Macrophages	Macrophages in stool are an indication of infection as they are protective cells		
Epithelial cells	Epithelial cells that normally line the body surface and internal organs show up in stool when there is inflammation or infection.		
Fat	Increased fat in stool maybe seen in conditions like diarrhoea or malabsorption.		
рН	Normal stool pH is slightly acidic to neutral. Breast-fed babies generally have an acidic stool.		

ADDITIONAL STOOL TESTS:

- Stool Culture: This test is done to find cause of GI infection, make decision about best treatment for GI infection & to find out if treatment for GI infection worked.
- **2. Fecal Calprotectin**: It is a marker of intestinal inflammation. This test is done to differentiate Inflammatory Bowel Disease (IBD) from Irritable Bowel Syndrome (IBS).
- 3. Fecal Occult Blood Test(FOBT): This test is done to screen for colon cancer & to evaluate possible cause of unexplained anaemia.
- **Clostridium Difficile Toxin Assay**: This test is strongly recommended in healthcare associated bloody or waterydiarrhoea, due to overuse of broad spectrum antibiotics which alter the normal GI flora.
- 5. <u>Biofire (Film Array) GI PANEL</u>: In patients of Diarrhoea, Dysentry, Rice watery Stool, FDA approved, Biofire Film Array Test,(Real Time Multiplex PCR) is strongly recommended as it identifies organisms, bacteria,fungi,virus ,parasite and other opportunistic pathogens, Vibrio cholera infections only in 3 hours. Sensitivity 96% & Specificity 99%.
- 6. Rota Virus Immunoassay: This test is recommended in severe gastroenteritis in infants & children associated with watery diarrhoea, vomitting& abdominal cramps. Adults are also affected. It is highly contagious in nature.











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CIN - U74899PB1995PLC045956

PATIENT NAME: KRISHNAMUR VANDANA KODANDAPANI

KRISF1306822

0002WB02139 AGE: 40 Years SEX: Female ABHA NO: ACCESSION NO:

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PATIENT ID:

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Test Report Status Results Biological Reference Interval Units **Final**

* ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP 0

METHOD: HAEMAGGLUTINATION (AUTOMATED)

POSITIVE RH TYPF

METHOD: HAEMAGGLUTINATION (AUTOMATED)

* XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

* TMT OR ECHO

TMT OR ECHO NEGATIVE

* ECG

ECG WITHIN NORMAL LIMITS

* MEDICAL HISTORY

RELEVANT PRESENT HISTORY HYPOTHROID SINCE 3 YRS JOINT PAIN ON AND OFF

COVID 19 IN 2021 RELEVANT PAST HISTORY

JAUNDICE IN CHILDHOOD

RELEVANT PERSONAL HISTORY **NOT SIGNIFICANT**

MENSTRUAL HISTORY (FOR FEMALES) **REGULAR** LMP (FOR FEMALES) 21/1/23

RELEVANT FAMILY HISTORY HYPERTENSION, DIABETES

HISTORY OF MEDICATIONS **NOT SIGNIFICANT**

* ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.58 mts WEIGHT IN KGS. Kgs 67.1

BMI 27 BMI & Weight Status as follows: kg/sqmts

Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obese

* GENERAL EXAMINATION

MENTAL / EMOTIONAL STATE **NORMAL** PHYSICAL ATTITUDE NORMAL GENERAL APPEARANCE / NUTRITIONAL STATUS **OVERWEIGHT BUILT / SKELETAL FRAMEWORK AVERAGE** FACIAL APPEARANCE NORMAL



Page 15 Of 21 Scan to View Report







CLIENT CODE: C000138356

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ACCESSION NO: **0002WB02139** AGE: 40 Years SEX: Female ABHA NO:

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Test Report Status <u>Final</u>	Results	Biological Reference Interval	Units
SKIN	NORMAL		
UPPER LIMB	NORMAL		
LOWER LIMB	NORMAL		
NECK	NORMAL		
NECK LYMPHATICS / SALIVARY GLANDS	NOT ENLARGED OR TENDE	R	
THYROID GLAND	NOT ENLARGED		
CAROTID PULSATION	NORMAL		

TEMPERATURE NORMAL
PULSE 80/MIN REGULAR, ALL P

PULSE 80/MIN REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID

BRUIT

RESPIRATORY RATE NORMAL

* CARDIOVASCULAR SYSTEM

BP 110/70 MM HG mm/Hg

(SUPINE) NORMAL

PERICARDIUM NORMAL APEX BEAT NORMAL

HEART SOUNDS S1, S2 HEARD NORMALLY

MURMURS ABSENT

* RESPIRATORY SYSTEM

SIZE AND SHAPE OF CHEST

MOVEMENTS OF CHEST

BREATH SOUNDS INTENSITY

NORMAL

BREATH SOUNDS QUALITY VESICULAR (NORMAL)

ADDED SOUNDS ABSENT

* PER ABDOMEN

APPEARANCE NORMAL
VENOUS PROMINENCE ABSENT
LIVER NOT PALPABLE

SPLEEN NOT PALPABLE
HERNIA ABSENT

* CENTRAL NERVOUS SYSTEM

HIGHER FUNCTIONS NORMAL
CRANIAL NERVES NORMAL
CEREBELLAR FUNCTIONS NORMAL
SENSORY SYSTEM NORMAL











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REFERRING DOCTOR:	SELF	CLIENT PATIENT ID.				
Test Report Status	<u>Final</u>	Results	Biological Reference Interval	Units		
MOTOR SYSTEM		NORMAL				
REFLEXES		NORMAL				
* MUSCULOSKELETA	N CVCTEM	NORMAL				
SPINE	AL SISILM	NORMAL				
JOINTS		NORMAL				
* BASIC EYE EXAMI	NATION	NORMAL				
CONJUNCTIVA	MATION	NORMAL				
EYELIDS		NORMAL				
EYE MOVEMENTS		NORMAL				
		NORMAL				
	CORNEA		REDUCE VISUAL ACUITY (6/12)			
	DISTANT VISION RIGHT EYE WITHOUT GLASSES DISTANT VISION LEFT EYE WITHOUT GLASSES		REDUCE VISUAL ACUITY (6/12)			
NEAR VISION RIGHT EYE WITHOUT GLASSES		WITHIN NORMAL LIMIT (N6)				
NEAR VISION LEFT EY		WITHIN NORMAL LIMIT (N6)				
COLOUR VISION			NORMAL (17/17)			
* BASIC ENT EXAMI	NATION	1101111112 (27/17)				
EXTERNAL EAR CANAL	-	NORMAL				
TYMPANIC MEMBRANE		NORMAL				
NOSE		NO ABNORMALITY DE	ЕТЕСТЕР			
SINUSES		NORMAL				
THROAT		NO ABNORMALITY DE	ETECTED			
TONSILS		NOT ENLARGED				
* BASIC DENTAL EX	AMINATION					
TEETH		NORMAL				
GUMS	GUMS		HEALTHY			
* SUMMARY						
RELEVANT HISTORY		NOT SIGNIFICANT				
RELEVANT GP EXAMINATION FINDINGS		NOT SIGNIFICANT				
RELEVANT LAB INVES	ΠGATIONS	LOW HAEMOGLOBIN RAISED FBS (110) LOW HDL CHOLESTE RAISED CHLORIDE (RAISED TSH (4.370)	ROL (33)			
RELEVANT NON PATHOLOGY DIAGNOSTICS		USG-MULTIPLE ANTERIOR AND POSTERIOR INTRAMURAL UTERINE FIBROIDS.				











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Test Report Status Results Biological Reference Interval Units **Final**

REMARKS / RECOMMENDATIONS LOW HAEMOGLOBIN, LOW HDL CHOLESTEROL, RAISED TSH, RAISED FBS

IRON RICH DIET

OMEGA 3 FATS SUPPLEMENTS

FOLLOW UP WITH PHYSICIAN FOR ANEMIA

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 fibringgen, 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. 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. GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-**Used For**:











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PATIENT NAME: KRISHNAMUR VANDANA KODANDAPANI

0002WB02139 AGE: 40 Years SEX: Female ABHA NO:

DRAWN: 11/02/2023 08:44 RECEIVED: 11/02/2023 08:46 REPORTED: 13/02/2023 18:11

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status Results Biological Reference Interval Units **Final**

1. Evaluating the long-term control of blood glucose concentrations in diabetic patients.

- 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:

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, 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''' globulin. Higher-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:

- · Mvasthenia Gravis
- Muscular dystrophy

URIC ACID, ŚERUM-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











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PRIME SQUARE BUILDING, PLOT NO 1, GAIWADI INDUSTRIAL ESTATE, S.V. ROAD, GOREGAON (W)

MUMBAI, 400062 MAHARASHTRA, INDIA

Tel: 9111591115, Fax: 022 - 67801212 CIN - U74899PB1995PLC045956

PATIENT NAME: KRISHNAMUR VANDANA KODANDAPANI

PATIENT ID: KRISF1306822

0002WB02139

AGE: 40 Years

SEX: Female

ABHA NO: REPORTED:

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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.

THIS REPORT CARRIES THE SIGNATURE OF OUR LABORATORY DIRECTOR. THIS IS AN INVIOLABLE FEATURE OF OUR LAB MANAGEMENT SOFTWARE. HOWEVER, ALL EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.











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MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

* ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

MULTIPLE ANTERIOR AND POSTERIOR INTRAMURAL UTERINE FIBROIDS.

End Of Report

Please visit www.srlworld.com for related Test Information for this accession TEST MARKED WITH '*' ARE OUTSIDE THE NABL ACCREDITED SCOPE OF THE LABORATORY.

Dr. Swati Karmarkar, MD, DNB, DMRD **Consultant Radiologist**

Dr. Sneha Wadalkar, M.D (Reg.no.MMC2012/06/1868 **Junior Biochemist**

Dr.Nidhi Garg (Reg.No.MMC 2009/09/3278) Histopsthologist

Dr. Ekta Patil, MD Microbiologist

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.
- 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.
- In case of queries please call customer care (91115 91115) within 48 hours of the report.

SRL Limited

Fortis Hospital, Sector 62, Phase VIII, Mohali 160062



