DIAGNOSTIC REPORT

agilus diagnostics

PATIENT NAME : KRISHNAMUR VANDANA KODANDAPANI **REF. DOCTOR :** SELF

ACCESSION NO	: **0002WL024265**	AGE/SEX	:41 Years Female
PATIENT ID	: KRISF1306822	DRAWN	:16/12/2023 09:00:04
CLIENT PATIENT ID:		RECEIVED	: 16/12/2023 09:01:16
ABHA NO	:	REPORTED	:18/12/2023 17:02:19

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

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

XRAY-CHEST

IMPRESSION	NO ABNORMALITY DETECTED	

ECG

ECG	ECG- SINUS RHYTHM R-S TRANSITION ZONE IN V LEADS DISPLACED TO THE RIGHT OTHERWISE NORMAL ECG	

MAMOGRAPHY (BOTH BREASTS)

MAMOGRAPHY BOTH BREASTS	DONE	

MEDICAL HISTORY

RELEVANT PRESENT HISTORY	HYPOTHYROIDISM 4 YRS WEAKNESS	
RELEVANT PAST HISTORY	C-SECTION 2 COVID 19 2021	
RELEVANT PERSONAL HISTORY	NOT SIGNIFICANT	
LMP (FOR FEMALES)	7/12/2023	
RELEVANT FAMILY HISTORY	HYPERTENSION,DIABETES	
HISTORY OF MEDICATIONS	NOT SIGNIFICANT	

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS	1.57	mts
WEIGHT IN KGS.	67.3	Kgs

Dr. J N Shukla ,MBBS, AFIH
Consultant Physician

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

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BMI	27	BMI & Weight Status as follows: Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obese	kg/sqmts

GENERAL EXAMINATION

MENTAL / EMOTIONAL STATE	NORMAL
PHYSICAL ATTITUDE	NORMAL
GENERAL APPEARANCE / NUTRITIONAL STATUS	HEALTHY
BUILT / SKELETAL FRAMEWORK	AVERAGE
FACIAL APPEARANCE	NORMAL
SKIN	NORMAL
UPPER LIMB	NORMAL
LOWER LIMB	NORMAL
NECK LYMPHATICS / SALIVARY GLANDS	NOT ENLARGED OR TENDER
THYROID GLAND	NOT ENLARGED
CAROTID PULSATION	NORMAL
TEMPERATURE	NORMAL
PULSE	84/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID BRUIT
RESPIRATORY RATE	NORMAL

CARDIOVASCULAR SYSTEM

BP	110/70 MM HG (SUPINE)	mm/Hg
APEX BEAT	NORMAL	
HEART SOUNDS	NORMAL	
MURMURS	ABSENT	

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RESPIRATORY SYSTEM

SIZE AND SHAPE OF CHEST	NORMAL
MOVEMENTS OF CHEST	SYMMETRICAL
BREATH SOUNDS QUALITY	VESICULAR (NORMAL)
ADDED SOUNDS	ABSENT

PER ABDOMEN

APPEARANCE	NORMAL
LIVER	NOT PALPABLE
SPLEEN	NOT PALPABLE
HERNIA	ABSENT

CENTRAL NERVOUS SYSTEM

HIGHER FUNCTIONS	NORMAL
CRANIAL NERVES	NORMAL
CEREBELLAR FUNCTIONS	NORMAL
SENSORY SYSTEM	NORMAL
MOTOR SYSTEM	SAMPLE NOT RECEIVED
REFLEXES	NORMAL

MUSCULOSKELETAL SYSTEM

SPINE	NORMAL
JOINTS	NORMAL

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BASIC EYE EXAMINATION

CONJUNCTIVA	NORMAL
EYELIDS	NORMAL
EYE MOVEMENTS	NORMAL
CORNEA	NORMAL
DISTANT VISION RIGHT EYE WITH GLASSES	WITH GLASSES NORMAL (6/6)
DISTANT VISION LEFT EYE WITH GLASSES	WITH GLASSES NORMAL (6/6)
NEAR VISION RIGHT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT (N6)
NEAR VISION LEFT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT (N6)
COLOUR VISION	NORMAL (17/17)

BASIC ENT EXAMINATION

EXTERNAL EAR CANAL	NORMAL
TYMPANIC MEMBRANE	NORMAL
NOSE	NO ABNORMALITY DETECTED
SINUSES	CLEAR
THROAT	NO ABNORMALITY DETECTED
TONSILS	NOT ENLARGED

SUMMARY

RELEVANT HISTORY	NOT SIGNIFICANT
RELEVANT GP EXAMINATION FINDINGS	NOT SIGNIFICANT
RELEVANT LAB INVESTIGATIONS	LOW HEMOGLOBIN (7.6) RAISED EAG (116.9) RAISED FBS (105) RAISED ALKALINE PHOSPHATASE (114)
RELEVANT NON PATHOLOGY DIAGNOSTICS	USG: ANTERIOR INTRAMURAL UTERINE FIBROID.
REMARKS / RECOMMENDATIONS	LOW HEMOGLOBIN, RAISED EAG, RAISED ALKALINE PHOSPHATASE ADV- IRON RICH DIET ASV- REDUCE SWEET AND SUGAR ADV- VITAMIN D NAD VITAMIN B12 LEVEL ADV- MONITOR THS PERIODICALLY FOLLOW UP WITH PHYSICIAN FOR ANEMIA

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MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

ANTERIOR INTRAMURAL UTERINE FIBROID (5 X 3 MM).

TMT OR ECHO

CLINICAL PROFILE

2 DECHO DONE : IMPRESSION.
-GOOD LV SYSTOLIC FUNCTION AT REST. NO RWMA
-LVEF 55-60%.
-ALL VALVES STRUCTURALLY NORMAL.
-NO EVIDENCE OF PE/CLOT/VEGETATION

Interpretation(s)
MEDICAL
HISTORY-**********
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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MC-5718

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HAEMATOLOGY - CBC

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

BLOOD COUNTS,EDTA WHOLE BLOOD

HEMOGLOBIN (HB) METHOD : CYANIDE FREE DETERMINATION	**7.6 Low**	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : FLUORESCENCE FLOW CYTOMETRY	4.25	3.8 - 4.8	mil/µL
WHITE BLOOD CELL (WBC) COUNT METHOD : ELECTRICAL IMPEDANCE	7.18	4.0 - 10.0	thou/µL
PLATELET COUNT METHOD : ELECTRONIC IMPEDENCE & MICROSCOPY	**478 High**	150 - 410	thou/µL

RBC AND PLATELET INDICES

HEMATOCRIT (PCV) METHOD : CALCULATED PARAMETER	**26.4 Low**	36 - 46	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM	**62.2 Low**	83.0 - 101.0	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER	**17.9 Low**	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER	**28.8 Low**	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM	**17.3 High**	11.6 - 14.0	%
MENTZER INDEX	14.6		
MEAN PLATELET VOLUME (MPV) METHOD : DERIVED PARAMETER FROM PLATELET HISTOGRAM	9.8	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS METHOD : FLUORESCENCE FLOW CYTOMETRY	71	40 - 80	%
LYMPHOCYTES METHOD : FLUORESCENCE FLOW CYTOMETRY	21	20 - 40	%
MONOCYTES	6	2 - 10	%

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METHOD : FLUORESCENCE FLOW CYTOMETRY			
EOSINOPHILS	2	1 - 6	%
METHOD : FLUORESCENCE FLOW CYTOMETRY			
BASOPHILS	0	0 - 1	%
METHOD : FLUORESCENCE FLOW CYTOMETRY			
ABSOLUTE NEUTROPHIL COUNT	5.10	2.0 - 7.0	thou/µL
METHOD : CALCULATED PARAMETER			
ABSOLUTE LYMPHOCYTE COUNT	1.51	1.0 - 3.0	thou/µL
METHOD : CALCULATED PARAMETER			
ABSOLUTE MONOCYTE COUNT	0.43	0.2 - 1.0	thou/µL
METHOD : CALCULATED PARAMETER			
ABSOLUTE EOSINOPHIL COUNT	0.14	0.02 - 0.50	thou/µL
METHOD : CALCULATED PARAMETER			
ABSOLUTE BASOPHIL COUNT	**0.00 Low**	0.02 - 0.10	thou/µL
METHOD : CALCULATED PARAMETER			
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	3.4		
METHOD : CALCULATED			

MORPHOLOGY

RBC	MILD ANISOPOIKILOCYTOSIS. MICROCYTIC HYPOCHROMIC WITH ELLIPTOCYTES AND OVALOCYTES.
WBC	NORMAL MORPHOLOGY
PLATELETS	ADEQUATE

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.

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HAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD

E.S.R	8	= or < 12	mm at 1 hr

METHOD : MODIFIED WESTERGREN METHOD BY AUTOMATED ANALYSER

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C	5.7	Non-diabetic Adult < 5.7 Pre-diabetes 5.7 - 6.4 Diabetes diagnosis: > or = 6.5 Therapeutic goals: < 7.0 Action suggested : > 8.0 (ADA Guideline 2021)	%

METHOD : ION- EXCHANGE HPLC

ESTIMATED AVERAGE GLUCOSE(EAG)	**116.9 High**	< 116	mg/dL

Interpretation(s)
ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA 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**:

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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 :

1. 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.
2.Vitamin C & E are reported to falsely lower test results.(possibly by inhibiting glycation of hemoglobin.
3. 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.
4. 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

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IMMUNOHAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP	O		
METHOD : HAEMAGGLUTINATION (AUTOMATED)			
RH TYPE	POSITIVE		
METHOD : HAEMAGGLUTINATION (AUTOMATED)			

Interpretation(s)
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.

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BIOCHEMISTRY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

GLUCOSE FASTING,FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)	**105 High**	Normal <100 Impaired fasting glucose:100 to 125 Diabetes mellitus: > = 126 (on more than 1 occassion) (ADA guidelines 2021)	mg/dL

METHOD : SPECTROPHOTOMETRY HEXOKINASE

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)	126	Normal <140 Impaired glucose tolerance:140 to 199 Diabetes mellitus : > = 200 (on more than 1 occassion) ADA guideline 2021	mg/dL

METHOD : SPECTROPHOTOMETRY HEXOKINASE

LIPID PROFILE WITH CALCULATED LDL

CHOLESTEROL, TOTAL	157	Desirable : < 200 Borderline : 200 - 239 High : > / = 240	mg/dL

METHOD : SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC - CHOLETSEROL OXIDASE, ESTERASE, PEROXIDASE

TRIGLYCERIDES	88	Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL

METHOD : SPECTROPHOTOMETRY, ENZYMATIC ENDPOINT WITH GLYCEROL BLANK

HDL CHOLESTEROL	40	At Risk: < 40 Desirable: > or = 60	mg/dL

METHOD : SPECTROPHOTOMETRY, HOMOGENEOUS DIRECT ENZYMATIC COLORIMETRIC

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CIN - U74899PB1995PLC045956

Patient Ref. No. 2000012126647

MC-5718

PATIENT NAME : KRISHNAMUR VANDANA KODANDAPANI **REF. DOCTOR :** SELF

ACCESSION NO	**0002WL024265**	AGE/SEX	:41 Years Female
PATIENT ID	: KRISF1306822	DRAWN	:16/12/2023 09:00:04
CLIENT PATIENT ID:		RECEIVED	:16/12/2023 09:01:16
ABHA NO	:	REPORTED	:18/12/2023 17:02:19

Test Report Status **Final**	Results	Biological Reference Interval	Units
CHOLESTEROL LDL	99	Optimal : < 100 Near optimal/above optimal : 100-129 Borderline high : 130-159 High : 160-189 Very high : = 190	mg/dL
METHOD : CALCULATED PARAMETER			
NON HDL CHOLESTEROL	117	Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
METHOD : CALCULATED PARAMETER			
VERY LOW DENSITY LIPOPROTEIN	18.0	< or = 30.0	mg/dL
METHOD : CALCULATED PARAMETER			
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 PARAMETER			
LDL/HDL RATIO	2.5	Desirable/Low Risk : 0.5 - 3.0 Borderline/Moderate Risk : 3.1 - 6.0 High Risk : > 6.0	
METHOD : CALCULATED PARAMETER			

Interpretation(s)

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

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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)
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	0.24	Upto 1.2	mg/dL
METHOD : SPECTROPHOTOMETRY, COLORIMETRIC -DIAZO METHOD			
BILIRUBIN, DIRECT	0.09	< or = 0.3	mg/dL
METHOD : SPECTROPHOTOMETRY, JENDRASSIK & GROFF - DIAZOTIZATION			
BILIRUBIN, INDIRECT	0.15	0.0 - 0.9	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	7.3	6.0 - 8.0	g/dL
METHOD : SPECTROPHOTOMETRY, COLORIMETRIC -BIURET, REAGENT BLANK, SERUM BLANK			
ALBUMIN	4.6	3.97 - 4.94	g/dL
METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING			
GLOBULIN	2.7	2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	1.7	1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	12	Upto 32	U/L
METHOD : SPECTROPHOTOMETRY, WITHOUT PYRIDOXAL PHOSPHATE ACTIVATION(P5P) - IFCC			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	14	Upto 33	U/L
METHOD : SPECTROPHOTOMETRY, WITHOUT PYRIDOXAL PHOSPHATE ACTIVATION(P5P) - IFCC			
ALKALINE PHOSPHATASE	**114 High**	35 - 104	U/L
METHOD : SPECTROPHOTOMETRY, PNPP, AMP BUFFER - IFCC			
GAMMA GLUTAMYL TRANSFERASE (GGT)	11	< 40	U/L

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METHOD : SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC - G-GLUTAMYL-CARBOXY-NITROANILIDE - IFCC			
LACTATE DEHYDROGENASE	115	< 223	U/L
METHOD : SPECTROPHOTOMETRY, LACTATE TO PYRUVATE - UV-IFCC			
BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN	8	6 - 20	mg/dL
METHOD : SPECTROPHOTOMETRY, UREASE -COLORIMETRIC			
CREATININE, SERUM			
CREATININE	0.60	0.60 - 1.10	mg/dL
METHOD : SPECTROPHOTOMETRY, JAFFE'S ALKALINE PICRATE KINETIC - RATE BLANKED - IFCC-IDMS STANDARIZED			
BUN/CREAT RATIO			
BUN/CREAT RATIO	14.00	8 - 15	
METHOD : CALCULATED PARAMETER			
URIC ACID, SERUM			
URIC ACID	3.3	2.4 - 5.7	mg/dL
METHOD : SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC- URICASE			
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN	7.3	6.0 - 8.0	g/dL
METHOD : SPECTROPHOTOMETRY, COLORIMETRIC -BIURET, REAGENT BLANK, SERUM BLANK			
ALBUMIN, SERUM			
ALBUMIN	4.6	3.97 - 4.94	g/dL
METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING			

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GLOBULIN			
GLOBULIN METHOD : CALCULATED PARAMETER	2.7	2.0 - 3.5	g/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM, SERUM METHOD : ISE INDIRECT	140	136 - 145	mmol/L
POTASSIUM, SERUM METHOD : ISE INDIRECT	4.20	3.5 - 5.1	mmol/L
CHLORIDE, SERUM METHOD : ISE INDIRECT	**107 High**	98 - 106	mmol/L

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.

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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)

Interpretation(s)
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: 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-
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, Pagets disease,Rickets,Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia,Malnutrition,Protein deficiency,Wilsons 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.
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,Waldenstroms 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 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, Muscuophy
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-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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Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma,Waldenstroms 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.

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CLINICAL PATH - URINALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

PHYSICAL EXAMINATION, URINE

COLOR	PALE YELLOW		
APPEARANCE	CLEAR		

CHEMICAL EXAMINATION, URINE

PH	6.0	5.00 - 7.50	
SPECIFIC GRAVITY	1.025	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

RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S)	2-3	0-5	/HPF
EPITHELIAL CELLS	1-2	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

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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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CIN - U74899PB1995PLC045956

Patient Ref. No. 2000012126647

MC-5718

PATIENT NAME : KRISHNAMUR VANDANA KODANDAPANI **REF. DOCTOR :** SELF

ACCESSION NO	: **0002WL024265**	AGE/SEX	:41 Years Female
PATIENT ID	: KRISF1306822	DRAWN	:16/12/2023 09:00:04
CLIENT PATIENT ID:		RECEIVED	: 16/12/2023 09:01:16
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Test Report Status **Final**	Results	Biological Reference Interval	Units

CYTOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

PAPANICOLAOU SMEAR

TEST METHOD	CONVENTIONAL GYNEC CYTOLOGY
SPECIMEN TYPE	ONE UNSTAINED CERVICAL SMEAR RECEIVED. (2CW-32958)
REPORTING SYSTEM	2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY
SPECIMEN ADEQUACY	SMEAR IS SATISFACTORY FOR EVALUATION.
MICROSCOPY	THE SMEAR SHOWS MAINLY SUPERFICIAL SQUAMOUS CELLS, FEW INTERMEDIATE SQUAMOUS CELLS, OCCASIONAL CLUSTERS OF ENDOCERVICAL 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.

Dr. Swathi Vadlamudi, MD
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Histopathologist

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CLINICAL PATH - STOOL ANALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

MICROSCOPIC EXAMINATION,STOOL

REMARK SAMPLE NOT RECEIVED

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 detects presence 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.
pH	Normal stool pH is slightly acidic to neutral. Breast-fed babies generally have an acidic stool.

ADDITIONAL STOOL TESTS :

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Senior Microbiologist

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1. **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.
4. **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. **Biofire (Film Array) GI PANEL:** 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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SPECIALISED CHEMISTRY - HORMONE

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

THYROID PANEL, SERUM

T3	102.0	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 ELECTROCHEMILUMINESCENCE IMMUNOASSAY

T4	7.64	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 : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY

TSH (ULTRASENSITIVE)	3.780	NonPregnant Women 0.27-4.20 Pregnant Women (As per American Thyroid Association) 1st Trimester 0.100 - 2.500 2nd Trimester 0.200 - 3.000 3rd Trimester 0.300 - 3.000	µIU/mL

METHOD : SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY

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

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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.

TSH in pregnancy

There's reduction in both the lower and the upper limit of maternal TSH relative to the non-pregnant TSH reference range. This is because of elevated levels of serum hCG that directly stimulates the TSH receptor, thereby increasing thyroid hormone production. The largest decrease in serum TSH is observed during the first trimester. Thereafter, serum TSH and its reference range gradually increases in the second and third trimesters, but nonetheless remains lower than in non-pregnant women.

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.

****End Of Report****

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DIAGNOSTIC REPORT

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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 AGILUS 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. AGILUS Diagnostics 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.
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