

Dr. Vinay Chopra
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 Chairman & Consultant Pathologist

Dr. Yugam Chopra
 MD (Pathology)
 CEO & Consultant Pathologist

NAME	: Mrs. RACHANA	PATIENT ID	: 1669342
AGE/ GENDER	: 43 YRS/FEMALE	REG. NO./LAB NO.	: 012411120018
COLLECTED BY	: SURJESH	REGISTRATION DATE	: 12/Nov/2024 10:08 AM
REFERRED BY	:	COLLECTION DATE	: 12/Nov/2024 11:02AM
BARCODE NO.	: 01520637	REPORTING DATE	: 12/Nov/2024 12:54PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		


Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIPID PROFILE : BASIC

CHOLESTEROL TOTAL: SERUM <i>by CHOLESTEROL OXIDASE PAP</i>	278.64^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM <i>by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)</i>	174.36^H	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM <i>by SELECTIVE INHIBITION</i>	46.79	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	196.98^H	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	231.85^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	34.87	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	731.64^H	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	5.96^H	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0




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LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	4.21^H	RATIO	MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	3.73	RATIO	3.00 - 5.00

INTERPRETATION:

- Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
- As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
- Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
- NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL.
- Additional testing for Apolipoprotein B, hsCRP, Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement



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URIC ACID

URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	3.57	mg/dL	2.50 - 6.80
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INTERPRETATION:-

1. GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.
2. Uric Acid is the end product of purine metabolism . Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

1. Idiopathic primary gout.
2. Excessive dietary purines (organ meats, legumes, anchovies, etc).
3. Cytolytic treatment of malignancies especially leukemias & lymphomas.
4. Polycythemia vera & myeloid metaplasia.
5. Psoriasis.
6. Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCRETION (BY KIDNEYS)

1. Alcohol ingestion.
2. Thiazide diuretics.
3. Lactic acidosis.
4. Aspirin ingestion (less than 2 grams per day).
5. Diabetic ketoacidosis or starvation.
6. Renal failure due to any cause etc.

DECREASED:-


(A).DUE TO DIETARY DEFICIENCY


1. Dietary deficiency of Zinc, Iron and molybdenum.
2. Fanconi syndrome & Wilsons disease.
3. Multiple sclerosis .
4. Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCRETION

1. Drugs:- Probenecid , sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosteroids and ACTH, anti-coagulants and estrogens etc.




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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM <i>by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)</i>	0.815	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM <i>by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)</i>	5.47	µg/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM <i>by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)</i>	2.212	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

TSH levels are subject to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50%. Hence time of the day has influence on the measured serum TSH concentrations. TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4) and triiodothyronine (T3). Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction (hyperthyroidism) of T4 and/or T3.

CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

LIMITATIONS:-

- T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG), and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
- Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin , salicylates).
- Serum T4 levels in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.
- TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothyroidism , pregnancy , phenytoin therapy.

TRIIODOTHYRONINE (T3)		THYROXINE (T4)		THYROID STIMULATING HORMONE (TSH)	
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range (µIU/mL)
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 – 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 – 17.04	3 Days – 6 Months	0.70 - 8.40
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 – 16.16	6 – 12 Months	0.70 - 7.00



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1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80
11 - 19 Years	0.35 - 1.93	11 - 19 Years	4.87 - 13.20
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (μ IU/mL)			
	1st Trimester		0.10 - 2.50
	2nd Trimester		0.20 - 3.00
	3rd Trimester		0.30 - 4.10

INCREASED TSH LEVELS:

- 1.Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.
- 2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
- 3.Hashimotos thyroiditis
- 4.DRUGS: Amphetamines, iodine containing agents & dopamine antagonist.
- 5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

DECREASED TSH LEVELS:

- 1.Toxic multi-nodular goiter & Thyroiditis.
- 2.Over replacement of thyroid hormone in treatment of hypothyroidism.
- 3.Autonomously functioning Thyroid adenoma
- 4.Secondary pituitary or hypothalamic hypothyroidism
- 5.Acute psychiatric illness
- 6.Severe dehydration.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
- 8.Pregnancy: 1st and 2nd Trimester



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IMMUNOPATHOLOGY/SEROLOGY

RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM

RHEUMATOID (RA) FACTOR QUANTITATIVE: SERUM by NEPHLOMETRY	4.1	IU/mL	NEGATIVE: < 18.0 BORDERLINE: 18.0 - 25.0 POSITIVE: > 25.0
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INTERPRETATION:-

RHEUMATOID FACTOR (RA):

- Rheumatoid factors (RF) are antibodies that are directed against the Fc fragment of IgG altered in its tertiary structure.
- Over 75% of patients with rheumatoid arthritis (RA) have an IgM antibody to IgG immunoglobulin. This autoantibody (RF) is diagnostically useful although it may not be etiologically related to RA.
- Inflammatory Markers such as ESR & C-Reactive protein (CRP) are normal in about 60 % of patients with positive RA.
- The titer of RF correlates poorly with disease activity, but those patients with high titers tend to have more severe disease course.
- The test is useful for diagnosis and prognosis of rheumatoid arthritis.

RHEUMATOID ARTHRITIS:

- Rheumatoid Arthritis is a systemic autoimmune disease that is multi-functional in origin and is characterized by chronic inflammation of the membrane lining (synovium) joints which leads to progressive joint destruction and in most cases to disability and reduction of quality life.
- The disease spreads from small to large joints, with greatest damage in early phase.
- The diagnosis of RA is primarily based on clinical, radiological & immunological features. The most frequent serological test is the measurement of RA factor.

CAUTION (FALSE POSTIVE):-

- RA factor is not specific for Rheumatoid arthritis, as it is often present in healthy individuals with other autoimmune diseases and chronic infections.
- Non rheumatoid and rheumatoid arthritis (RA) populations are not clearly separate with regard to the presence of rheumatoid factor (RF) (15% of RA patients have a nonreactive titer and 8% of nonrheumatoid patients have a positive titer).
- Patients with various nonrheumatoid diseases, characterized by chronic inflammation may have positive tests for RF. These diseases include systemic lupus erythematosus, polymyositis, tuberculosis, syphilis, viral hepatitis, infectious mononucleosis, and influenza.
- Anti-CCP have been discovered in joints of patients with RA, but not in other form of joint disease. Anti-CCP2 is HIGHLY SENSITIVE (71%) & more specific (98%) than RA factor.
- Upto 30 % of patients with Seronegative Rheumatoid arthritis also show Anti-CCP antibodies.
- The positive predictive value of Anti-CCP antibodies for Rheumatoid Arthritis is far greater than Rheumatoid factor.

*** End Of Report ***



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