



| | | y & Microbiology) Consultant Pathologist | | (Pathology) |
|----------------|-------------------------|---|--------------------------|--------------------------------------|
| IAME | : Mrs. SHALINI | | | |
| AGE/ GENDER | : 30 YRS/FEMALE | | PATIENT ID | : 1365736 |
| COLLECTED BY | : | | REG. NO./LAB NO. | : 012411160014 |
| REFERRED BY | : | | REGISTRATION DATE | : 16/Nov/2024 09:12 AM |
| BARCODE NO. | : 01520890 | | COLLECTION DATE | : 16/Nov/2024 09:18AM |
| CLIENT CODE. | : KOS DIAGNOSTIC LAB | : | REPORTING DATE | : 16/Nov/2024 10:37AM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROA | D, AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | CLIN | ICAL CHEMIST | FRY/BIOCHEMIST | 'RY |
| | | CLUCOCE | FASTING (F) | |
| | | GLUCUSE | rasting (r) | |

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

A fasting plasma glucose level below 100 mg/dl is considered normal.
 A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood

test (after consumption of 75 gms of glucose) is recommended for all such patients. 3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





| | Dr. Vinay C MD (Pathology Chairman & Co | | Dr. Yugam MD CEO & Consultant | (Pathology) |
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| CLIENT CODE. | : KOS DIAGNOSTIC LAB | REF | PORTING DATE | : 16/Nov/2024 10:54AM |
| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD | , AMBALA CANTT | | |
| Test Name | | Value | Unit | Biological Reference interval |
| | | LIPID PROFII | F.BASIC | |
| CHOLESTEROL TO | TAL SEDUM | 171.85 | | OPTIMAL: < 200.0 |
| by CHOLESTEROL OX | | 171.85 | mg/dL | BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0 |
| TRIGLYCERIDES: S by GLYCEROL PHOSP | ERUM PHATE OXIDASE (ENZYMATIC) | 103.9 | mg/dL | OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0 |
| HDL CHOLESTERO | L (DIRECT): SERUM 70N | 43.91 | mg/dL | LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0 |
| LDL CHOLESTEROI by CALCULATED, SPE | | 107.16 | mg/dL | OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 |
| NON HDL CHOLEST by Calculated, spe | | 127.94 | mg/dL | VERY HIGH: > OR = 190.0 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 CHOLESTERC | | 20.78 | mg/dL | 0.00 - 45.00 |
| TOTAL LIPIDS: SER by CALCULATED, SPE | RUM | 447.6 | mg/dL | 350.00 - 700.00 |
| CHOLESTEROL/HD by CALCULATED, SPE | DL RATIO: SERUM | 3.91 | RATIO | LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0 |
| | | | | |



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| Test Name | | Value | Unit | Biological Reference interval |
| LDL/HDL RATIO: S by CALCULATED, SPE | | 2.44 | RATIO | LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0 |
| TRIGLYCERIDES/H by CALCULATED, SPE | IDL RATIO: SERUM | 2.37 ^L | RATIO | 3.00 - 5.00 |

INTERPRETATION:

1. 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.

2. 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 atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, 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

5. 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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| Test Name | | Value | Unit | Biological Reference i | nterval |
| | | URIC A | CID | | |
| URIC ACID: SERUM | | 5.95 | mg/dL | 2.50 - 6.80 | |
| by URICASE - OXIDASE | PEROXIDASE | 0.00 | ing, all | 2.00 0.00 | |
| INTERPRETATION:- | high levels of Uric Acid in the bloc | ad causa crystals to | form & accumulate ar | ound a joint | |
| 2 Uric Acid is the end r | product of purine metabolism | ric acid is excreted t | to a large degree by the | kidneys and to a smaller degree in the | he |
| intestinal tract by mici | robial degradation. | | to a large degree by the | Refleys and to a smaller degree in th | |
| NCREASED:- | | | | | |
| (A).DUE TO INCREASED | | | | | |
| 1. Idiopathic primary g | out. | | | | |
| 2.Excessive dietary pur | ines (organ meats,legumes,anch | ovies, etc). | | | |
| | of malignancies especially leuker | nais & lymphomas. | | | |
| 4.Polycythemai vera & 5.Psoriasis. | myelold metaplasia. | | | | |
| 6.Sickle cell anaemia e | tc | | | | |
| | EXCREATION (BY KIDNEYS) | | | | |
| 1.Alcohol ingestion. | , | | | | |
| 2.Thiazide diuretics. | | | | | |
| 3.Lactic acidosis. | | | | | |
| | ss than 2 grams per day). | | | | |
| 5.Diabetic ketoacidosi | | | | | |
| 6.Renal failure due to a | any cause etc. | | | | |
| decreased:- (a).due to dietary de | FICIENCY | | | | |
| | Zinc, Iron and molybdenum. | | | | |
| 2.Fanconi syndrome & | Wilsons disease | | | | |
| | | | | | |
| S.IVIUITIPIE SCIELOSIS . | priate antidiuretic hormone (SIA | DH) secretion & low | purine diet etc. | | |
| Multiple sclerosis . Syndrome of inappro | priate antialarette normone (Siv | | | | |
| 4.Syndrome of inappro (B).DUE TO INCREASED | EXCREATION | | | | |
| 4.Syndrome of inappro (B).DUE TO INCREASED | EXCREATION | more than 4 grams | per day), corticosterroi | ds and ACTH, anti-coagulants and est | rogens e |





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| CLIENT ADDRESS | : 6349/1, NICHOLSON ROAD, AM | BALA CANTI | 7 | | | |
| Test Name | | Value | Unit | Biological Reference interva | | |
| | | ENDOC | RINOLOGY | | | |
| | THYR | ROID FUNG | CTION TEST: TOTAL | | | |
| TRIIODOTHYRONI | | 1.157 | ng/mL | 0.35 - 1.93 | | |
| by CMIA (CHEMILUMIN THYROXINE (T4): S | ESCENT MICROPARTICLE IMMUNOASSA | y) 11.36 | μgm/d | L 4.87 - 12.60 | | |
| | IESCENT MICROPARTICLE IMMUNOASSA | | μgiii/u | L 4.07 - 12.00 | | |
| | TING HORMONE (TSH): SERUM | 1.892 | µIU/m | L 0.35 - 5.50 | | |
| by CMIA (CHEMILUMIN 3rd GENERATION, ULT | IESCENT MICROPARTICLE IMMUNOASSA RASENSITIVE | Y) | | | | |
| INTERPRETATION: | | | | | | |
| day has influence on the trilodothyronine (T3).Fai | | imulates the pr | oduction and secretion of the | pm. The variation is of the order of 50%.Hence time of metabolically active hormones, thyroxine (T4)and ther underproduction (hypothyroidism) or | | |
| CLINICAL CONDITION | Т3 | | T4 | TSH | | |
| Primary Hypothyroidis | | | Reduced | Increased (Significantly) | | |
| Subclinical Hypothyroi | dism: Normal or Low Nor | mal | Normal or Low Normal | High | | |

| LIM | ΙΤΑΤ | IONS:- | |
|-----|------|--------|--|

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

1. 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.

Increased

Normal or High Normal

Reduced (at times undetectable)

Reduced

2. 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).

3. 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.

4. TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothyroidism , pregnancy , phenytoin therapy.

| TRIIODOTH | DOOTHYRONINE (T3) THYROXINE (T4) | | INE (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 | |

Increased

Normal or High Normal





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| Test Name | | | Value Unit | t | Biological Reference interva | |
|---------------------|---------------|----------------------|------------------|---------------------|------------------------------|--|
| 1 - 10 Years | 0.92 - 2.28 | 1 - 10 Years | 6.00 - 13.80 | 1 – 10 Years | 0.60 - 5.50 | |
| 11- 19 Years | 0.35 - 1.93 | 11 - 19 Years | 4.87- 13.20 | 11 – 19 Years | 0.50 - 5.50 | |
| > 20 years (Adults) | 0.35 - 1.93 | > 20 Years (Adults) | 4.87 - 12.60 | > 20 Years (Adults) | 0.35-5.50 | |
| | RECO | MMENDATIONS OF TSH L | EVELS DURING PRE | GNANCY (µIU/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

*** End Of Report *





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