

(A Unit of KOS Healthcare)



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

NAME : Mrs. AMRIT KAUR

AGE/ GENDER : 70 YRS/FEMALE PATIENT ID : 1656207

COLLECTED BY: SURJESH REG. NO./LAB NO. : 012410290015

 REFERRED BY
 : 29/Oct/2024 09:45 AM

 BARCODE NO.
 : 01519742
 COLLECTION DATE
 : 29/Oct/2024 09:48AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 29/Oct/2024 10:15AM

CLIENT ADDRESS: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

| HAEMOGLOBIN (HB) by CALORIMETRIC | 11.3 ^L | gm/dL | 12.0 - 16.0 |
|---|-------------------|--------------|--|
| RED BLOOD CELL (RBC) COUNT by Hydro dynamic focusing, electrical impedence | 4.24 | Millions/cmm | 3.50 - 5.00 |
| PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 36.9 ^L | % | 37.0 - 50.0 |
| MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 87.1 | fL | 80.0 - 100.0 |
| MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 26.6 ^L | pg | 27.0 - 34.0 |
| MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 30.5 ^L | g/dL | 32.0 - 36.0 |
| RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 12.8 | % | 11.00 - 16.00 |
| RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 41.6 | fL | 35.0 - 56.0 |
| MENTZERS INDEX by CALCULATED | 20.54 | RATIO | BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0 |
| GREEN & KING INDEX by CALCULATED | 26.24 | RATIO | BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0 |
| WHITE BLOOD CELLS (WBCS) | | | |
| TOTAL LEUCOCYTE COUNT (TLC) by flow cytometry by sf cube & microscopy | 6360 | /cmm | 4000 - 11000 |
| NUCLEATED RED BLOOD CELLS (nRBCS) by automated 6 part hematology analyzer | NIL | | 0.00 - 20.00 |
| NUCLEATED RED BLOOD CELLS (nRBCS) % | NIL | % | < 10 % |



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by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER



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| DIFFERENTIAL LEUCOCYTE COUNT (DLC) | | | |
| NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 61 | % | 50 - 70 |
| LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 27 | % | 20 - 40 |
| EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 5 | % | 1 - 6 |
| MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 7 | % | 2 - 12 |
| BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 0 | % | 0 - 1 |
| ABSOLUTE LEUKOCYTES (WBC) COUNT | | | |
| ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 3880 | /cmm | 2000 - 7500 |
| ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 1717 | /cmm | 800 - 4900 |
| ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 318 | /cmm | 40 - 440 |
| ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 445 | /cmm | 80 - 880 |
| PLATELETS AND OTHER PLATELET PREDICTIVE | MARKERS. | | |
| PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 307000 | /cmm | 150000 - 450000 |
| PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 0.35 | % | 0.10 - 0.36 |
| MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 11 | fL | 6.50 - 12.0 |
| PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 104000 ^H | /cmm | 30000 - 90000 |
| PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 34 | % | 11.0 - 45.0 |
| PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD | 15.9 | % | 15.0 - 17.0 |



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Test Name Value Unit Biological Reference interval

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM 0.821 ng/mL 0.35 - 1.93

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROXINE (T4): SERUM 5.18 $\mu gm/dL$ 4.87 - 12.60

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROID STIMULATING HORMONE (TSH): SERUM 3.808 $\mu\text{IU/mL}$ 0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

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 | Т3 | 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:-

- 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, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
- 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.

| 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 | 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 | |
| | RECOM | MENDATIONS OF TSH LI | EVELS DURING PREC | 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



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Value Unit **Biological Reference interval Test Name**

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM ng/mL DEFICIENCY: < 20.0 13.6^{L}

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0

TOXICITY: > 100.0

INTERPRETATION:

| DEFICIENT: | < 20 | ng/mL |
|------------------|----------|-------|
| INSUFFICIENT: | 21 - 29 | ng/mL |
| PREFFERED RANGE: | 30 - 100 | ng/mL |
| INTOXICATION: | > 100 | ng/mL |

- 1. Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.

 2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose
- tissue and tightly bound by a transport protein while in circulation.
- 3. Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).
- 4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults. DECREASED:
- 1.Lack of sunshine exposure.
- 2.Inadequate intake, malabsorption (celiac disease)
- 3. Depressed Hepatic Vitamin D 25- hydroxylase activity
- 4. Secondary to advanced Liver disease
- 5. Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- 6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism. INCREASED:
- 1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.



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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM **1125^H** pg/mL 190.0 - 890.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:-

| INCREASED VITAMIN B12 | DECREASED VITAMIN B12 |
|-------------------------------|---|
| 1.Ingestion of Vitamin C | 1.Pregnancy |
| 2.Ingestion of Estrogen | 2.DRUGS:Aspirin, Anti-convulsants, Colchicine |
| 3.Ingestion of Vitamin A | 3.Ethanol Igestion |
| 4.Hepatocellular injury | 4. Contraceptive Harmones |
| 5.Myeloproliferative disorder | 5.Haemodialysis |
| 6. Multiple Myeloma | |

- 1. Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
- 2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
- 3. The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
- 4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg. gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
- 5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
- 6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
- 7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.

 NOTE:A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

*** End Of Report ***



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