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<b>NAME</b>	: Mr. SUKHWINDER	<b>PATIENT ID</b>	: 1538023
<b>AGE/ GENDER</b>	: 45 YRS/MALE	<b>REG. NO./LAB NO.</b>	: <b>012407040004</b>
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 04/Jul/2024 07:38 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 04/Jul/2024 07:46AM
<b>BARCODE NO.</b>	: 01512479	<b>REPORTING DATE</b>	: 04/Jul/2024 08:57AM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
<b>CLIENT ADDRESS</b>	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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**HAEMATOLOGY**

**BLOOD GROUP (ABO) AND RH FACTOR TYPING**

ABO GROUP  
*by SLIDE AGGLUTINATION*

RH FACTOR TYPE  
*by SLIDE AGGLUTINATION*

B  
  
POSITIVE



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**ENDOCRINOLOGY**

**THYROID STIMULATING HORMONE (TSH)**

**THYROID STIMULATING HORMONE (TSH): SERUM**      9.939<sup>H</sup>      μIU/mL      0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

**INTERPRETATION:**

AGE	REFERENCE RANGE (μIU/mL)
0 – 5 DAYS	0.70 – 15.20
6 Days – 2 Months	0.70 – 11.00
3 – 11 Months	0.70 – 8.40
1 – 5 Years	0.70 – 7.00
6 – 10 Years	0.60 – 5.50
11 - 15	0.50 – 5.50
> 20 Years (Adults)	0.27 – 5.50
PREGNANCY	
1st Trimester	0.10 - 3.00
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

**NOTE:- TSH levels are subjected 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 concentration.**

**USE:-** TSH controls biosynthesis and release of thyroid hormones T4 & T3. It is a sensitive measure of thyroid function, especially useful in early or subclinical hypothyroidism, before the patient develops any clinical findings or goitre or any other thyroid function abnormality.


**INCREASED LEVELS:**


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

**DECREASED LEVELS:**

- 1.Toxic multi-nodular goitre & 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.



  
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7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.


8.Pregnancy: 1st and 2nd Trimester

**LIMITATIONS:**

- 1.TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.
- 2.Autoimmune disorders may produce spurious results.

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**IMMUNOPATHOLOGY/SEROLOGY**

**RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM**

RHEUMATOID (RA) FACTOR QUANTITATIVE: SERUM <i>by NEPHLOMETRY</i>	7.32	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.



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**VITAMINS**

**VITAMIN D/25 HYDROXY VITAMIN D3**

<b>VITAMIN D (25-HYDROXY VITAMIN D3): SERUM</b> <i>by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)</i>	28.28 <sup>L</sup>	ng/mL	<b>DEFICIENCY:</b> < 20.0 <b>INSUFFICIENCY:</b> 20.0 - 30.0 <b>SUFFICIENCY:</b> 30.0 - 100.0 <b>TOXICITY:</b> > 100.0
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**INTERPRETATION:**

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

- 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.
- 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.
- 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 hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

**DECREASED:**

- Lack of sunshine exposure.
- Inadequate intake, malabsorption (celiac disease)
- Depressed Hepatic Vitamin D 25- hydroxylase activity
- Secondary to advanced Liver disease
- Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

**INCREASED:**

- 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 hyperphosphatemia.

**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 interfere with Vitamin D absorption.



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**VITAMIN B12/COBALAMIN**

VITAMIN B12/COBALAMIN: SERUM	208.52	pg/mL	190.0 - 830
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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 lgestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

- Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
- In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
- 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.
- 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).
- 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.
- Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
- 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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