



# P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

**A PIONEER DIAGNOSTIC CENTRE**

☎ 0171-2532620, 8222896961 ✉ pkrjainhealthcare@gmail.com

<b>NAME</b>	: Miss. BHUMI	<b>PATIENT ID</b>	: 1810662
<b>AGE/ GENDER</b>	: 18 YRS/FEMALE	<b>REG. NO./LAB NO.</b>	: 122503290011
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 29/Mar/2025 10:08 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 29/Mar/2025 10:26AM
<b>BARCODE NO.</b>	: 12507783	<b>REPORTING DATE</b>	: 29/Mar/2025 05:08PM
<b>CLIENT CODE.</b>	: P.K.R JAIN HEALTHCARE INSTITUTE		
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Test Name	Value	Unit	Biological Reference interval
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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### FERRITIN

FERRITIN: SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	4.75 <sup>L</sup>	ng/mL	13.0 - 147.0
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#### INTERPRETATION:

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy.

#### DECREASED:

1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.
2. Hypothyroidism.
3. Vitamin-C deficiency.

#### INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

1. Hemochromatosis or hemosiderosis.
2. Wilson Disease.

#### INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

1. Transfusion overload
2. Excess dietary Iron
3. Porphyria Cutanea tarda
4. Ineffective erythropoiesis.


#### INCREASED FERRITIN WITHOUT IRON OVERLOAD:


1. Liver disorders (NASH) or viral hepatitis (B/C).
2. Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
3. Leukaemia, hodgekin's disease.
4. Alcohol excess.
5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma, synthesis of ferritin by tumour cells.
6. Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

#### NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can therefore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive proteins to rule out any inflammatory conditions.
2. Patients with iron deficiency anaemia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.



  
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## ENDOCRINOLOGY

### THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 1.707  $\mu$ IU/mL 0.50 - 5.50  
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

#### INTERPRETATION:

AGE	REFERENCE RANGE ( $\mu$ IU/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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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM  $\gamma$ L ng/mL  
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

DEFICIENCY: < 20.0  
INSUFFICIENCY: 20.0 - 30.0  
SUFFICIENCY: 30.0 - 100.0  
TOXICITY: > 100.0

#### INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFERRED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 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 reservoir 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 homeostasis. 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 Hyperparathyroidism (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  
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

161<sup>L</sup> pg/mL 190.0 - 890.0

### 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 ingestion
4. Hepatocellular injury	4. Contraceptive Hormones
5. Myeloproliferative disorder	5. Haemodialysis
6. Uremia	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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