



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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

NAME	: Mr. REWAT	PATIENT ID	: 1538076
AGE/ GENDER	: 37 YRS/MALE	REG. NO./LAB NO.	: 122407040006
COLLECTED BY	:	REGISTRATION DATE	: 04/Jul/2024 10:31 AM
REFERRED BY	:	COLLECTION DATE	: 04/Jul/2024 10:40AM
BARCODE NO.	: 12503430	REPORTING DATE	: 04/Jul/2024 04:56PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE		
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

FERRITIN

FERRITIN: SERUM	63.02	ng/mL	10.0 - 290.0
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by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

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 FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.032	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	5.32	µgm/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	2.305	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRA SENSITIVE

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 (eg: 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




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6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 - 12 Months	0.70 - 7.00
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
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (μ U/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, idonie 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 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.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
- 8.Pregnancy: 1st and 2nd Trimester




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CORTISOL: TOTAL

CORTISOL TOTAL	107.6	ng/mL	52.0 - 350.0
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by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:

1.A cortisol test is done to measure the level of the hormone cortisol in the blood. The cortisol level may show problems with the adrenal glands or pituitary gland. Cortisol is made by the adrenal glands .
2.Cortisol levels go up when the pituitary gland releases another hormone called adrenocorticotrophic hormone (ACTH).
3.Most cortisol in the blood is bound to a protein; only a small percentage is "free" and biologically active. Blood cortisol testing evaluates both protein-bound and free cortisol while urine and saliva testing evaluate only free cortisol, which should correlate with the levels of free cortisol in the blood. Multiple blood and/or saliva cortisol levels collected at different times, such as at 8 am and 4 pm, can be used to evaluate both cortisol levels and diurnal variation. A 24-hour urine cortisol sample will not show diurnal variation; it will measure the total amount of unbound cortisol excreted in 24 hours.

CORTISOL FUNCTIONS:


- 1.It helps the body use sugar (glucose) and fat for energy (metabolism), and it helps the body manage stress.
- 2.Bone growth
- 3.Blood pressure control
- 4.Immune system function
- 5.Metabolism of fats, carbohydrates, and protein
- 6.Nervous system function
- 7.Stress response

THINGS TO KNOW ABOUT CORTISOL MEASUREMENT:

- 1.An increased or normal cortisol level just after waking along with a level that does not drop by bedtime suggests excess cortisol and Cushing syndrome. If this excess cortisol is not suppressed after an overnight dexamethasone suppression test, or if the 24-hour urine cortisol is elevated, or if the late-night salivary cortisol level is elevated, it suggests that the excess cortisol is due to abnormal increased ACTH production by the pituitary or a tumor outside of the pituitary or abnormal production by the adrenal glands. Additional testing will help to determine the exact cause.
- 2.If insufficient cortisol is present and the person tested responds to an ACTH stimulation test, then the problem is likely due to insufficient ACTH production by the pituitary. If the person does not respond to the ACTH stimulation test, then it is more likely that the problem is based in the adrenal glands. If the adrenal glands are underactive, due to pituitary dysfunction and/or insufficient ACTH production, then the person is said to have secondary adrenal insufficiency. If decreased cortisol production is due to adrenal damage, then the person is said to have primary adrenal insufficiency or Addison disease.
- 3.Once an abnormality has been identified and associated with the pituitary gland, adrenal glands, or other cause, then the health practitioner may use other testing such as CT (computerized tomography) or MRI (magnetic resonance imaging) scans to locate the source of the excess (such as a pituitary, adrenal, or other tumor) and to evaluate the extent of any damage to the glands.
- 4.Similar to those with adrenal insufficiency, people with a condition called congenital adrenal hyperplasia (CAH) have low cortisol levels and




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do not respond to ACTH stimulation tests. Cortisol measurement is one of many tests that may be used to help evaluate a person for CAH.

5.Heat, cold, infection, trauma, exercise, obesity, and debilitating disease can influence cortisol concentrations. Pregnancy, physical and emotional stress, and illness can increase cortisol levels. Cortisol levels may also increase as a result of hyperthyroidism or obesity. A number of drugs can also increase levels, particularly oral contraceptives (birth control pills), hydrocortisone (the synthetic form of cortisol), and spironolactone.

6.Adults have slightly higher cortisol levels than children do.

7.Hypothyroidism may decrease cortisol levels. Drugs that may decrease levels include some steroid hormones.

8.Salivary cortisol testing is being used more frequently to help diagnose Cushing syndrome and stress-related disorders but still requires specialized expertise to perform.

NOTE:

1.Normally, cortisol levels rise during the early morning hours and are highest about 7 a.m. They drop very low in the evening and during the early phase of sleep. But if you sleep during the day and are up at night, this pattern may be reversed. If you do not have this daily change (diurnal rhythm) in cortisol levels, you may have overactive adrenal glands. This condition is called Cushing's syndrome.

2.The timing of the cortisol test is very important because of the way cortisol levels vary throughout a day. If your doctor thinks you might make too much cortisol, the test will probably be done late in the day. If your doctor thinks you may not be making enough, a test is usually done in the morning.




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TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM 6.25 ng/mL 0.47 - 9.80
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:

1. Testosterone is secreted in females by the ovary and formed indirectly from androstenedione in adrenal glands.
2. In males it is secreted by the testes. It circulates in blood bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form.
3. The bioavailable fraction includes the free form and that "weakly bound" to albumin (40% of the total in men and 20% of the total in women) and bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone.
4. The total testosterone bound to SHBG fluctuates since SHBG levels are affected by medication, disease, sex steroids and insulin.

CLINIC USE:

1. Assessment of testicular functions in males
2. Management of hirsutism and virilization in females


INCREASED LEVELS:


1. Precocious puberty (Males)
2. Androgen resistance
3. Testotoxicosis
4. Congenital Adrenal Hyperplasia
5. Polycystic ovarian disease
7. Ovarian tumors

DECREASED LEVELS:

1. Delayed puberty (Males)
2. Gonadotropin deficiency
3. Testicular defects
4. Systemic diseases




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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

28.71^L

ng/mL

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 201.9 pg/mL 200 - 940
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

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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