

Dr. Vinay Chopra
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 Chairman & Consultant Pathologist

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

NAME	: Miss. VAANI	PATIENT ID	: 1775660
AGE/ GENDER	: 25 YRS/FEMALE	REG. NO./LAB NO.	: 012503020015
COLLECTED BY	: SHYAM	REGISTRATION DATE	: 02/Mar/2025 09:26 AM
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	COLLECTION DATE	: 02/Mar/2025 09:47AM
BARCODE NO.	: 01526324	REPORTING DATE	: 02/Mar/2025 02:51PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

IRON PROFILE

IRON: SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	51.36	µg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	148.65^L	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM <i>by SPECTROPHOTOMETRY</i>	200.01^L	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY (FERENE)</i>	25.68	%	15.0 - 50.0
TRANSFERRIN: SERUM <i>by SPECTROPHOTOMETRY (FERENE)</i>	142.01^L	mg/dL	200.0 - 350.0

INTERPRETATION:-

VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:

1. Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia. i.e iron deficiency anemia, zinc deficiency anemia, anemia of chronic disease and thalassemia syndromes.

2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BINDING CAPACITY (TIBC):

1. It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:

1. Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.




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ENDOCRINOLOGY

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 2.322 μ IU/mL 0.35 - 5.50
 by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

AGE	REFERENCE RANGE (μ 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.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.




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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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FOLLICLE STIMULATING HORMONE (FSH)

FOLLICLE STIMULATING HORMONE (FSH): SERUM	4.48	mIU/mL	FEMALE FOLLICULAR PHASE: 3.03 - 8.08 FEMALE MID-CYCLE PEAK: 2.55 - 16.69 FEMALE LUTEAL PHASE: 1.38 - 5.47 FEMALE POST-MENOPAUSAL: 26.72 - 133.41 MALE: 0.95 - 11.95
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)			

INTERPRETATION:

1. Gonadotropin-releasing hormone from the hypothalamus controls the secretion of the gonadotropins, follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the anterior pituitary.
2. The menstrual cycle is divided by a midcycle surge of both FSH and LH into a follicular phase and a luteal phase.
3. FSH appears to control gametogenesis in both males and females.

The test is useful in the following settings:

1. An adjunct in the evaluation of menstrual irregularities.
2. Evaluating patients with suspected hypogonadism.
3. Predicting ovulation
4. Evaluating infertility
5. Diagnosing pituitary disorders
6. In both males and females, primary hypogonadism results in an elevation of basal follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels.

FSH and LH LEVELS ELEVATED IN:

1. Primary gonadal failure
2. Complete testicular feminization syndrome.
3. Precocious puberty (either idiopathic or secondary to a central nervous system lesion)
4. Menopause (postmenopausal FSH levels are generally >40 IU/L)
5. Primary ovarian hypofunction in females
6. Primary hypogonadism in males

NOTE:

1. Normal or decreased FSH is seen in polycystic ovarian disease in females
2. FSH and LH are both decreased in failure of the pituitary or hypothalamus.




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TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	0.29	ng/mL	0.0 - 0.80
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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

*** End Of Report ***




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