

Dr. Vinay Chopra
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 Chairman & Consultant Pathologist

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

NAME	: Mr. RAJDEEP SINGH	PATIENT ID	: 1327455
AGE/ GENDER	: 29 YRS/MALE	REG. NO./LAB NO.	: 042408140003
COLLECTED BY	:	REGISTRATION DATE	: 14/Aug/2024 09:25 AM
REFERRED BY	:	COLLECTION DATE	: 14/Aug/2024 03:17PM
BARCODE NO.	: A0465218	REPORTING DATE	: 14/Aug/2024 05:30PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.65	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.21	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.44	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	21.6	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	24.6	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.88	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	118	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	20.58	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.7	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.33	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.37	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.83	RATIO	1.00 - 2.00

INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Reference Range.
USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0




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INTRAHEPATIC CHOLESTATIS	> 1.5		
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)		

DECREASED:

- Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
- Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

PROGNOSTIC SIGNIFICANCE:

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6




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ENDOCRINOLOGY

ESTRADIOL (E2)

ESTRADIOL (E2): SERUM 43^H pg/mL 0.0 40.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTEPRETATION:

OTHER MATERNAL FACTORS AND PREGNANCY	UNITS	RANGE
Hormonal Contraceptives	pg/mL	15.0 – 95.0
1st Trimester (0 – 12 Weeks)	pg/mL	38.0 – 3175.0
2nd Trimester (13 – 28 Weeks)	pg/mL	678.0 – 16633.0
3rd Trimester (29 – 40 Weeks)	pg/mL	43.0 – 33781.0
Post Menopausal	Pg/mL	< 50.0
MALES:	pg/mL	< 40.0

1. Estrogens are involved in development and maintenance of the female phenotype, germ cell maturation, and pregnancy. They also are important for many other, nongender-specific processes, including growth, nervous system maturation, bone metabolism/remodeling, and endothelial responsiveness.

2. E2 is produced primarily in ovaries and testes by aromatization of testosterone.

3. Small amounts are produced in the adrenal glands and some peripheral tissues, most notably fat. E2 levels in premenopausal women fluctuate during the menstrual cycle.

4. They are lowest during the early follicular phase. E2 levels then rise gradually until 2 to 3 days before ovulation, at which stage they start to increase much more rapidly and peak just before the ovulation-inducing luteinizing hormone (LH)/follicle stimulating hormone (FSH) surge at 5 to 10 times the early follicular levels. This is followed by a modest decline during the ovulatory phase. E2 levels then increase again gradually until the midpoint of the luteal phase and thereafter decline to trough, early follicular levels.

INDICATIONS FOR ASSAY: -

1. Evaluation of hypogonadism and oligo-amenorrhea in females.
2. Assessing ovarian status, including follicle development, for assisted reproduction protocols (eg, in vitro fertilization)
3. In conjunction with lutenizing hormone measurements, monitoring of estrogen replacement therapy in hypogonadal premenopausal women
4. Evaluation of feminization, including gynecomastia, in males.
5. Diagnosis of estrogen-producing neoplasms in males, and, to a lesser degree, females
6. As part of the diagnosis and work-up of precocious and delayed puberty in females, and, to a lesser degree, males
7. As part of the diagnosis and work-up of suspected disorders of sex steroid metabolism, eg: aromatase deficiency and 17 alpha-hydroxylase deficiency
8. As an adjunct to clinical assessment, imaging studies and bone mineral density measurement in the fracture risk assessment of postmenopausal women, and, to a lesser degree, older men
9. Monitoring low-dose female hormone replacement therapy in post-menopausal women
10. Monitoring antiestrogen therapy (eg, aromatase inhibitor therapy).



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CAUSES FOR INCREASED E2 LEVELS:

1. High androgen levels caused by tumors or androgen therapy (medical or sport performance enhancing),with secondary elevations in E1 and E2 due to aromatization
2. Obesity with increased tissue production of E1
3. Decreased E1 and E2 clearance in liver disease
4. Estrogen producing tumors
5. Estrogen Ingestion



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TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	5.51	ng/mL	0.47 - 9.80
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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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