



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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

NAME : Mrs. NAMITA
AGE/ GENDER : 48 YRS/FEMALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12503410
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1536898
REG. NO./LAB NO. : 122407030001
REGISTRATION DATE : 03/Jul/2024 08:07 AM
COLLECTION DATE : 03/Jul/2024 08:10AM
REPORTING DATE : 03/Jul/2024 04:33PM

Test Name	Value	Unit	Biological Reference interval
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ENDOCRINOLOGY

LUTEINISING HORMONE (LH)

LUTEINISING HORMONE (LH): SERUM 2.61 mIU/mL
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

MALES: 1.0 -12.5
FOLLICULAR PHASE: 1.2 - 12.7
MID-CYCLE PEAK: 15.5 - 90.0
LUTEAL PHASE: 0.50 - 14.6
POST MENOPAUSAL: 15.6 - 72.0

INTERPRETATION:

1. Luteinizing hormone (LH) is a glycoprotein hormone consisting of 2 non covalently bound subunits (alpha and beta). Gonadotropin-releasing hormone from the hypothalamus controls the secretion of the gonadotropins, FSH and LH, from the anterior pituitary.
2. In both males and females, LH is essential for reproduction. In females, the menstrual cycle is divided by a mid cycle surge of both LH and FSH into a follicular phase and a luteal phase.
3. This "LH surge" triggers ovulation thereby not only releasing the egg, but also initiating the conversion of the residual follicle into a corpus luteum that, in turn, produces progesterone to prepare the endometrium for a possible implantation.
4. LH supports thecal cells in the ovary that provide androgens and hormonal precursors for estradiol production. LH in males acts on testicular interstitial cells of Leydig to cause increased synthesis of testosterone.

The test is useful in the following situations:

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

FSH AND LH 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
5. Primary ovarian hypo dysfunction in females
6. Polycystic ovary disease in females
7. Primary hypogonadism in males


LH IS DECREASED IN:


1. Primary ovarian hyper function in females
2. Primary hypergonadism in males

NOTE

1. FSH and LH are both decreased in failure of the pituitary or hypothalamus.




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FOLLICLE STIMULATING HORMONE (FSH)

FOLLICLE STIMULATING HORMONE (FSH): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	13.09	mIU/mL	FEMALE FOLLICULAR PHASE: 2.5 - 11.4 FEMALE MID-CYCLE PEAK: 3.3 - 21.7 FEAMLE LUTEAL PHASE: 1.2 - 7.0 FEMALE POST-MENOPAUSAL: 18.8 - 132 MALE: 1.0 - 12.1
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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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PROLACTIN

PROLACTIN: SERUM

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

32.7^H

ng/mL

3 - 25

INTERPRETATION:

1. Prolactin is secreted by the anterior pituitary gland and controlled by the hypothalamus.
2. The major chemical controlling prolactin secretion is dopamine, which inhibits prolactin secretion from the pituitary.
3. Physiological function of prolactin is the stimulation of milk production. In normal individuals, the prolactin level rises in response to physiologic stimuli such as sleep, exercise, nipple stimulation, sexual intercourse, hypoglycemia, postpartum period, and also is elevated in the newborn infant.

INCREASED (HYPERPROLACTEMIA):

1. Prolactin-secreting pituitary adenoma (prolactinoma, which is 5 times more frequent in females than males).
2. Functional and organic disease of the hypothalamus.
3. Primary hypothyroidism.
4. Section compression of the pituitary stalk.
5. Chest wall lesions and renal failure.
6. Ectopic tumors.
7. DRUGS:- Anti-Dopaminergic drugs like antipsychotic drugs, anti-nausea/antiemetic drugs, Drugs that affect CNS serotonin metabolism, serotonin receptors, or serotonin reuptake (anti-depressants of all classes, ergot derivatives, some illegal drugs such as cannabis), Antihypertensive drugs, Opiates, High doses of estrogen or progesterone, anticonvulsants (valproic acid), anti-tuberculous medications (Isoniazid).

SIGNIFICANCE:

1. In loss of libido, galactorrhea, oligomenorrhea, hyperprolactinemia often results in menorrhea or amenorrhea, and infertility in premenopausal females.
2. Loss of libido, impotence, infertility, and hypogonadism in males. Postmenopausal and premenopausal women, as well as men, can also suffer from decreased muscle mass and osteoporosis.
3. In males, prolactin levels >13 ng/mL are indicative of hyperprolactinemia.
4. In women, prolactin levels >27 ng/mL in the absence of pregnancy and postpartum lactation are indicative of hyperprolactinemia.
5. Clear symptoms and signs of hyperprolactinemia are often absent in patients with serum prolactin levels <100 ng/mL.
4. Mild to moderately increased levels of serum prolactin are not a reliable guide for determining whether a prolactin-producing pituitary adenoma is present, 5. Whereas levels >250 ng/mL are usually associated with a prolactin-secreting tumor.

CAUTION:

Prolactin values that exceed the reference values may be due to macroprolactin (prolactin bound to immunoglobulin). Macroprolactin should be evaluated if signs and symptoms of hyperprolactinemia are absent, or pituitary imaging studies are not informative.



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TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	<0.100	ng/mL	0.1 - 0.9
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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




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ANTI MULLERIAN HORMONE (AMH) GEN II

ANTI MULLERIAN HORMONE (AMH) GEN II: SERUM 0.071 ng/mL 0.02 - 6.35
by ECLIA (ELECTROCHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:-

A Correlation of FERTILITY POTENTIAL and AMH levels are :

OVARIAN FERTILITY POTENTIAL	AMH VALUES IN (ng/mL)
OPTIMAL FERTILITY:	4.00 – 6.80 ng/mL
SATISFACTORY FERTILITY:	2.20 – 4.00 ng/mL
LOW FERTILITY:	0.30 – 2.20 ng/mL
VERY LOW/UNDETECTABLE:	0.00 – 0.30 ng/mL
HIGH LEVEL:	>6.8 ng/mL (PCOD/GRANULOSA CELL TUMOUR)

Anti Mullerian Hormone (AMH) is also known as Mullerian Inhibiting Substance provided by sertoli cells of the testis in males and by ovarian granulose cells in females upto antral stage in females.

IN MALES:

1.It is used to evaluate testicular presence and function in infants with intersex conditions or ambiguous genitalia, and to distinguish between cryptorchidism and anorchia in males


IN FEMALES:


- 1.During reproductive age, follicular AMH production begins during the primary stage, peaks in preantral stage & has influence on follicular sensitivity to FSH which is important in selection for follicular dominance. AMH levels thus represents the pool or number of primordial follicles but not the quality of oocytes. AMH does not vary significantly during menstrual cycle & hence can be measured independently of day of cycle.
- 2.Polycystic ovarian syndrome can elevate AMH 2 to 5 fold higher than age specific reference range & predict anovulatory, irregular cycles, ovarian tumours like Granulosa cell tumour are often associated with higher AMH levels.
- 3.Obese women are often associated with diminished ovarian reserve and can have 65% lower mean AMH levels than non-obese women.
- 4.In females , AMH levels do not change significantly throughout the menstrual cycle and decrease with age.
- 5.Assess Ovarian Reserve - correlates with the number of antral follicles in the ovaries.
- 6.Evaluate fertility potential and ovarian response in IVF- Women with low AMG levels are more likely to the poor ovarian responders.
- 7.Assess the condition of Polycystic Ovary and premature ovarian failure.

A combination of Age, Ultrasound markers-Ovarian Volume and Antral Follicle Count, AMH and FSH levels are useful for optimal assessment of ovarian reserve. Studies in various fertility clinics are ongoing to establish optimal AMH concentration for predicting response to invitro fertilization, however, given below is suggested interpretative reference.

AMH levels (ng/mL)	Suggested patient	Anticipated Antral	Anticipated FSH levels	Anticipated Response
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Test Name	Value	Unit	Biological Reference interval
	Categorization for fertility based on AMH for age group (20 to 45 yrs)	Follicle counts (day 3)	to IVF/COH cycle
Below 0.3	Very low	Below 4	Above 20
0.3 to 2.19	Low	4 - 10	Usually 16 - 20
2.19 to 4.00	Satisfactory	11 - 25	Within reference range or between 11 - 15
Above 4.00	Optimal	Upto 30 and Above	Within reference range or between 11 - 15 or Above 15

INCREASED:

1. Polycystic ovarian syndrome (most common)
2. Ovarian Tumour: Granulosa cell tumour

DECREASED:

1. Anorchia , Abnormal or absence of testis in males
2. Pseudohermaphroditism
3. Post Menopause

NOTE:

1. AMH measurement alone is seldom sufficient for diagnosis and results should be interpreted in the light of clinical finding and other relevant test such as ovarian ultrasonography(In fertility applications); abdominal or testicular ultrasound(intersex or testicular function applications); measurement of sex steroids (estradiol, Progesterone, Testosterone), FSH, Inhibin B (For fertility), and Inhibin A and B (for tumour work up).
2. Conversion of AMH from ng/mL to pmol/L can be performed by using equation $1 \text{ ng/mL} = 7.14 \text{ pmol/L}$



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DEHYDROEPIANDROSTERONE SULPHATE (DHEA-S)

DIHYDROEPIANDROSTERONE SULPHATE (DHEA-S)	61.641	µg/dL	19.00 - 231.00
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by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:-

CLINICAL USE:

1. Marker for Adrenal cortical function and disease
2. Differential diagnosis of virilised patient. In patients with virilising tumours, DHEAS levels usually exceed 7000 g/dL

INCREASED LEVELS:

1. Adrenogenital syndromes due to deficiency of 3 beta-dehydrogenase, 21-hydroxylase and 11 beta-hydroxylase.
2. Congenital Adrenal Hyperplasia
3. Adrenal Carcinoma
4. Virilizing tumor of adrenal gland.
5. Cushing's disease, pituitary dependent.
6. Hirsutism
7. Polycystic ovarian Syndrome (PCOD)

DECREASED LEVELS:


1. Addison's disease
2. Adrenal Hypoplasia
3. Hyperlipidaemia
4. Psychoses
5. Psoriasis
6. Increasing age.


NOTE:

1. DHEA decreases in the elderly to a greater extent than do other steroids.

*** End Of Report ***




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