NAME	**: Mrs. HARJEET KAUR**		
AGE/ GENDER	: 41 YRS/FEMALE	**PATIENT ID**	: 1687940
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122412020015**
REFERRED BY	:	**REGISTRATION DATE**	: 02/Dec/2024 10:21 AM
BARCODE NO.	: 12505955	**COLLECTION DATE**	: 02/Dec/2024 10:26AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 02/Dec/2024 12:29PM
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Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	1.35	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	8.74	µgm/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.63	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRASENSITIVE

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:-

1. 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.
2. 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 (e.g.: phenytoin , salicylates).
3. 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.
4. 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
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 – 16.16	6 – 12 Months	0.70 - 7.00

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Test Name			Value	Unit	Biological Reference interval
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 (µIU/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, iodine 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 goiter & 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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Test Name	Value	Unit	Biological Reference interval
CORTISOL: MORNING (8 A.M. - 10 A.M.)			
CORTISOL MORNING (8 A.M. - 10 A.M.) *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**38.81^{H}**	µg/dL	4.26 - 24.85

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 adrenocorticotropic 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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Test Name	Value	Unit	Biological Reference interval

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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Test Name	Value	Unit	Biological Reference interval

LUTEINISING HORMONE (LH)

LUTEINISING HORMONE (LH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	4.73	mIU/mL	MALES: 0.57 - 12.07 FOLLICULAR PHASE: 1.80 - 11.78 MID-CYCLE PEAK: 7.59 - 89.08 LUTEAL PHASE: 0.56 - 14.0 POST MENOPAUSAL WITHOUT HRT: 5.16 - 61.99

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 possiblei mplantation.
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 adjunctin 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 ELEVTED 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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Test Name	Value	Unit	Biological Reference interval

FOLLICLE STIMULATING HORMONE (FSH)

FOLLICLE STIMULATING HORMONE (FSH): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	4.19	mIU/mL	FEMALE FOLLICULAR PHASE: 3.03 - 8.08 FEMALE MID-CYCLE PEAK: 2.55 - 16.69 FEAMLE LUTEAL PHASE: 1.38 - 5.47 FEMALE POST-MENOPAUSAL: 26.72 - 133.41 MALE: 0.95 - 11.95

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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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

PROLACTIN

PROLACTIN: SERUM	20.3	ng/mL	3 - 25

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

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, antinausea/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 (valporic acid), anti-tuberculous medications (Isoniazid).

SIGNIFICANCE:

1.In loss of libido, galactorrhea, oligomHyperprolactinemia often results enorrhea 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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Test Name	Value	Unit	Biological Reference interval

ADRENOCORTICOTROPHIC HORMONE (ACTH)

ADRENOCORTICOTROPHIC HORMONE (ACTH) *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**62.9^{H}**	pg/mL	0.00 - 46.00

INTERPRETATION:

CONDITION	UNITS	REFERENCE RANGE FOR ACTH
HEALTHY ADULT	pg/mL	10 - 46
CORD BLOOD SERUM	pg/mL	50 - 570
NEW BORNS	pg/mL	10 - 185

Adrenocorticotropic hormone (ACTH), the primary stimulator of adrenal cortisol production, is synthesized by the pituitary in response to corticotropin-releasing hormone (CRH), which is released by the hypothalamus.

Plasma ACTH and cortisol levels are both pulsatile and circardian exhibit peaks (6-8 a.m.) and nadirs (11 p.m.).

Cortisol, the main glucocorticoid, plays a central role in glucose metabolism and in the body's response to stress.

In a patient with hypocortisolism, an elevated adrenocorticotropic hormone (ACTH) indicates primary adrenal insufficiency, whereas a value that is not elevated is consistent with secondary adrenal insufficiency from a pituitary or hypothalamic cause

In a patient with hypercortisolism (Cushing syndrome), a suppressed value is consistent with a cortisol-producing adrenal adenoma or carcinoma, primary adrenal micronodular hyperplasia, or exogenous corticosteroid use.

Normal or elevated ACTH in a patient with Cushing syndrome puts the patient in the ACTH-dependent Cushing syndrome category. This is due t either an ACTH-producing pituitary adenoma or ectopic production of ACTH (bronchial carcinoid, small cell lung cancer, others). Furthe diagnostic studies such as dexamethasone suppression testing, corticotropin-releasing hormone stimulation testing, petrosal sinus sampling, and imaging studies are usually necessary to define the ACTH source.

CLINICAL USE

1.Diagnose disorders of the hypothalamic pituitary system
2.Differentiate Cushing's syndrome from normal patients when ACTH levels are low

INCREASED LEVELS

1.Stress
2.Addison's disease
3.Pituitary Cushing's disease

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Test Name	Value	Unit	Biological Reference interval

4.ACTH secreting tumor

DECREASED LEVELS

1.Adrenal adenoma
2.Adrenal carcinoma

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Test Name	Value	Unit	Biological Reference interval
GROWTH HORMONE			
GROWTH HORMONE (GH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.3	ng/mL	0.0 - 5.0

Intrepretation:-

1.The anterior pituitary secretes human growth hormone (hGH) in response to exercise, deep sleep, hypoglycemia, and protein ingestion.
2.hGH stimulates hepatic insulin-like growth factor-1 and mobilizes fatty acids from fat deposits to the liver.
3. Hyposecretion of hGH causes dwarfism in children. Hypersecretion causes gigantism in children or acromegaly in adults.
4.Because hGH levels in normal and diseased populations overlap, hGH suppression and stimulation tests are needed to evaluate conditions of hGH excess and deficiency; random hGH levels are inadequate.
5.The test has limited value in assessing growth hormone secretion in normal children.

Note:-

(a). IGF1I / Insulin-Like Growth Factor 1, Serum is recommended as the first test for assessing deficient or excess growth during childhood and adolescent development. Suspected causes of dwarfism need to be diagnosed with the aid of provocative testing.
(b). This test is not useful as a screen for acromegaly; IGF1I / Insulin-Like Growth Factor 1, Serum is preferred. Elevated levels of human growth hormone indicate the possibility of gigantism or acromegaly, but must be confirmed with stimulation and suppression testing.
(c). Growth hormone is secreted in surges; single measurements are of limited diagnostic value.

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Test Name	Value	Unit	Biological Reference interval

SPECIAL INVESTIGATIONS

INSULIN GROWTH FACTOR - 1/SOMATOMEDIN-C

INSULIN GROWTH FACTOR (IGF) - 1 SOMATOMEDIN-C: SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	109	ng/mL	58.0 - 219.0

INTERPRETATION:

1.Insulin-like growth factor- I (IGF- I) bioactivity is regulated by genetic and non-genetic factors like growth hormone, nutrition and insulin.
2.The rate of development of microalbuminuria (MA), an important early marker of diabetic nephropathy, has been related not only to factors such as age at diagnosis, sex and blood pressure, but also with the activity of the growth hormone–insulin-like growth factor- I (GH–IGF- I) axis.
3.Poor glycaemic control in type I diabetes, the most important factor for diabetic complications, is associated with elevated GH secretion and serum IGF binding protein (IGFBP)-1 levels, as well as reduced serum IGF- I levels.
4.In addition, derangements of the GH–IGF- I axis have been associated with hyperfiltration and MA in type I diabetes.
5.The mechanism behind this imbalance in the GH–IGF- I axis in type 1 diabetes has been suggested to be due to relatively low portal insulin levels resulting from s.c. administration of insulin.
6.Complete correction of the GH–IGF- I axis only seems possible with portal administration of insulin.
7.In the type I, II diabetes, GH / IGF- I axis is abnormal, GH increased, IGF- I reduced.
8.In type I diabetes, liver resistant GH, leading the liver IGF- I concentrations decreased.
9.At the same time, more IGFBP-I are generated, IGFBP-I can play a role in binding to and inhibit IGF- I .
10.This reduction of IGF- I cause the feedback of growth hormone's decrease.
11.Increased release of GH will lead to high blood sugar by antagonizing the function of insulin.
12.At the same time, the reduction of IGF- I also led to j growth retardation of juvenile or young with type I diabetes.
13.In poorly controlled type II diabetes, there will be also a high release of GH, antagonising the effect of peripheral tissues' insulin.
14.In any kind of diabetes, IGF- I can improve the control of blood sugar and reduce the serum GH's insulin-resistance in addition, IGF- I is important factor to adjust the function of bone cell and metabolism

INCREASED

1.gigantism
2.acromegaly
3.pregnancy.

DECREASED

1.growth hormone deficiencies
2.hypopituitarism.

NOTE:

IGF-1 may be normal in 5-10 % cases of acromegaly and 10-20 % cases of dwarfism.

*** End Of Report ***

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