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NAME	: Miss. ARCHI AHUJA	PATIENT ID	: 1795921
AGE/ GENDER	: 24 YRS/FEMALE	REG. NO./LAB NO.	: 012503180023
COLLECTED BY	: SURJESH	REGISTRATION DATE	: 18/Mar/2025 11:00 AM
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	COLLECTION DATE	: 18/Mar/2025 11:05AM
BARCODE NO.	: 01527325	REPORTING DATE	: 18/Mar/2025 01:06PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HbA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	5.2	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	102.54	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):

REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HbA1C) in %
Non diabetic Adults >= 18 years	<5.7
At Risk (Prediabetes)	5.7 – 6.4
Diagnosing Diabetes	>= 6.5
Therapeutic goals for glycemic control	Age > 19 Years
	Goals of Therapy:
	Actions Suggested:
	Age < 19 Years
	Goal of therapy:

COMMENTS:

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targeting a goal of < 7.0% may not be appropriate.
- High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shortens RBC life span like acute blood loss, hemolytic anemia falsely lowers HbA1c results.
- HbA1c results from patients with HbSS, HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.




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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F) AND POST PRANDIAL (PP)

GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	93.99	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
GLUCOSE POST PRANDIAL (PP): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	109.11	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > OR = 200.0

INTERPRETATION:

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose below 100 mg/dL and post-prandial plasma glucose level below 140 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl and post-prandial plasma glucose level between 140 – 200 mg/dL is considered as glucose intolerant or pre diabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dL and post-prandial plasma glucose level above 200 mg/dL is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.





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ENDOCRINOLOGY LUTEINISING HORMONE (LH)

LUTEINISING HORMONE (LH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	6.55	mIU/ml	FEMALE FOLLICULAR PHASE: 4.0 - 20.0 MID CYCLE PEAK: 20.0 - 120.0 FEMALE LUTEAL PHASE: 4.0 - 30.0 MENOPAUSAL: 40.0 - 200.0 PRIMARY HYPOGONADISM: >40.0 MALE: 2.0 - 12.0
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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





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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	3.43	mIU/ml	FEMALE FOLLICULAR PHASE: 2.0 - 15.0 FEMALE LUTEAL PHASE: 2.0 - 12.0 FEMALE OVULATORY PHASE: 2.0 - 25.0 MENOPAUSAL: >40.0 PREGNANCY: 0.0 - 12.0 PRIMARY OVARIAN FAILURE: 40.0 - 150.0 MALE: 2.0 - 15.0
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by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:

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

The test is useful in the following settings:

- An adjunct in the evaluation of menstrual irregularities.
- Evaluating patients with suspected hypogonadism.
- Predicting ovulation
- Evaluating infertility
- Diagnosing pituitary disorders
- 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:

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

NOTE:

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




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PROLACTIN

PROLACTIN: SERUM **29^H** 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, 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 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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INSULIN FASTING (F)

INSULIN FASTING (F)	13.34	μIU/ml	2.0 - 25.0
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by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:-

1. Insulin is a hormone produced by the beta cells of the pancreas. It regulates the uptake and utilization of glucose and is also involved in protein synthesis and triglyceride storage.
2. Type 1 diabetes (insulin-dependent diabetes) is caused by insulin deficiency due to destruction of insulin producing pancreatic islets (beta) cells.
3. Type 2 diabetes (noninsulin dependent diabetes) is characterized by resistance to the action of insulin (insulin resistance).
4. The test is useful for management of diabetes mellitus and for diagnoses of insulinomas, when used in conjunction with proinsulin and C-peptide measurements.

NOTE:


1. No standard reference range has yet been established for INSULIN POST-PRANDIAL (PP) in Indian population, therefore same could not be provided along with test. However various studies done on several populations mention that the range of INSULIN PP can vary somewhere from 5-79 mIU/L which can be used for clinical purpose.

2. This assay has 100% cross-reactivity with recombinant human insulin (Novolin R and Novolin N). It does not recognize other commonly used analogues of injectable insulin (ie, insulin lispro, insulin aspart, and insulin glargine).

INTERPRETATIVE GUIDE:

1. During prolonged fasting, when the patient's glucose level is reduced to <40 mg/dL, elevated insulin level plus elevated levels of proinsulin and C-peptide suggest insulinoma.
2. Insulin levels generally decline in patients with type 1 diabetes mellitus.
3. In the early stage of type 2 diabetes, insulin levels are either normal or elevated. In the late stage of type 2 diabetes, insulin levels decline.
4. In normal individuals, insulin levels parallel blood glucose levels.
5. Patients on insulin therapy may develop anti-insulin antibodies. These antibodies may interfere in the assay system, causing inaccurate results. In such individuals, measurement of free insulin FINS / Insulin, Free, Serum should be performed.




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TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	0.59	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




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IMMUNOPATHOLOGY/SEROLOGY

ANTI THYROID PEROXIDASE (TPO/AMA) ANTIBODIES

ANTI TPO/AMA ANTIBODIES: SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	29.31^H	IU/mL	0.00 - 10.0 DIABETES (II): < 25.0
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INTERPRETATION:

1. Thyroperoxidase (TPO) is an enzyme involved in thyroid hormone synthesis, catalyzing the oxidation of iodide on tyrosine residues in thyroglobulin for the synthesis of triiodothyronine and thyroxine (tetraiodothyronine).
2. TPO is a membrane-associated hemo glycoprotein expressed only in thyrocytes and is one of the most important thyroid gland antigens.
3. Anti-TPO is technically superior and a more specific method for measuring thyroid auto-antibodies, It is especially useful in patients presenting with subclinical hypothyroidism where TSH is elevated but Free T4 levels are normal.

INCREASED LEVELS (Autoimmune thyroid disease):

1. Hashimoto thyroiditis.
2. Idiopathic myxedema.
3. Graves disease
4. Post-partum thyroiditis.
5. Primary hypothyroidism due to Hashimoto thyroiditis.

NOTE:

1. The highest TPO antibody levels are observed in patients suffering from Hashimoto thyroiditis. In this disease, the prevalence of TPO antibodies is about 90% of cases, confirming the autoimmune origin of the disease.
2. These auto-antibodies also frequently occur (60%-80%) in the course of Graves disease.
3. In patients with subclinical hypothyroidism, the presence of TPO antibodies is associated with an increased risk of developing overt hypothyroidism.

*** End Of Report ***




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