



LABORATORY REPORT

NAME	: MR.BC0865	REFERRED BY	: SELF	VISIT NO	: VAMP26148126
AGE	: 40Y 0M 0D	ZERO TARIFF CLIENT CODE		COLLECTED ON	: 21-04-2026 10:00
GENDER	: Male	LAB MR#	: AAMP01479405	RECEIVED ON	: 21-04-2026 19:51
OP / IP / DG #	:			APPROVED ON	: 22-04-2026 18:05
				REPORT STATUS	: Final Report



Test Name	Result	Biological Ref. Interval	Unit
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Hirsutism Evaluation Panel Iii

BIOCHEMISTRY

Dehydroepiandrosterone (DHEA) (Serum)

Dehydroepiandrosterone (DHEA) ELISA	4.25	1.28 - 9.21	ng/mL
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Dehydroepiandrosterone Sulphate, DHEA-S (Serum)

Dehydroepiandrosterone Sulphate, DHEA-S ECLIA	328.00	88.90 - 427.00	µg/dL
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Interpretation:

DHEAS is a steroid hormone synthesized from cholesterol in zona reticularis and broad fascia of adrenal cortex. DHEAS is an excellent indicator of adrenal cortex androgen production. Elevated DHEAS seen in adrenocortical carcinoma Cushing's disease, congenital adrenal hyperplasia. Decreased levels seen in adrenal insufficiency due to primary adrenal insufficiency in hirsute females, increased DHEAS levels have been associated with virilising adrenal tumours patients with polycystic ovary syndrome having elevated levels of DHEAS suggesting an adrenal androgen contribution to the defect in this disorder

Testosterone - Free (Serum)

Testosterone - Free ELISA	21.200	5.7 - 30.7	pg/mL
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Interpretation:

Testosterone circulates in blood bound to three proteins: sex hormone binding globulin (SHBG, 60-80%), albumin and cortisol binding globulin. About 1 - 2% of the total circulating testosterone remains unbound or free. Measurement of free testosterone permits the estimation of the biologically active hormone. Free testosterone determination is recommended to overcome the influences caused by variations in transport proteins on the total testosterone concentration. High concentration of SHBG (as seen in obesity, advanced age etc) may mask true deficit in testosterone levels. In Polycystic Ovarian Syndrome and related conditions, there is often significant insulin resistance, which is associated with low SHBG levels. Consequently, bioavailable or free testosterone levels may be more significantly elevated.

Clinical Use

As second-level test for suspected increases or decreases in physiologically active testosterone

- To assess androgen status in cases with suspected or known sex hormone-binding globulin-binding abnormalities
- To assess functional circulating testosterone in early pubertal boys and older men
- To assess functional circulating testosterone in women with symptoms or signs of hyperandrogenism but normal total testosterone levels

Testosterone - Total (Serum)

Testosterone - Total ECLIA	4.25	2.80-8.0	ng/mL
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Interpretation:

In men testosterone is synthesized almost exclusively by the leydig cells of testis. Most of the circulation testosterone is bound to carrier proteins. in women, small quantities of testosterone are formed in the ovaries

- Determination of testosterone in woman is helpful in diagnosis of
- Polycystic ovaries (Stein – Leventhal syndrome)
- Management of hirsutism & virilisation in females

In men reduced production:

- Hypogonadism
- Oestrogen therapy
- Chromosome aberrations (as in Klinefelter’s syndrome)
- Liver cirrhosis
- Delayed puberty

Increased production:

- Precocious puberty
- Congenital adrenal hyperplasia

Leutinizing Hormone,LH (Serum)

Leutinizing Hormone,LH ECLIA	4.85	1.7 - 8.6	mIU/mL
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Interpretation:

A glycoprotein gonadotropic hormone secreted by anterior pituitary that acts with FSH to promote ovulation and androgen and progesterone production.
Elevated levels of LH:
-Primary gonadal failure in both males and females.
-Precocious puberty
-Menopause
-Primary hypogonadism in males.
Decreased in pituitary or hypothalamus insufficiency.

Follicle Stimulating Hormone, FSH (Serum)

Follicle Stimulating Hormone, FSH ECLIA	6.6	1.5 - 12.40	mIU/mL
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Hirsutism Evaluation Panel Iii

Sex Hormone Binding Globulin (SHBG) (Serum)

Sex Hormone Binding Globulin (SHBG) ECLIA	45.00	18.3-54.1	nmol/L
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Interpretation:

INTERPRETATIONS OF SHBG:

Sex hormone-binding globulin (SHBG) is the blood transport protein for testosterone and estradiol (E2). SHBG is produced mainly by the liver and its synthesis and secretion are regulated by estrogen and negatively influenced by liver fat content and inflammatory cytokines.

Decreased SHBG serum levels are seen in:

1. Elevated androgen levels are present or where the effect of androgen on its target organs is excessive.
2. Inflammation and in case of a diet leading to fat build-up in the liver e.g. rich in monosaccharides, particularly fructose.
3. It correlates with cardiovascular disease risk, type 2 diabetes as well as breast cancer.
4. Hypothyroidism, polycystic ovarian syndrome, obesity, hirsutism, elevated androgen levels, alopecia, and acromegaly

Elevated SHBG levels can be seen in:

1. Elderly men
2. Hyperthyroidism and cirrhosis
3. Oral contraceptives or antiepileptic drugs are taken
4. Pregnant women have markedly higher SHBG serum concentrations due to their increased estrogen production.





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Hirsutism Evaluation Panel Iii

17 - Alpha- Hydroxy Progesterone (Serum)

17 - Alpha- Hydroxy Progesterone ELISA	8.52	2.7 - 19.9	ng/mL
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Interpretation:

Measurements of levels of 17 α -OHP are useful in the evaluation of patients with suspected congenital adrenal hyperplasia as the typical enzymes that are defective, namely 21-hydroxylase and 11 β -hydroxylase, lead to a build-up of 17 α -OHP. In contrast, the rare patient with 17 α -hydroxylase deficiency will have very low or undetectable levels of 17 α -OHP. 17 α -OHP levels can also be used to measure contribution of progesterone activity of the corpus luteum during pregnancy as progesterone but note, 17 α -OHP is also contributed by the placenta

Androstenedione (Serum)

Androstenedione CLIA	2.45	0.7 - 3.6	ng/mL
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Interpretation:

1. This is a major adrenal androgen also produced by the testes and ovaries.
2. It is increased in polycystic ovarian disease, hirsutism, virilization, congenital adrenal hyperplasia, adrenal and ovarian tumors and Cushing's disease, pregnancy and exercise.
3. It is decreased in Addison's disease.

Free androgen Index (FAI) (Serum)

Testosterone - Total ECLIA	4.25	2.80-8.0	ng/mL
Sex Hormone Binding Globulin (SHBG) ECLIA	45.00	18.3-54.1	nmol/L
Free androgen Index (FAI) Calculation	32.77	30-140	

Interpretation:

Androgen index (AI) is a useful indicator of an abnormal androgen status. The AI is often increased in severe acne, male androgenic alopecia (balding), hirsutism, and other conditions in which a normal total testosterone level is found with a low SHBG level. In non-obese, non-hirsute oligomenorrheic women, an elevated AI during the early follicular phase is reported to be a sensitive and specific indicator of Polycystic ovarian disease (PCOD).

Dihydrotestosterone (DHT) (Serum)

Dihydrotestosterone (DHT)	642.00	143 - 842	pg/mL
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Page 4 of 6

Sin No: 20385423





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Hirsutism Evaluation Panel Iii
ELISA

Interpretation:

The principal prostatic androgen is dihydrotestosterone (DHT). DHT is generated by reduction of testosterone by 5-alpha reductase. It exerts full androgenic effect on the certain target tissues such as hair follicles and external genitalia. DHT should serve as the primary marker of peripheral androgen production. However, because it is metabolized rapidly and has a very high affinity for sex hormone-binding globulin (SHBG), DHT does not reflect peripheral androgen action. Instead, its distal metabolite, 3-alpha, 17-beta-androstenediol glucuronide, serves as a better marker of peripheral androgen action.

Patients taking 5-alpha reductase inhibitor have decreased dihydrotestosterone (DHT) serum levels.

Patients with genetic 5-alpha reductase deficiency (a rare disease) also have reduced DHT and its metabolites. They fail to develop male genitalia and appear to be phenotypic females.

Levels of DHT remain normal with aging, despite a decrease in the plasma testosterone, and are not elevated in benign prostatic hyperplasia.

Patients with benign prostatic hyperplasia (BPH) or prostatic cancer may not have elevated dihydrotestosterone (DHT) levels even though growth of the prostate gland may be stimulated by DHT.

Sanjeeta

Dr. Sanjeeta
MBBS, MD (Biochemistry)
Consultant Biochemist

Disclaimer:

1. All results released pertain to the specimen as received by the lab for testing and under the assumption that the patient indicated or identified on the bill/test requisition form is the owner of the specimen.
2. Clinical details and consent forms, especially in Genetic testing, histopathology, as well as wherever applicable, are mandatory to be accompanied with the test requisition form. The non-availability of such information may lead to delay in reporting as well as misinterpretation of test results. The lab will not be responsible for any such delays or misinterpretations thereof.
3. Test results are dependent on the quality of the sample received by the lab. In case the samples are preprocessed elsewhere (e.g., paraffin blocks), results may be compromised.
4. Tests are performed as per the schedule given in the test listing and in any unforeseen circumstances, report delivery may be affected.
5. Test results may show inter-laboratory as well as intra-laboratory variations as per the acceptable norms.
6. Genetic reports as well as reports of other tests should be correlated with clinical details and other available test reports by

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Page 5 of 6



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Hirsutism Evaluation Panel Iii

a qualified medical practitioner. Genetic counselling is advised in genetic test reports by a qualified genetic counsellor, medical practitioner or both.

7. Samples will be discarded post processing after a specified period as per the laboratory's retention policy. Kindly get in touch with the lab for more information.

8. If accidental damage, loss, or destruction of the specimen is not attributable to any direct or negligent act or omission on the part of Ampath Labs or its employees, Ampath shall in no event be liable. Ampath lab's liability for a lack of services, or other mistakes and omissions, shall be restricted to the amount of the patient's payment for the pertinent laboratory services.

