LABORATORY REPORT

SRL RANBAXY
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Toll Free No.: 1800 222 333. E-mail: srlbom@srlranbaxy.co.in

CLIENT CODE : C000000613

CLIENT'S NAME AND ADDRESS :

TRIVEDI MAHENDRA KUMAR

PHN NO : 9869118769/32910208

REFERRING DOCTOR DR SHRIKANT A PATIL

DRAWN 06/02/2007 09:00 RECEIVED 06/02/2007 09:05 REPORTED 10/02/2007 15:52

PATIENT NAME TRIVEDI MAHENDRA KUMAR

ACCESSION NO. 0002GB012612 AGE 43 Years SEX Male DATE OF BIRTH 10/02/1963 PATIENT ID

CLINICAL INFORMATION

TEST REPORT STATUS **FINAL**

	RESULTS IN RANGE	OUT OF RANGE	REFERENCE RANGE	UNITS
GROWTH HORMONE, SERUM				
HUMAN GROWTH HORMONE		L < 0.05	0.06 - 5.00	ng/mL
ADRENOCORTICOTROPIC HORMONE, PLASMA				
ADRENOCORTICOTROPIC HORMONE	39.9		10.0 - 46.0	pg/mL
TSH 3RD GENERATION, SERUM				
TSH 3RD GENERATION	4.40		0.35 - 5.50	µIU/mL
FSH & LH EVALUATION, SERUM				
LUTEINIZING HORMONE	4.01		1.50 - 9.30	mIU/mL
FOLLICLE STIMULATING HORMONE		L 1.11	1.40 - 18.10	mIU/mL
LH/FSH RATIO		H 3.61	0.00 - 2.00	
ANTIDIURETIC HORMONE				
ANTIDIURETIC HORMONE	1.75		1.0 - 14.0	pg/ml
TSH RECEPTOR ANTIBODIES, SERUM				
TSH RECEPTOR ANTIBODIES	<5.0		< 9.0 (Negative) 9.0 - 14.0 (Indeterminate) > 14.0 (Positive)	U/L
CORTISOL, SERUM				
CORTISOL	17.99		7:00-9:00 a.m.: 4.30 - 22.40 3:00-5:00 p.m.: 3.09 - 16.66	ug/dL
PROGESTERONE, SERUM				
PROGESTERONE	1.17		0.28 - 1.22	ng/mL
PARATHYROID HORMONE (INTACT), SERUM				
CALCIUM	9.0		8.5 - 10.1	mg/dL
PTH (INTACT)	45.1		14.0 - 72.0	pg/mL
IMMUNOGLOBULIN				
TOTAL IgA	2.85		0.90 - 4.50	g/L
TOTAL	13.37		8.00 - 18.00	g/L

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	IN RANGE	OUT OF RANGE	REFERENCE RANGE	UNITS
TOTAL IgM	0.70		0.60 - 2.50	g/L

COMMENT:

****

NOTE : RECHECKED FOR SERUM FSH .
PLEASE CORRELATE CLINICALLY.

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ACCESSION NO.	AGE	SEX	DATE OF BIRTH	PATIENT ID
0002GB012612	43 Years	Male	10/02/1963	

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	RESULTS			
	IN RANGE	OUT OF RANGE	REFERENCE RANGE	UNITS

CORTISOL, SERUM

Cortisol is a primary glucocorticoid hormone synthesized and secreted by the adrenal cortex. Cortisol plays an important role in regulating carbohydrate, protein and lipid metabolism, maintaining normal blood pressure and inhibiting allergic and inflammatory reactions. Cortisol is synthesized and secreted by the cortex of the adrenal gland under the effect of adrenocorticotropic hormone (ACTH).

Circulating Cortisol levels follow a diurnal pattern in healthy individuals. Levels are highest in the morning after waking and lowest in the evening. Disorders of the hypothalamic pituitary adrenal axis override this diurnal pattern.

Decreased Cortisol levels are induced by either primary or secondary adrenal insufficiency. Addison's disease is caused by primary adrenal insufficiency due to metabolic errors or destruction of the adrenal cortex. Secondary adrenal insufficiency is caused by pituitary destruction or failure, resulting in loss of ACTH stimulation of the adrenal gland.

Increased levels of Cortisol due to either primary or secondary adrenal hyper function cause Cushing's syndrome. Causes of primary adrenal hyper function are adrenal tumors and nodular adrenal hyperplasia. Secondary adrenal hyper function is caused by pituitary overproduction of ACTH or ectopic production of ACTH by a tumor. Increased Cortisol levels are induced by pregnancy and by stress due to depression, trauma, surgery, hypoglycemia, alcoholism, uncontrolled diabetes and starvation.

Due to the diurnal pattern of secretion, an assessment of serum Cortisol at a single time-point is of little diagnostic value. The ACTH stimulation test is used to evaluate Addison's disease. The dexamethasone suppression test is used to diagnose Cushing's syndrome or depression due to neuroendocrine disorders.

Test method: Chemiluminescence.

GROWTH HORMONE, SERUM

Human growth hormone (hGH, somatotropin) is a polypeptide originating in the anterior pituitary. It is 191 amino acids in length and has a molecular mass of approximately 22,000 daltons. Its metabolic effects are primarily anabolic. It promotes protein conservation and engages a wide range of mechanisms for protein synthesis. It also enhances glucose transport and facilitates the buildup of glycogen stores.

Measurement of hGH is primarily of interest in the diagnosis and treatment of various forms of inappropriate growth hormone secretion. Clinical disorders of hyposecretion include dwarfism and unattained growth potential. Hypersecretion is associated with gigantism and acromegaly.

Caution must be exercised in the clinical interpretation of growth hormone levels. These vary throughout the day, making it difficult to define a reference range or to judge an individual's status based on single determinations. Many factors are known to influence the rate of growth hormone secretion, including periods of sleep and wakefulness, exercise, stress, hypoglycemia, estrogens, corticosteroids, L-dopa, and others.

Growth hormone-deficient individuals have fasting/resting levels similar to those found in healthy individuals. Various challenge tests have therefore been devised to differentiate these groups. Thus with the onset of deep sleep or after 15 to 20 minutes of vigorous exercise, growth hormone levels normally show a rise. Other tests of growth hormone responsiveness are based on the administration of L-dopa, arginine and insulin. Propanolol and estrogen are sometimes given in conjuction with the primary stimulus to accentuate the response.

A small number of cases of dwarfism have been documented in which both the basal level and the response to challenge testing were normal. Such cases may involve tissue insensitivity to either growth hormone or somatomedins, or the presence of antibodies or immunoreactive but biologically inactive growth hormone.

REFEREN FOR GROWTH HORMONE STIMULATION TEST.
Post- sti mal peak levels of GH are 10 ng/mL or more. In children, GH levels 7.0 ng/mL or less and in adults GH levels of 5.0 ng/mL or less indicate deficiency. GH levels between normal and deficient states are considered as Indeterminate.

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	IN RANGE	OUT OF RANGE	REFERENCE RANGE	UNITS

ADRENOCORTICOTROPIC HORMONE, PLASMA

Plasma levels of ACTH exhibit a significant diurnal variation. ACTH determinations are valuable in the differential diagnosis of Adrenal Insufficiency and hypersecretion.
In Addison's disease (primary adrenal insufficiency), elevated levels are typical, where as low levels are the rule when adrenal insufficiency is secondary to pituitary dysfunction.

ACTH determinations can also help to identify the cause of Cortisol hypersecretion in Cushing's syndrome. ACTH levels are typically low when this is due to lesions or Hyperplasia of the Adrenal Cortex, and high when it is due to ectopic ACTH production or Hypersecretion of ACTH by the Pituitary.

Important: The minimum detectable level of ACTH by this Assay is 10 pg/mL.

Test method: Chemiluminescence

PROGESTERONE, SERUM

Progesterone, in conjunction with estrogens, regulates reproductive tract functions during the menstrual cycle. Progesterone is critical in preparing the endometrium for blastocyst implantation and the maintenance of pregnancy.

The major sources of progesterone are the corpus luteum and the placenta in women. Minor sources of progesterone are the adrenal cortex in men and women, and the testes in men.

Progesterone levels are low during the follicular phase of the menstrual cycle. After ovulation, progesterone production by the corpus luteum increases rapidly, reaching a maximum concentration 4 to 7 days after ovulation. These levels are maintained for 4 to 6 days then fall to baseline levels, inducing menstruation.

During pregnancy, progesterone levels rise steadily to their highest levels in the third trimester.
Reference ranges for Pregnant Females:
First Trimester: 11.22 - 90.00
Second Trimester: 25.55 - 89.40
Third Trimester: 48.40 - 422.50

Clinical evaluation of progesterone confirms ovulation and normal luteal function in nonpregnant women. Inadequate progesterone production by the corpus luteum may indicate luteal phase deficiency (LPD), which is associated with infertility and early miscarriage. Women using oral contraceptives have suppressed progesterone level.

Heterophilic antibodies in human serum can react with reagent immunoglobulins, interfering with in vitro immunoassays. Patients routinely exposed to animals or to animal serum products can be prone to this interference and anomalous values may be observed.

Hormone assay values are to be correlated with the age and clinical status of the patient irrespective of whether the values are appearing in the 'In Range' or 'Out of Range' columns.

Test method: Chemiluminescence.

TSH 3RD GENERATION, SERUM

Thyroid-stimulating hormone is a glycoprotein with two non-covalently bound subunits. The alpha subunit is similar to those of follicle-stimulating hormone, human chorionic gonadotrophin (hCG) and Luteinizing hormone (LH). The beta subunit of TSH is unique, which results in the specific biochemical and immunological properties of this hormone. TSH is synthesized and secreted by the anterior pituitary in response to a negative feedback mechanism involving concentrations of FT3 (Free T3) and FT4 (Free T4). Additionally, the hypothalamic tripeptide, thyrotropin-releasing hormone (TRH),

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RESULTS

IN RANGE OUT OF RANGE REFERENCE RANGE UNITS

directly stimulates TSH production. TSH interacts with specific cell receptors on the thyroid cell surface and exerts two main actions. The first action is to stimulate cell reproduction and hypertrophy. Secondly, TSH stimulates the thyroid gland to synthesize and secrete T3 and T4.

The ability of quantitate circulating levels of TSH is important in evaluating thyroid function. It is especially useful in the differential diagnosis of primary (thyroid) from secondary (pituitary) and tertiary (hypothalamic) hypothyroidism. In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels are low. TRH stimulation differentiates secondary and tertiary hypothyroidism by observing the change in patient TSH levels. Typically, the TSH response to TRH stimulation is absent in cases of secondary hypothyroidism and normal to exaggerated in tertiary hypothyroidism. Historically, TRH stimulation has been used to confirm primary hyperthyroidism, indicated by elevated T3 and T4 levels and low or undetectable TSH levels. TSH assays with increased sensitivity and specificity provide a primary diagnostic tool to differentiate hyperthyroid from euthyroid patients.

Below mentioned are the guidelines for age related reference ranges of TSH:

Age	Reference Range	Units
Cord Blood	2.0 - 40.0	µIU/mL
1 - 6 days	0.4 - 15.0	µIU/mL
1 - 3 weeks	0.4 - 10.0	µIU/mL
1 month & over	0.4 - 5.0	µIU/mL

Test method: Chemiluminescence

FSH & LH EVALUATION, SERUM

Circulating FSH levels vary throughout the menstrual cycle in response to Estradiol and Progesterone. A small but significant increase in circulating FSH accompanies the mid- cycle LH surge. FSH declines in the luteal phase in response to Estradiol and Progesterone production by the developing Corpus Luteum. FSH is elevated and gonadal steroids are depressed include Menopause, Premature Ovarian Failure, and Ovariactomy, while with Polycystic Ovarian Syndrome the LH / FSH ratio may be increased. At Menopause, FSH and LH increase sufficiently in response to diminished feed back mechanism of Gonadotropin release. Elevated concentration of LH may indicate Primary Amenorrhea, Menopause, Premature Ovarian Failure, Polycystic Ovarian Syndrome, or Hypergonadotropic Hypogonadism. The levels of LH & FSH in women are to be correlated with the day of the menstrual cycle.

FSH, LH and Testosterone regulate spermatogenesis by the Sertoli cells in seminiferous tubules of the testes. FSH may also be elevated in Klinefelter's Syndrome (Seminiferous Tubule Dysgenesis) or as a consequence of Sertoli cell Failure. Elevated concentration of LH and FSH accompanied by low concentration of gonadal steroids may result in infertility due to Gonadal Failure. Elevated concentration of LH may result in Primary Testicular Failure, Seminiferous Tubule Dysgenesis. (Klinefelter's Syndrome), Sertoli cell Failure & Hypergonadotropic Hypogonadism.

Hormone assay values are to be correlated with the age and clinical status of the patient irrespective of whether the values are appearing in the 'In Range' or 'Out of Range' columns.

Test method: Chemiluminescence.

PARATHYROID HORMONE (INTACT), SERUM

Parathyroid hormone (PTH) produced by the parathyroid gland is the major circulating factor regulating extra cellular calcium concentration.

The intact PTH peptide (MW~9425) consists of 84 amino acids that are sequenced and designated according to reactivity. The N-terminal or amino-terminal 1-34 region of the intact PTH molecule is biologically active. This region of the molecule contains the amino acid sequence that enables PTH to bind to parathyroid hormone receptors in target tissues and regulate extra cellular calcium concentrations. The middle and carboxy terminal 35-84 region of the intact PTH molecule is biologically inert but possesses immunological reactivity.

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IN RANGE	OUT OF RANGE	REFERENCE RANGE	UNITS

Quantification of circulating intact PTH assists in the differential diagnosis of hypercalecmia. In conjunction with the measurement of ionized calcium, intact PTH evaluations can be used to distinguish between patients with hypoparathyroidism, hypoparathyroidism or hypercalcemia of malignancy.

The diagnosis of primary hyperparathyroidism, a common cause of hyper calcemia is confirmed by elevated ionized calcium concentrations and elevated parathyroid hormone concentrations. Intact PTH levels are also used to assess and manage other metabolic bone disorders including osteoporosis and renal osteodystrophy. The measurement of intact PTH using two site immunoassays provides a more accurate assessment of parathyroid tissue secretary status, especially in patients with renal impairment.

Interpretation of intact PTH values should always take into account serum Calcium results and inter-relationship between these two elements in various disorders involving PTH & Calcium. It is recommended that the intact PTH results should always be interpreted with caution & with consideration of the overall manifestations even when used in conjunction with calcium values.

Measurement of intact PTH is useful in differentiation between hypercalcemia due to hyperparathyroidism & hypercalcemia of malignancy. However the assay is not intended as and should not be relied upon as a diagnostic indicator of malignancy.

Heterophilic antibodies in human serum can react with reagent immunoglobulins, interfering with in vitro immunoassays. Patients routinely exposed to animals or to animal serum products can be prone to this interference and anomalous values may be observed. Additional information may be required for diagnosis.

Test method: Chemiluminescence.

Total Immunoglobulin

Serum IgG levels are decreased in several immunodeficiencies. In congenital hypogammaglobulinemia IgG is less than 200 MG/DL by 6 months of age. Acquired hypogammaglobulinemia may occur at any age and has IgG levels less than 500 MG/DL. IgG levels may also be decreased in combined cell-mediated and antibody immunodeficiencies. Lymphocyte phenotype and function studies may be helpful in evaluation of immunodeficiencies. Suspected paraproteinemias should be screened for with immuno electro phoresis. Selective deficiency of one or more IgG subclasses is associated with a variety of recurrent infections or asthma.

Total IgM evaluates humoral immunity; establishes the diagnosis and monitors therapy in Macroglobulinemia of Waldenstrom & Plasma Cell Myeloma. IgM levels are used to evaluate likehood of in utero infections or acuteness of infections.

IgA deficiency is the most common of the primary immunodeficiency diseases. It can be induced by drug such as penicillamine, phenytoin, sulfasalzine & captoril. IgA deficiency is also seen in autoimmune diseases.
Polymeric IgA is found in conditions resulting in parenchymal liver damage, IgA Nephropathy, utreated Coeliac Disease, Chronic bronchial suppurative disorders, herpes Simplex, Encephalites, Herpes zoster, Mumps & Meningitis.

Test method: Immunoturbidimetric assay.

** End Of Report **

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