# LABORATORY REPORT

SRL RANBAXY

**CLIENT CODE :** C000005460

**CLIENT'S NAME AND ADDRESS :**

OMKAR SURGICALS
RAMESH BHUVAN,SHOP NO.2,
OPP.KEM HOSPITAL MAIN GATE, J M STREET,PAREL,
MUMBAI, 400 012
MAHARASHTRA, INDIA, PHN NO : 4127589,9869384399

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**REFERRING DOCTOR**

| DRAWN | RECEIVED | REPORTED |
|---|---|---|
| 10/03/2008 08:00 | 10/03/2008 08:54 | 10/03/2008 20:41 |

**PATIENT NAME** MAHENDRAKUMAR TRIVEDI

| ACCESSION NO. | AGE | SEX | DATE OF BIRTH | PATIENT ID |
|---|---|---|---|---|
| 0002HC026841 | 45 Years | Male | 10/02/1963 | |

CLINICAL INFORMATION

**PRELIMINARY** RESULTS

| TEST REPORT STATUS | IN RANGE | OUT OF RANGE | REFERENCE RANGE | UNITS |
|---|---|---|---|---|
| **THYROID PANEL BY CHEMILUMINESCENCE, SERUM** | | | | |
| T3 | 161.3 | | 60.0 - 181.0 | ng/dl |
| T4 | 5.3 | | 4.5 - 12.6 | µg/dl |
| TSH | | H 6.61 | 0.35 - 5.50 | µIU/ml |
| **LH, FSH, PROLACTIN, SERUM** | | | | |
| LUTEINIZING HORMONE | 3.67 | | 1.50 - 9.30 | mIU/mL |
| FOLLICLE STIMULATING HORMONE | | L 0.66 | 1.40 - 18.10 | mIU/mL |
| PROLACTIN | 9.28 | | 2.10 - 17.70 | ng/mL |
| **CORTISOL, SERUM** | | | | |
| CORTISOL | 17.03 | | 7:00-9:00 a.m.: 4.30 - 22.40<br>3:00-5:00 p.m.: 3.09 - 16.66 | ug/dL |
| **GROWTH HORMONE, SERUM** | | | | |
| HUMAN GROWTH HORMONE | < 0.01 | | <0.01 - 1.00 | ng/mL |
| **PARATHYROID HORMONE (INTACT), SERUM** | | | | |
| CALCIUM | 9.4 | | 8.5 - 10.1 | mg/dL |
| PTH (INTACT) | 18.9 | | 14.0 - 72.0 | pg/mL |
| **ADRENOCORTICOTROPIC HORMONE, PLASMA** | RESULT PENDING | | | |
| **CALCITONIN (THYROCALCITONIN), SERUM** | RESULT PENDING | | | |
| **TESTOSTERONE, TOTAL, SERUM** | | | | |
| TESTOSTERONE, TOTAL | 588.12 | | 241.00 - 827.00 | ng/dl |
| **LACTATE DEHYDROGENASE, SERUM** | | | | |
| LACTATE DEHYDROGENASE | 139 | | 100 - 190 U/L | |
| **HOMOCYSTEINE, SERUM/PLASMA** | | | | |
| HOMOCYSTEINE | | H 23.16 | 5.00 - 13.90 | µmol/L |

Accredited by
**College of American Pathologists**



Cert.No.M-0025

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| **INSULIN, SERUM** | | | | |
| INSULIN | 17.07 | | 1.70 - 31.00 | mU/L |
| **GLUCOSE TOLERANCE TEST (GTT) - 8** | | | | |
| GLUCOSE,FASTING | 97 | | 70.0 - 115.0 | mg/dL |
| GLUCOSE,1/2 HR | | H 196 | 70.0 - 170.0 | mg/dL |
| GLUCOSE,1 HR | | H 184 | 90.0 - 160.0 | mg/dL |
| GLUCOSE,1 1/2 HR | 140 | | 80.0 - 140.0 | mg/dL |
| GLUCOSE,2 HR | 97 | | 75.0 - 130.0 | mg/dL |
| GLUCOSE,2 1/2 HR | TEST NOT PERFORMED | | 75.0 - 130.0 | mg/dL |
| GLUCOSE,3HR | TEST NOT PERFORMED | | 60.0 - 110.0 | mg/dL |
| GLUCOSE,3 1/2 HR | TEST NOT PERFORMED | | 70.0 - 115.0 | mg/dL |
| URINE SUGAR, 1/2 HOUR | NOT DETECTED | | NOT DETECTED | |
| URINE SUGAR, 1 HOUR | NOT DETECTED | | NOT DETECTED | |
| URINE SUGAR, 1 1/2 HOUR | NOT DETECTED | | NOT DETECTED | |
| URINE SUGAR, 2 HOUR | NOT DETECTED | | NOT DETECTED | |
| URINE SUGAR, FASTING | NOT DETECTED | | NOT DETECTED | |
| **INSULIN LIKE GROWTH FACTOR - I, SERUM** | | | | |
| INSULIN LIKE GROWTH FACTOR - I | 295.6 | | Refer to the chart below | ng/mL |
| **PROGESTERONE, SERUM** | | | | |
| PROGESTERONE | 0.50 | | 0.28 - 1.22 | ng/mL |
| **ESTRADIOL, SERUM** | | | | |
| ESTRADIOL | 34.71 | | 10.00 - 52.00 | pg/mL |
| **25 - HYDROXYVITAMIN D, SERUM** | RESULT PENDING | | | |
| **ASPARTATE AMINOTRANSFERASE, SERUM** | | | | |
| ASPARTATE AMINOTRANSFERASE (AST/SGOT) | 24 | | 15 - 37 | U/L |

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| **ALANINE AMINOTRANSFERASE, SERUM** | | | | |
| ALANINE AMINOTRANSFERASE (ALT/SGPT) | 53 | | 30 - 65 | U/L |
| **BLOOD UREA NITROGEN, SERUM** | | | | |
| BLOOD UREA NITROGEN | | L 5 | 7 - 18 | mg/dL |
| **CREATININE, SERUM** | | | | |
| CREATININE | 0.9 | | 0.6 - 1.3 | mg/dL |

***COMMENT:***

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***NOTE : RECHECKED FOR SERUM LH.***
***PLEASE CORRELATE CLINICALLY.***

**********

***NOTE: 2.5, 3.0 AND 3.5 HRS PLASMA AND URINE SPECIMENS NOT RECEIVED.***

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***NOTE FOR INSULIN LIKE GROWTH FACTOR-1: IN RANGE AND OUT OF RANGE COLUMNS ARE NOT APPLICABLE FOR THIS PARTICULAR TEST, SO PLEASE REFER THE AGE BASED REFERENCE RANGES GIVEN BELOW***

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TESTOSTERONE, TOTAL, SERUM

Testosterone is the major androgen in males and is produced by the Leydig cells of the testes. Testosterone circulates primarily as a protein bound steroid and strongly binds to plasma proteins such as sex hormone binding globulin (SHBG) or testosterone-estradiol-binding globulin (TeBG). Testosterone also binds with low affinity to cortisol binding globulin (CBG) and albumin. Less than 2.5% of testosterone circulates unbound to plasma proteins.

In adult males, testosterone levels show a diurnal variation with the highest levels detected in the early morning and the lowest level in the evening. Levels also increase after exercise and gradually decrease with advancing age. In adult females, testosterone levels are much lower than adult males (usually 5-10% that of males levels). The major sources of testosterone in females are the ovaries, the adrenal glands and the peripheral conversion of precursors, specifically androstenedione to testosterone.

Clinical entities in which testosterone is increased include gonadal & adrenal tumors, adrenal hyperplasia and polycystic ovaries (Stein-Leventhal syndrome). The clinical manifestations of excess testosterone include infertility, hirsutism, ammenorhea and obesity. Decreased levels of testosterone are associated with conditions such as hypogonadism, hypopitutarism, orchiectomy, estrogen therapy, testicular failure, hyperprolactinemia, some cases of Klinefelter's syndrome, some types of liver and kidney diseases and critical illness. Clinical applications of serum testosterone tests in pediatrics include detection precocious puberty, hypogonadism in adolescent boys, pituitary or hypothalamic disease, where both testosterone and gonadotropin concentrations are low and virilization in girls.

Heterophilic antibodies in human serum can react with reagent immunogobulins, interfering with in-vitro immunoassays. Patients routinely exposed to animals or animal serum products can be prone to this interference and anomalous values may be observed. "In range" and "Out of range" flagging is applicable for testosterone assay, only if age of the patient is mentioned.

Test method: Chemiluminescence

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.

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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.

REFERENCE RANGE FOR GROWTH HORMONE STIMULATION TEST.
Post- stimulation normal 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 GH deficiency. GH levels between normal and deficient states are considered as Indeterminate.

INSULIN, SERUM

Insulin is protein hormone that is synthesized, stored and secreted by the beta cells located in the islets of Langerhans in the pancreas. Insulin is responsible for regulating glucose concentrations in the blood. Initially in the beta cells, insulin exists as a large molecule called preproinsulin. Preproinsulin is a single-chain precursor consisting of 110 amino acids. A chain of 24 amino acids of preproinsuiln is cleaved forming proinsulin, the precursor of insulin and C-peptide.

Proinsulin consists of two amino acids chains of insulin connected by disulfide bonds and a connective peptide, called C-peptide. The alpha (A) chain of insulin consists of 21 amino acids, the beta (B) chain of insulin consists of 30 amino acids, and C-peptide consists of 31 amino acids. Proinsulin is stored in the secretory granules in the Golgi apparatus of the beta cells until proinsulin undergoes proteolysis to form insulin and C-peptide. At the cell membrane, insulin and C-peptide are released into the portal circulation in equimolar amounts.

Insulin is released in response to the presence of glucose in the blood typically after the ingestion of a meal. A normal healthy individual produces 40 to 50 units of insulin each day. The half-life of insulin in serum or plasma is 5 to 10 minutes. Approximately 50% of the insulin released into the portal circulation is cleared by the liver. Insulin binds to receptor cells located on cell membranes of target tissues. The target tissues are primarily liver, fat, and muscle tissue. Insulin modulates glucose concentration in the blood by stimulating glucose uptake (Glycogenesis) in muscle, Lipogenesis in Adipose tissue and inhibiting Glycogenolysis and Gluconeogenesis in the liver.

If insulin production is not stimulated, blood glucose levels will not be lowered and hyperglycemia results. Fasting hyperglycemia supports the diagnosis of diabetes mellitus. There are two types of diabetes mellitus: type I or insulin-dependent diabetes mellitus (IDDM) and type II or non-insulin-dependent diabetes mellitus (NIDDM). Insulin therapy is used for insulin-dependent diabetes mellitus (IDDM) patients and many non-insulin-dependent diabetes mellitus (NIDDM) patients. In type I diabetes (IDDM) there is a deficiency of insulin. This can be the result of autoimmune destruction of the beta cells or the presence of autoantibodies to insulin. Many factors can play a role in the development of Type II diabetes (NIDDM). Type II diabetes (NIDDM) can

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**PRELIMINARY**

TEST REPORT STATUS RESULTS

IN RANGE OUT OF RANGE REFERENCE RANGE UNITS

result if there is a decreased biological response to circulating insulin (insulin resistance) or if there is decreased or diminished insulin secretion due to beta cell failure.

Insulin levels are not typically used in the diagnosis or management of diabetic patients. Insulin levels can be useful in evaluating patients with fasting hypoglycemia, in determining insulin resistance in the general population, and in assessing abnormalities in beta cell secretory function. Insulin levels are used in studying the pathophysiology of diabetes.

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

ESTRADIOL, SERUM

Estradiol plays an essential role throughout the human menstrual cycle. During the early follicular phase, the estradiol level is relatively constant and low. By day seven, the dominant follicle is established and the estradiol level rises significantly. The elevated estradiol level suppresses the FSH level by negative feedback on the hypothalamus and pituitary gland and triggers a rapid rise of LH. The estradiol level falls significantly as LH reaches its peak. Normally, ovulation occurs 10 to 12 hours after the LH peak and 24 to 36 hours after the estradiol peak. During the luteal phase the estradiol level increases, achieving a maximum level about 8 days after ovulation. The elevated estradiol level is involved in the regression of the corpus luteum. Unless fertilization of the ovum takes places, the estradiol level decreases, signaling the start of a new cycle.

In normal, non-pregnant females, estradiol is secreted mainly by the combined function of the theca and granulosa cells of the developing follicle and the corpus luteum. During pregnancy, the placenta is a source of estradiol secretion. Estradiol enters the blood stream where 1 to 3% is non-protein bound, 40% is bound to sex-hormone binding globulin (SHBG) and the remainder is bound to albumin. The primary function of estradiol is to stimulate growth of the female sex organs and development of secondary sexual characteristics.

Elevated estradiol levels in females may also result from primary or secondary ovarian hyper function. Very high estradiol levels are found during the induction of ovulation for assisted reproduction therapy or in pregnancy. Measuring the circulating levels of estradiol is important for assessing ovarian function and monitoring follicular development for assisted reproduction protocols.

Decreased estradiol levels in females may result from either the lack of ovarian synthesis (primary ovarian hypo function and menopause) or a lesion in the hypothalamus-pituitary axis (Secondary ovarian hypo function).

Estradiol levels are normally low in males. Elevated estradiol levels in males may be due to increased aromatization of androgens, resulting in gynecomastia.

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.

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.

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IN RANGE OUT OF RANGE REFERENCE RANGE UNITS

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.

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Test method: Chemiluminescence.

CREATININE, SERUM

Creatinine estimation is done to assess kidney function. It is not dependent on dietary factors. Normal values are obtained in kidney diseases, excepting in advanced Renal Failure and therefore its estimation is more valuable if coupled with clearance studies and other Renal Function tests.

Test Technique: Alkaline picrate-kinetic (Jaffe's Kinetic)

ALANINE AMINOTRANSFERASE, SERUM

ALT activity is predominantly associated with liver tissue followed by comparatively lower levels in heart, muscles & kidneys. Quantitation of ALT is useful in evaluating liver function.

Test Technique: UV with P5P

BLOOD UREA NITROGEN, SERUM

Urea Nitrogen- a metabolic product of protein metabolism- is affected by diet, systemic blood circulation, renal conditions as also post- renal conditions affecting renal function.

Test Technique: Urease

ASPARTATE AMINOTRANSFERASE, SERUM

Aspartate Aminotransferase (SGOT/ AST) is widely distributed in tissues with highest concentration in liver, heart and skeletal muscle. Acute destruction of these tissue results in release of sizeable amounts of AST in systemic circulation following myocardial infarction, serum AST level begin to rise within

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RESULTS

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**4- 6 hours after the onset of angina, reaching a peak in 24- 36 hours.**

Serum AST activity is also high in hepatitis and other forms of liver diseases associated with hepatocellular necrosis, infectious mononucleosis, muscular dystrophy, dermatomyositis and in other forms of muscle and liver injury.

Test Technique: UV with P5P

LACTATE DEHYDROGENASE, SERUM

Test Technique: Modification of Enzymatic Lactate - Pyruvate

HOMOCYSTEINE, SERUM/PLASMA

Homocysteine (HCY) is a naturally occurring amino acid that is formed from methionine as a product of numerous S-adenosylmethionine-dependent transmethylation reactions. Three enzymatic pathways that either convert HCY into cysteine or remethylate it back into methionine regulate the metabolism of HCY. Homocysteine readily forms disulfide bond and is present in plasma in three forms: free or unbound HCY (1 to 2%), Homocysteine-cysteine or Homocysteine dimers (10 to 20%) or protein bound (>80%). Total plasma Homocysteine (HCY) free and bound is commonly referred to as either Homocysteine or Homocyst (e) ine.

If one or more of the HCY metabolic pathways are inhibited due to enzymatic defects or vitamin deficiencies, HCY accumulates, causing an increased HCY level in plasma. Homocysteinuria is a rare group of genetic diseases where a deficiency in one of the HCY regulating enzymes (usually cystathionine B-synthetase) results in high plasma HCY levels and HCY excretion in urine. Individuals who are homozygous for these disorders can have plasma HCY levels exceeding 400 umol/L. Individuals who are heterozygous for one of the enzyme deficiencies will exhibit hyperhomocysteinemia and have HCY level between 20 to 40 umol/L. Homocysteinemia due to enzyme deficiency has an occurrence of 1 per 100 population. Deficiencies in folic acid, vitamin B6 or vitamin B12 can produce hyperhomocysteinemia. Other studies showed that chronic renal failure is also associated with elevated HCY levels.

A relationship between Homocysteinuria and the development of premature arteriosclerotic disease was first observed over 30 years ago. More recently, several clinical and epidemiological studies have indicated that even moderately elevated plasma HCY level is a predictor for cardiovascular disease

A dose dependent effect was noted between HCY level and risk. Homocysteine levels also have been found to correlate to the extent of atherosclerotic plaque in the aorta. Homocysteine can impair the ability to repair vascular endothelial cell injury and thus promote the development of atherosclerosis.

Patients taking methotrexate, nicotinic acid, Theophylline, nitrous oxide, S-adenosyl-methionine and L-dopa can have falsely elevated serum HCY levels.

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.

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 the 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.

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.

LABORATORY REPORT

SRL RANBAXY

CLIENT CODE : C000005460

CLIENT'S NAME AND ADDRESS :

OMKAR SURGICALS
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REFERRING DOCTOR

DRAWN 10/03/2008 08:00 RECEIVED 10/03/2008 08:54 REPORTED 10/03/2008 20:41

PATIENT NAME MAHENDRAKUMAR TRIVEDI

| ACCESSION NO. | AGE | SEX | DATE OF BIRTH | PATIENT ID |
|---|---|---|---|---|
| 0002HC026841 | 45 Years | Male | 10/02/1963 | |

CLINICAL INFORMATION

**PRELIMINARY** RESULTS

TEST REPORT STATUS

| | IN RANGE | OUT OF RANGE | REFERENCE RANGE | UNITS |
|---|---|---|---|---|

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.

LH, FSH AND PROLACTIN, SERUM

FSH and LH are Pituitary hormones, useful to distinguish Primary Gonadal Failure from secondary (Hypothalamic/ Pituitary) causes of Gonadal Failure, and Amenorrhea. High values of LH & FSH occur with Castration, Ovarian Failure and in Post Menopausal State. Excessive LH and FSH are found in Hypogonadism, Gonadal Failure, Testicular Feminization Syndrome & Menopause. LH and FSH is mainly useful in defining menstrual cycle phases in Infertility evaluation of women and Testicular Dysfunction in men. In Pituitary or Hypothalamic Failure both would be low but when one hormone is high & other one is low a Gonadotropin - producing Pituitary Tumor is likely. Elevated basal LH with High LH / FSH ratio in essentially nonovulatory adult female could indicate Stein- Leventhal Syndrome. Prolactin levels may be elevated in Pituitary tumors, Amenorrhea & Polycystic Ovarian Syndrome.

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.

THYROID PANEL BY CHEMILUMINESCENCE, SERUM

Primary malfunction of the thyroid gland may result in excessive (hyper) or below normal (hypo) release of T3 or T4. In addition, as TSH directly affects thyroid function, malfunction of the pituitary or the hypothalamus influences the thyroid gland activity. Disease in any portion of the thyroid-pituitary-hypothalamus system may influence the levels of T3 and T4 in the blood. In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels may be low. In addition, in the Euthyroid Sick Syndrome, multiple alterations in serum thyroid function test findings have been recognized in patients with a wide variety of nonthyroidal illness (NTI) without evidence of preexisting thyroid or hypothalamic-pituitary disease.
Thyroid Binding Globulin (TBG) concentrations remain relatively constant in healthy individuals. However, pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids are known to alter TBG levels and may cause false thyroid values for Total T3 and T4 tests.

Below mentioned are the guidlines for Pregnancy related reference ranges for Total T4, TSH & Total T3

| Levels in Pregnancy | TOTAL T4 (µg/dL) | TSH (µIU/mL) | TOTAL T3 (ng/dL) |
|---|---|---|---|
| First Trimester | 6.6 - 12.4 | 0.3 - 4.5 | 81 - 190 |

LABORATORY REPORT

CLIENT CODE : C000005460

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PATIENT NAME MAHENDRAKUMAR TRIVEDI

ACCESSION NO. 0002HC026841 AGE 45 Years SEX Male DATE OF BIRTH 10/02/1963 PATIENT ID

CLINICAL INFORMATION

**PRELIMINARY**

TEST REPORT STATUS RESULTS

| | IN RANGE | OUT OF RANGE | REFERENCE RANGE | UNITS |
|---|---|---|---|---|
| 2nd Trimester | 6.6 - 15.5 | 0.5 - 4.6 | 100 - 260 | |
| 3rd Trimester | 6.6 - 15.5 | 0.8 - 5.2 | 100 - 260 | |

Below mentioned are the guidelines for age related reference ranges for T3, T4 and TSH result

| T3 (ng/dL) | T4 (µg/dL) | TSH (µIU/mL) |
|---|---|---|
| Cord Blood: 30 - 70 | 1-3 day: 8.2 - 19.9 | Birth-4 Day: 1.0 - 38.9 |
| New Born: 75 - 260 | 1 Week: 6.0 - 15.9 | 2 - 20 Weeks: 1.7 - 9.1 |
| 1-5 Years: 100 - 260 | 1-12 Months: 6.1 - 14.9 | 20 Weeks-20 Years: 0.7 -6.4 |
| 5 - 10 Years: 90 - 240 | 1 - 3 Years: 6.8 - 13.5 | |
| 10 - 15 Years: 80 - 210 | 3 - 10 Years: 5.5 - 12.8 | |

Reference:

1. Burtis C.A., Ashwood E. R. Bruns D.E. Teitz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.
2. Gowenlock A.H. Varley's Practical Clinical Biochemistry, 6th Edition.
3. Behrman R.E. Kilegman R.M., Jenson H. B. Nelson Text Book of Pediatrics, 17th Edition

Test method: Chemiluminescence

INSULIN LIKE GROWTH FACTOR - I, SERUM

Insulin like growth factor-I is a single polypeptide chain with three intra-molecule disulphide bonds. It is structurally homologous to IGF-II and insulin. Production of IGF-I, also known as Somatomedin C, is growth hormone (GH) dependent. The liver is the main source of circulating IGF-I and its synthesis is regulated by GH.

In humans serum IGF-1 level are low during foetal and neonatal life, increase gradually during puberty, peaking at tanner stages 3-4, and show a decline similar to GH with ageing. In females at each age, average IGF-1 plasma levels are slightly higher than in males. IGF measurement has been advocated as a screening and management tool in growth hormone deficient children. Its use in diagnosis along with growth hormone measurements or as a tool to assess a child's response to administered growth hormone has led to prominent place in the endocrine laboratory, particularly when dealing with growth disorders. A second major use of IGF-1 measurement is in the diagnosis and treatment of acromegaly. IGF-1 levels may be helpful to assess the results of bromocryptine treatment of acromegaly.

Expected normal values in ng/mL

| Ages (years) | Range |
|---|---|
| 0 - 1 | 12.5 - 116.0 |
| 1 - 2 | 16.4 - 137.2 |
| 2 - 3 | 18.4 - 149.4 |
| 3 - 4 | 23.7 - 197.1 |
| 4 - 5 | 30.1 - 252.2 |
| 5 - 6 | 38.4 - 313.8 |

LABORATORY REPORT

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PATIENT NAME MAHENDRAKUMAR TRIVEDI

ACCESSION NO. 0002HC026841 AGE 45 Years SEX Male DATE OF BIRTH 10/02/1963 PATIENT ID

CLINICAL INFORMATION

TEST REPORT STATUS **PRELIMINARY** RESULTS

| | | IN RANGE | OUT OF RANGE | REFERENCE RANGE | UNITS |
|---|---|---|---|---|---|
| 6 - 7 | 48.5 - 380.5 | | | | |
| 7 - 8 | 60.6 - 450.2 | | | | |
| 8 - 9 | 69.6 - 488.6 | | | | |
| 9 - 10 | 78.1 - 517.0 | | | | |
| 10 - 11 | 91.3 - 575.4 | | | | |
| 11 - 12 | 105.0 - 629.3 | | | | |
| 12 - 13 | 118.4 - 677.5 | | | | |
| 13 - 14 | 131.2 - 718.9 | | | | |
| 14 - 16 | 148.4 - 767.2 | | | | |
| 16 - 17 | 166.5 - 805.4 | | | | |
| 17 - 20 | 181.2 - 816.2 | | | | |
| 20 - 25 | 179.9 - 777.6 | | | | |
| 25 - 30 | 155.3 - 667.1 | | | | |
| 30 - 35 | 122.9 - 570.1 | | | | |
| 35 - 40 | 98.0 - 499.1 | | | | |
| 40 - 45 | 83.7 - 454.7 | | | | |
| 45 - 50 | 75.6 - 430.1 | | | | |
| 50 - 55 | 68.6 - 416.5 | | | | |
| 55 - 60 | 61.0 - 404.2 | | | | |
| 60 - 65 | 54.0 - 384.7 | | | | |
| 65 - 70 | 45.8 - 352.7 | | | | |
| 70 - 75 | 39.7 - 331.5 | | | | |
| 75 - 80 | 32.9 - 299.2 | | | | |
| 80 - 85 | 32.5 - 251.8 | | | | |
| 85 - 95 | 17.6 - 219.7 | | | | |

LABORATORY REPORT

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| DRAWN | RECEIVED | REPORTED |
|---|---|---|
| 10/03/2008 08:00 | 10/03/2008 08:54 | 10/03/2008 20:41 |

**PATIENT NAME** MAHENDRAKUMAR TRIVEDI

| ACCESSION NO. | AGE | SEX | DATE OF BIRTH | PATIENT ID |
|---|---|---|---|---|
| 0002HC026841 | 45 Years | Male | 10/02/1963 | |

CLINICAL INFORMATION

**PRELIMINARY**

TEST REPORT STATUS

RESULTS

| | IN RANGE | OUT OF RANGE | REFERENCE RANGE | UNITS |
|---|---|---|---|---|
| **Test method: Enzyme Immunoassay** | | | | |

**** End Of Report ****

Barnali Das

**Dr. Barnali Das, MD, DNB**

**Biochemist - Clinical Chem, RIA & Chemiluminescence**

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REFERRING DOCTOR

DRAWN 11/03/2008 00:00 RECEIVED 11/03/2008 18:26 REPORTED 21/03/2008 17:49

PATIENT NAME MAHENDRAKUMAR TRIVEDI

ACCESSION NO. 0002HC031491 AGE 45 Years SEX Male DATE OF BIRTH PATIENT ID

CLINICAL INFORMATION

TEST REPORT STATUS **FINAL**

| | RESULTS | | | |
|---|---|---|---|---|
| | IN RANGE | OUT OF RANGE | REFERENCE RANGE | UNITS |
| **ANTIDIURETIC HORMONE** | | | | |
| ANTIDIURETIC HORMONE | 1.06 | | 0.00 - 13.00 | pg/mL |

**** End Of Report ****

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