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NAME	**: Mrs. REKHA**		
AGE/ GENDER	: 38 YRS/FEMALE	**PATIENT ID**	: 1679035
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012411220027**
REFERRED BY	:	**REGISTRATION DATE**	: 22/Nov/2024 10:41 AM
BARCODE NO.	: 01521251	**COLLECTION DATE**	: 22/Nov/2024 10:44AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 22/Nov/2024 12:06PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA *by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)*	86.66	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. 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 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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Test Name	Value	Unit	Biological Reference interval
CALCIUM			
CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	9.5	mg/dL	8.50 - 10.60

INTERPRETATION:-

1.Serum calcium (total) estimation is used for the diagnosis and monitoring of a wide range of disorders including diseases of bone, kidney, parathyroid gland, or gastrointestinal tract.
2. Calcium levels may also reflect abnormal vitamin D or protein levels.
3.The calcium content of an adult is somewhat over 1 kg (about 2% of the body weight).Of this, 99% is present as calcium hydroxyapatite in bones and <1% is present in the extra-osseous intracellular space or extracellular space (ECS).
4. In serum, calcium is bound to a considerable extent to proteins (approximately 40%), 10% is in the form of inorganic complexes, and 50% is present as free or ionized calcium.
NOTE:-Calcium ions affect the contractility of the heart and the skeletal musculature, and are essential for the function of the nervous system. In addition, calcium ions play an important role in blood clotting and bone mineralization.

HYPOCALCEMIA (LOW CALCIUM LEVELS) CAUSES :-

1.Due to the absence or impaired function of the parathyroid glands or impaired vitamin-D synthesis.
2. Chronic renal failure is also frequently associated with hypocalcemia due to decreased vitamin-D synthesis as well as hyperphosphatemia and skeletal resistance to the action of parathyroid hormone (PTH).
3.***NOTE:-*** A characteristic symptom of hypocalcemia is latent or manifest tetany and osteomalacia.

HYPERCALCEMIA (INCREASE CALCIUM LEVELS) CAUSES:-

1.Increased mobilization of calcium from the skeletal system or increased intestinal absorption.
2.Primary hyperparathyroidism (pHPT)
3.Bone metastasis of carcinoma of the breast, prostate, thyroid gland, or lung.
NOTE:-Severe hypercalcemia may result in cardiac arrhythmia.

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Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

PROLACTIN

PROLACTIN: SERUM	12.34	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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IMMUNOPATHOLOGY/SEROLOGY

TORCH COMPLETE ANTIBODIES PANEL IgG AND IgM: 8

Test Name	Value	Unit	Biological Reference interval
TORCH ANTIBODIES EVALUATION IgG			
TOXOPLASMA ANTIBODIES IgG *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	0.584	IU/mL	NEGATIVE: < 2.0 POSITIVE: > 2.0
RUBELLA ANTIBODIES IgG *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**12.954^{H}**	IU/mL	NEGATIVE: < 2.0 POSITIVE: > 2.0
CYTOMEGALOVIRUS ANTIBODIES IgG *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**2.236^{H}**	IU/mL	NEGATIVE: < 2.0 POSITIVE: > 2.0
HERPES SIMPLEX VIRUS 1+2 ANTIBODIES IgG *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**4.56^{H}**	IU/mL	NEGATIVE: < 2.0 POSITIVE: > 2.0
TORCH ANTIBODIES EVALUATION IgM			
TOXOPLASMA ANTIBODIES IgM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	0.478	IU/mL	NEGATIVE: < 2.0 EQUIVOCAL: 2.0 - 2.60 POSITIVE: > 2.60
RUBELLA ANTIBODIES IgM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	1.635	IU/mL	NEGATIVE: < 2.0 EQUIVOCAL: 2.0 - 3.0 POSITIVE: > 3.0
CYTOMEGALOVIRUS ANTIBODIES IgM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	0.546	IU/mL	NEGATIVE: < 2.0 EQUIVOCAL: 2.0 - 4.2 POSITIVE: > 4.20
HERPES SIMPLEX VIRUS 1+2 ANTIBODIES IgM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	0.41	IU/mL	NEGATIVE: < 2.0 EQUIVOVAL: 2.0 - 4.0 POSITIVE: > 4.0

INTERPRETATION:

TOXOPLASMA:

1. Toxoplasma gondii is a ubiquitous intracellular parasite casuing serious infections in humans and domestic animals. Toxoplasma infection is asymptomatic in vast majority of immunocompetent individuals and is different from toxoplasmosis, the clinical or pathological disease. Latent (chronic infection) ensues in all infected people after resolution of acute phase, due to asymptomatic persistence of parasite. Reactivation of latent infection is usually seen in severely immunocompromised individuals.
2. Acquired Toxoplasmosis is usually asymptomatic and benign in pregnant women. However, the infection acquires a special significance as the parasite may enter the foetal circulation by transplacental route and casuse congenital toxoplasmosis. The risk and severity of congenital toxoplasmosis is greatest when acquired during first 3 months of pregnancy. The consequences of congenital toxoplasmosis range from

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Test Name	Value	Unit	Biological Reference interval

spontaneous abortion and prematurity to generalized and neurological symtoms.

CLINICAL UTILITY OF TOXOPLASMA:

1. Toxoplasma specific IgM develops 2 – 4 weeks after the onset of clinical signs and gradually declines hereafter, disappearing in 3 – 9 months. Therefore, the presence Of IgM and IgA in the absence og IgG or in the presence of low IgG levels is a strong evidence of ACUTE TOXOPLASMOSIS. Conversely, the presence of IgM in the presence of decreasing or constant IgG levels indicates subacute infection.
2. Specific IgG antibodies to Toxoplasma rise gradually and peak 2 – 5 months after the onset of clinical signs. Therefore, the presence of IgG is usefull in distinguishing subjects who have acquired the disease from those who have not. Increased level of toxoplasma specific IgG suggests reactivation of disease. IgG may be falsely negative in immunocompromised patients.
3. Accurate dating of the duration of maternal toxoplasmosis is required in order to assess the risk of subsequent congenital infection. However, positive IgM results are not easy to interpret, because specific IgM has a tendency to persist, even at high levels, after primary infection.

NOTE:

1. FALSE-POSITIVE POSITIVE IgM may occur due to RHEUMATOID FACTOR AND ANTI-NUCLEUR ANTIBODIES.
2. IgG avidity testing is recommended to differentiate between primary infection, IgM persistence and reactivation. A positive IgM accompanied by low-avidity IgG is suggestive of a primary infection, whereas a high-avidity IgG indicates either IgM persistence or reactivation. A low avidity index may also be seen in a proportion of infected persons for month. Hence it is adviced to perform IgM testing initially to point to the need for IgG avidity to avoid misinterpretation of results.

RUBELLA:

Rubella virus, the only member of rubivirus genus, causes rubella (also known as german measles), an acute exanthematous infection of children and adults. The clinical illnss is characterized by rash, fever and lymphadenopathy and can resemble a mild case of measles. The virus also cause arthralgias and occasional encephalitis. Infection is particularly disastrous if contracted during the first 4 months of pregnancy. If not immunologically protected, women infected during pregnancy run a high risk of embryo-foetal damage. Congenital Rubella causes a wide range of severe defects in foetus, including cataract, deafness, hepatosplenomegaly, psychomotor retardation, bone alterations, cardiopathies, neuropathies and diabetes.

TEST UTILITY FOR RUBELLA:

1. IgM antibodies become detectable in a few days after the onset of signs and symptoms and reach peak level in 7 – 10 days. These antibodies persist, but rapidly diminishes in concentration over the next 4 – 5 weeks until the antibody is no longer clinically detectable. While the presence of IgM antibodies suggests current or recent infection, low levels of IgM antibodies may occasionally persist for more than 12 months post-infection or immunization. The presence of IgM antibodies in a new born indicates that the bay was infected during pregnancy because the mother IgM antibodies do not pass to the baby through umbilical cord.
2. Rubella IgG antibody can be formed following rubella infection or after rubella vaccination. A reactive result is consistent with immune status to rubella virus. The presence of IgG antibodies, but not IgM antibodies, in a newborn means that the mothers IgG antibodies have passed to the baby in utero and these antibodies may protect the infant from rubella infection during the initial six months of life.

LIMITATIONS OF RUBELLA:

1. Rubella IgM test results are intended as an aid to the diagnose of active or recent infection. They should however, be interpreted in conjugation with other clinical findings and diagnostic procedures
2. The antibody titre of a single serum specimen cannot be used to determine recent infection. Specimens obtained too early, or too late, during the course of infection, may not demonstrate detectable levels of IgM antibody. Samples collected too early may not have detectable levels of IgG. Paired samples (acute & convalescent) should be collected and tested concurrently to demonstrate seroconversation.
3. A positive Rubella IgM result may not always indicate a primary acute infection, as IgM has a tendency to persist, even at high levels, after primay

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Test Name	Value	Unit	Biological Reference interval

infection. FALSE POSITIVE RESULTS MAY ALSO OCCUR DUE TO RHEUMATOID FACTOR AND ANTI-NUCLEUR ANTIBODIES. Hence, IgG avidity testing is recommended to differentiate between primay infection, IgM persistence and reactivation. IgG antibody results should be interpreted in conjugation with clinical evaluation and the and the results of other diagnostic procedures.

CYTOMEGALOVIRUS:

1. Cytomegalovirus (CMV) is a member of the Herpesviridae family and is classified as human herpesvirus Type 5. CMV causes a number of protean disease syndromes in infants as well as adults. Infection is common and reaches most of the population, whereas associated disease is relatively rare. CMV is a recognized cause of mononucleosis and hepatitis amongst normal immune-competent individuals. But it is among the immunosuppresed (immature neonates) organ transplant recipients, AIDS patients) that CMV causes most significant disease, manifesting as hepatitis, retinitis, pneumonitis, encephalitis, colitis etc.
2. The risk of an infected pregnant woman transmitting CMV to the foetus is highest in 3rd trimester and during the birth process(Perinatal infection) .Intrauterine / congenital CMV infections, though less frequently seen than perinatal infections, are responsible for causing severe CMV disease that may be fatal. Such Intauterine/congenital CMV infections are usually seen in infants born to mothers suffering from a primary infection during pregnancy.

TEST UTILITY FOR CYTOMEGALO VIRUS:

1. CMV specific IgM develops a few weeks after acute infection followed by development of IgG about a week later. IgM levels usually increases for some weeks and then decrease slowly in 4 – 6 months. Occassionally, IgM may circulate for years.
2. A positive CMV IgM result may not always indicate a primary acute infection, as IgM has a tendency to persist, even at high levels, after primary infection.FALSE-POSITIVE IgM RESULTS MAY OCCUR DUE TO RHEUMATOID FACTOR AND ANTI-NUCLEAR ANTIBODIES. Hence, IgG avidity testing is recommended to differentiate between primary infection, IgM persistence and reactivation.
3. Avidity is defined as the functional binding strength of antibodies to multiple binding sites (epitopes) on the antigen. The test is based on the principle that antibodies formed in response to primary infection have relatively low avidity to the corresponding antigen. With time, broader antibody response develops with antibodies being formed to more epitopes on the antigen and with the corresponding increase in the antibody antigen avidity. Therefore, when a secondary antibody response occurs with reinfection, it stimulates clonal expansion of memory B cells to a much wider spectrum epitopes, producing antibodies of considerably greater avidity.
4. A positive IgM accompanied by low-avidity IgG is suggestive of a primary infection, whereas a high-avidity IgG indicated either IgM persistence or reactivation. A low avidity index may also be seen in a proportion of infected persons for months. Hence it is adviced to perform IgM testing initially to point to the need for IgG avidity to avoid misinterpretation of results.

LIMITATIONS OF CYTOMEGALO VIRUS TEST:

Samples which are strongly positive for the presence of anti-Varicella Zoster and Ebstein Barr IgM antibodies can give false positive results.Because of all the complications of serological diagnosis of congenital infection, virus isolation from Urine in the first week of life remains the best way to diagnose intrauterine involvement. Absence of CMV specific IgM does not exclude the possibility of CMV infection.About 10 – 30 % of infants may fail to develop CMV IgM antibody response despite congenital infection with CMV.

HERPES SIMPLEX VIRUS (HSV) 1 AND 2:

1. Herpes Simplex Virus (HSV) is a widespread human pathogen with a tendency to induce lifelong latency in the sensory nerve ganglia, following the primary infection. Recurrent HSV infections are common due to endogeneous reactivation of the virus. Precipitating factors for recurrence can include exposure to sunlight, fever , local trauma, trigeminal nerve manipulation, menstruation and emotional stress. HSV-1 and HSV-2 are 2 serologically distinguishable types. Hsv-1 is primarily transmitted by contact with oral secretions and is usually associated with oral infections and lesions above waist. HSV-2, on the other hand, is primarily transmitted by contact with genital secretions and is associated with genital infections and lesions below the waist. However the correlation between HSV type and location of the lesion is not absolute. Transmission can

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Test Name	Value	Unit	Biological Reference interval

occur from overtly infected persons as well as asymptomatic excretors. HSV is known to cause severe generalized and fatal infections in newborns and immunocompromised people.

2. Pregnant women who develop genital hrpes are two-three times more likely to have spontaneous abortions or deliver a premature infant that are pregnant non-infected women. Active virus excreation in genital secretionsof pregnant women may result in severe neonatal HSV infection that is associated with high morbidity and mortality rates if untreated.

TEST UTILITY OF HERPES SIMPLEX VIRUS I AND 2:

HSV specific IgM becomes detectable aftr about 1 week of infection. Presence of IgM usually indicates recent or active recurrent infection. Specific IgG generally appears 2-3 after primary infection, but may fall in titer after a few months. Sero-conversion of HSV-specific IgG from negative to positive also suggests recent or active recurrent infection. However some patients with recurring disease may not show an increase in titer. Detection of IgG allows assessment of patients immune status and provide serological evidence of prior exposure to HSV. *TESTING PAIRED SERA TO DEMONSTRATE SEROCONVERSION IS RECOMMENDED FOR ACCURATE DIAGNOSIS OF RECENT (PRIMARY OR RECURRENT) HSV INFECTION.*

LIMITATIONS OF HERPES SIMPLEX VIRUS 1 AND 2:

Due to high seroprevalence of various community-related infectious disease in the general Indian population, all results must be interpreted in context of the total clinical history and supplementary findings of other investigative procedure. Due to strong serological cross-reactivity between HSV-1 and HSV-2, antibodies produced in response to infection by one virus can cross react with other, through the response to the homologous, i.e, the infection virus is generally greater. For this reason, tsting paired acute/covalescent specimens is useful to show change in antbody activity. Patients with intermediate results should be tested with another sample taken 1-2 weeks after the first, if clinically indicated

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Test Name	Value	Unit	Biological Reference interval

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**23.514[L]**	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFFERED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

1.Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
3.Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).
4.Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:
1.Lack of sunshine exposure.
2.Inadequate intake, malabsorption (celiac disease)
3.Depressed Hepatic Vitamin D 25- hydroxylase activity
4.Secondary to advanced Liver disease
5.Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:
1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:*-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.*

*** End Of Report ***

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