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NAME	**: Dr. AASTHA DHAMIJA**		
AGE/ GENDER	: 32 YRS/Female	**PATIENT ID**	: 1597578
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012411160060**
REFERRED BY	: LOOMBA HOSPITAL (AMBALA CANTT)	**REGISTRATION DATE**	: 16/Nov/2024 05:11 PM
BARCODE NO.	: 01520936	**COLLECTION DATE**	: 16/Nov/2024 06:23PM
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Test Name	Value	Unit	Biological Reference interval

HAEMATOLOGY

PROTHROMBIN TIME STUDIES (PT/INR)

PT TEST (PATIENT) *by PHOTO OPTICAL CLOT DETECTION*	12	SECS	11.5 - 14.5
PT (CONTROL) *by PHOTO OPTICAL CLOT DETECTION*	12	SECS	
ISI *by PHOTO OPTICAL CLOT DETECTION*	1.1		
INTERNATIONAL NORMALISED RATIO (INR) *by PHOTO OPTICAL CLOT DETECTION*	1		0.80 - 1.20
PT INDEX *by PHOTO OPTICAL CLOT DETECTION*	100	%	

INTERPRETATION:-

1.INR is the parameter of choice in monitoring adequacy of oral anti-coagulant therapy. Appropiate therapeutic range varies with the disease and treatment intensity.
2. Prolonged INR suggests potential bleeding disorder /bleeding complications
3. Results should be clinically correlated.
4. *Test conducted on Citrated Plasma*

RECOMMENDED THERAPEUTIC RANGE FOR ORAL ANTI-COAGULANT THERAPY (INR)		
INDICATION		**INTERNATIONAL NORMALIZED RATIO (INR)**
Treatment of venous thrombosis	Low Intensity	2.0 - 3.0
Treatment of pulmonary embolism		
Prevention of systemic embolism in tissue heart valves		
Valvular heart disease		
Acute myocardial infarction		
Atrial fibrillation		
Bileaflet mechanical valve in aortic position		
Recurrent embolism	High Intensity	2.5 - 3.5
Mechanical heart valve		

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Test Name	Value	Unit	Biological Reference interval
Antiphospholipid antibodies^{+}			

COMMENTS:

The prothrombin time (PT) and its derived measures of prothrombin ratio (PR) and international normalized ratio (INR) are measures of the efficacy of the extrinsic pathway of coagulation. PT test reflects the adequacy of factors I (fibrinogen), II (prothrombin), V, VII, and X. It is used in conjunction with the activated partial thromboplastin time (aPTT) which measures the intrinsic pathway.

The common causes of prolonged prothrombin time are :

1.Oral Anticoagulant therapy.
2.Liver disease.
3.Vit K. deficiency.
4.Disseminated intra vascular coagulation.
5.Factor 5, 7 , 10 or Prothrombin dificiency

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Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	0.64	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.18	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.46	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	20.4	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	36.4	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.56	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	65.62	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM *by SZASZ, SPECTROPHTOMETRY*	8.61	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	7.07	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	4.25	gm/dL	3.50 - 5.50
GLOBULIN: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	2.82	gm/dL	2.30 - 3.50
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.51	RATIO	1.00 - 2.00

INTERPRETATION

NOTE:- **To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.**

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5

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Test Name	Value	Unit	Biological Reference interval
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)		

DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

PROGNOSTIC SIGNIFICANCE:

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6

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Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* **3rd GENERATION, ULTRASENSITIVE**	1.652	µIU/mL	0.35 - 5.50

INTERPRETATION:

AGE	REFFERENCE RANGE (µIU/mL)
0 – 5 DAYS	0.70 – 15.20
6 Days – 2 Months	0.70 – 11.00
3 – 11 Months	0.70 – 8.40
1 – 5 Years	0.70 – 7.00
6 – 10 Years	0.60 – 5.50
11 - 15	0.50 – 5.50
> 20 Years (Adults)	0.27 – 5.50
PREGNANCY	
1st Trimester	0.10 - 3.00
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

NOTE:-*TSH levels are subjected to circardian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50 %. Hence time of the day has influence on the measured serum TSH concentration.*

USE:- TSH controls biosynthesis and release of thyroid harmones T4 & T3. It is a sensitive measure of thyroid function, especially useful in early or subclinical hypothyroidism, before the patient develops any clinical findings or goitre or any other thyroid function abnormality.

INCREASED LEVELS:

1.Primary or untreated hypothyroidism, may vary from 3 times to more than 100 times normal depending on degree of hypofunction.
2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
3.Hashimotos thyroiditis.
4.DRUGS: Amphetamines, Iodine containing agents and dopamine antagonist.
5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge.

DECREASED LEVELS:

1.Toxic multi-nodular goitre & Thyroiditis.
2.Over replacement of thyroid harmone in treatment of hypothyroidism.
3.Autonomously functioning Thyroid adenoma
4.Secondary pituatary or hypothalmic hypothyroidism
5.Acute psychiatric illness
6.Severe dehydration.
7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

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Test Name	Value	Unit	Biological Reference interval

8.Pregnancy: 1st and 2nd Trimester

LIMITATIONS:

1.TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.

2.Autoimmune disorders may produce spurious results.

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Test Name	Value	Unit	Biological Reference interval

ESTRADIOL (E2)

ESTRADIOL (E2): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	34.414	pg/mL	FEMALE FOLLICULAR PHASE: 19.5 - 144.2 FEMALE MID CYCLE PHASE: 63.9 - 356.7 FEMALE PRE OVULATORY PHASE: 136.0 - 251.0 FEMALE LUTEAL PHASE: 55.8 - 214.2 POST MENOPAUSAL:< 50.0

INTEPRETATION:

OTHER MATERNAL FACTORS AND PREGNANCY	UNITS	RANGE
Hormonal Contraceptives	pg/mL	15.0 – 95.0
1st Trimester (0 – 12 Weeks)	pg/mL	38.0 – 3175.0
2nd Trimester (13 – 28 Weeks)	pg/mL	678.0 – 16633.0
3rd Trimester (29 – 40 Weeks)	pg/mL	43.0 – 33781.0
Post Menopausal	Pg/mL	< 50.0
MALES:	pg/mL	< 40.0

1. Estrogens are involved in development and maintenance of the female phenotype,germ cell maturation,and pregnancy.They also are important for many other,nongender-specific processes, including growth,nervous system maturation,bone metabolism/remodeling,and endothelial responsiveness.
2. E2 is produced primarily in ovaries and testes by aromatization of testosterone.
3. Small amounts are produced in the adrenal glands and some peripheral tissues,most notably fat.E2 levels in premenopausal women fluctuate during the menstrual cycle.
4. They are lowest during the early follicular phase. E2 levels then rise gradually until 2 to 3 days before ovulation, at which stage they start to increase much more rapidly and peak just before the ovulation-inducing luteinizing hormone (LH)/follicle stimulating hormone (FSH) surge at 5 to 10 times the early follicular levels.This is followed by a modest decline during the ovulatory phase.E2 levels then increase again gradually until the midpoint of the luteal phase and thereafter decline to trough,early follicular levels.

INDICATIONS FOR ASSAY: -

1. Evaluation of hypogonadism and oligo-amenorrhea in females.
2. Assessing ovarian status, including follicle development,for assisted reproduction protocols (eg, in vitro fertilization)
3. In conjunction with lutenizing hormone measurements,monitoring of estrogen replacement therapy in hypogonadal premenopausal women
4. Evaluation of feminization,including gynecomastia,in males.
5. Diagnosis of estrogen-producing neoplasms in males,and,to a lesser degree,females
6. As part of the diagnosis and work-up of precocious and delayed puberty in females,and,to a lesser degree, males
7. As part of the diagnosis and work-up of suspected disorders of sex steroid metabolism,eg:aromatase deficiency and 17 alpha-hydroxylase

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Test Name	Value	Unit	Biological Reference interval

deficiency
8. As an adjunct to clinical assessment,imaging studies and bone mineral density measurement in the fracture risk assessment of postmenopausal women,and,to a lesser degree,older men
9. Monitoring low-dose female hormone replacement therapy in post-menopausal women
10. Monitoring antiestrogen therapy (eg, aromatase inhibitor therapy).

CAUSES FOR INCREASED E2 LEVELS:

1. High androgen levels caused by tumors or androgen therapy (medical or sport performance enhancing),with secondary elevations in E1 and E2 due to aromatization
2. Obesity with increased tissue production of E1
3. Decreased E1 and E2 clearance in liver disease
4. Estrogen producing tumors
5. Estrogen Ingestion

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Test Name	Value	Unit	Biological Reference interval

PROGESTERONE

PROGESTERONE: SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	< 0.10	ng/mL	FEMALE FOLLICULAR PHASE: 0.10 - 1.50 FEMALE OVULATORY PHASE: 0.40 - 3.00 FEMALE LUTEAL PHASE: 1.20 - 18.80 POST MENOPAUSAL: < 1.40 MALES: < 2.80

INTERPRETATION:

EXPECTED VALUES OF PROGESTERONE DURING PREGNANCY	
	UNITS (ng/mL)
First trimester (0 - 12 Wweeks)	15.8 - 46.0
Second trimester (13 - 28 Wweeks)	15.6 - 74.0
Third trimester (29 - 40 Wweeks)	45.0 - 143.0
Post Menopausal	< 1.40

1. Progesterone is produced by the adrenal glands, corpus luteum, and placenta.
2. After ovulation, there is a significant rise in serum Progesterone levels as the corpus luteum begins To produce progesterone in increasing amounts. This causes changes in the uterus, preparing it for implantation of a fertilized egg. If implantation occurs, the trophoblast begins to secrete human chorionic gonadotropin, which maintains the corpus luteum and its secretion of progesterone. If there is no implantation, the corpus luteum degenerates and circulating progesterone levels decrease rapidly, reaching follicular phase levels about 4 days before the next menstrual period.

The test is indicated for:

1. Ascertaining whether ovulation occurred in a menstrual cycle
2. Evaluation of placental function in pregnancy
3. Workup of some patients with adrenal or testicular tumors

NOTE:

In patients receiving therapy with high biotin doses (ie, >5 mg/day), no specimen should be drawn until at least 8 hours after the last biotin administration.

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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

LIVER KIDNEY MICROSOMAL (LKM) - 1 ANTIBODY: ELISA

LIVER KIDNEY MICROSOMAL (LKM) ANTIBODY - ELISA : SERUM *by ELISA (ENZYME LINKED MMUNOSORBENT ASSAY)*	3.4	IU/mL	NEGATIVE: < 25.0 BORDERLINE:20.0 - 25.0 POSITIVE: > 25.0

INTERPRETATION:

1.Autoimmune hepatitis (AIH) is a distinct chronic inflammatory liver disease, characterized by the attack of the immune system directed against "self" antigens, especially those expressed in the liver 1, 2.
2.It occurs in both sexes and all age groups, however, women are more likely victims of AIH than men. In women, 70 % of diagnosed cases of AIH occur between the ages of 15 and 40.
3.Hepatomegaly and spleenomegaly are the most common pathological findings associated with AIH.
4.Abnormalities of the immune system that mark AIH include autoantibodies to liver antigens, hyper-gammaglobulinemia, and an increased CD4/CD8 ratio in peripheral blood and liver.
5.Liver-Kidney Microsomal (LKM1) antibodies can be induced not only by autoimmune mechanisms, but also by drugs such as tienic acid, dihydralazine, halothane, phenytoin, phenobarbital, carbamazepine and by Hepatitis C and D infections

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Test Name	Value	Unit	Biological Reference interval

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA *by ELISA (ENZYME LINKED IMMUNOASSAY)*	11.29	IU/mL	NEGATIVE: < 20.0 POSITIVE: > 20.0

INTERPRETATION:

1.Anti-transglutaminase antibodies (ATA) are autoantibodies against the transglutaminase protein.
2.Antibodies to tissue transglutaminas are found in patients with several conditions, including coeliac disease, juvenile diabetes, inflammatory bowel disease, and various forms ofarthritis.
3.In coeliac disease, ATA are involved in the destruction of the villous extracellular matrix and target the destruction of intestinal villous epithelial cells by killer cells.
4.Deposits of anti-tTG in the intestinal epithelium predict coeliac disease.
5.Celiac disease (gluten-sensitive enteropathy, celiac sprue) results from an immune-mediated inflammatory process following ingestion of wheat, rye, or barley proteins that occurs in genetically susceptible individuals. The inflammation in celiac disease occurs primarily in the mucosa of the small intestine, which leads to villous atrophy.

CLINICAL MANIFESTATIONS RELATED TO GASTROINTESTINAL TRACT:

1.Abdominal pain
2.Malabsorption
3.Diarrhea and Constipation.

CLINICAL MANIFESTATION OF CELIAC DISEASE NOT RESTRICTED TO GIT:

1.Failure to grow (delayed puberty and short stature)
2.Iron deficiency anemia
3.Recurrent fetal loss
4.Osteoporosis and chronic fatigue
5.Recurrent aphthous stomatitis (canker sores)
6.Dental enamel hypoplasia, and dermatitis herpetiformis.
7.Patients with celiac disease may also present with neuropsychiatric manifestations including ataxia and peripheral neuropathy, and are at increased risk for development of non-Hodgkin lymphoma.
8.The disease is also associated with other clinical disorders including thyroiditis, type I diabetes mellitus, Down syndrome, and IgA deficiency.

NOTE:

1.The finding of tissue transglutaminase (tTG)-IgA antibodies is specific for celiac disease and possibly for dermatitis herpetiformis. For individuals with moderately to strongly positive results, a diagnosis of celiac disease is likely and the patient should undergo biopsy to confirm the diagnosis.
2.If patients strictly adhere to a gluten-free diet, the unit value of IgA-anti-tTG should begin to decrease within 6 to 12 months of onset of dietary therapy.

CAUTION:

1.This test should not be solely relied upon to establish a diagnosis of celiac disease. It should be used to identify patients who have an increased probability of having celiac disease and in whom a small intestinal biopsy is recommended.
2.Affected individuals who have been on a gluten-free diet prior to testing may have a negative result.
3.For individuals who test negative, IgA deficiency should be considered. If total IgA is normal and tissue transglutaminase (tTG)-IgA is negative

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Test Name	Value	Unit	Biological Reference interval

there is a low probability of the patient having celiac disease and a biopsy may not be necessary.
4.If serology is negative or there is substantial clinical doubt remaining, then further investigation should be performed with endoscopy and bowel biopsy. This is especially important in patients with frank malabsorptive symptoms since many syndromes can mimic celiac disease. For the patient with frank malabsorptive symptoms, bowel biopsy should be performed regardless of serologic test results.
5.The antibody pattern in dermatitis herpetiformis may be more variable than in celiac disease; therefore, both endomysial and tTG antibody determinations are recommended to maximize the sensitivity of the serologic tests.

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NAME	**: Dr. AASTHA DHAMIJA**		
AGE/ GENDER	: 32 YRS/Female	**PATIENT ID**	: 1597578
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012411160060**
REFERRED BY	: LOOMBA HOSPITAL (AMBALA CANTT)	**REGISTRATION DATE**	: 16/Nov/2024 05:11 PM
BARCODE NO.	: 01520936	**COLLECTION DATE**	: 16/Nov/2024 06:23PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 17/Nov/2024 05:46PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

IMMUNOGLOBIN IgG

IMMUNOGLOBIN-G (IgG): SERUM *by NEPHLOMETRY*	13.5	g/L	7.0-16.0

INTERPRETATION:

1.Immunoglobulin is a humoral antibody consisting of two light and two heavy chains in the molecule.
2.Approximately 80% of serum immunoglobulins is IgG. Its major function is neutralization of toxin in tissues spaces.
3.Antibodies of the IgG class are produced in response to most bacteria and viruses.IgG is the only immunogloblin that can cross the placental barrier and provide passive immune protection for fetus and new born till about 6 month.
4.Increased levels may be seen in SLE, chronic liver diseases, infectious diseases and cystic fibrosis. Monoclonal IgG increases in IgG myeloma.
5.Decreased synthesis of IgG is found in congenital/ acquired immunodeficiencies and in selective subclass deficiency such as bruton type agammaglobinulinemia.
6.Decreased IgG concentrations are seen in protein-losing enteropathies, nephrotic syndrome and in skin burns.

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BARCODE NO.	: 01520936	**COLLECTION DATE**	: 16/Nov/2024 06:23PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 20/Nov/2024 05:36PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

SPECIAL INVESTIGATIONS

ANTI NUCLEAR ANTIBODY/FACTOR (ANA/ANF) - WITH REFLEX TO TITRES: IFA (HEP-2)

ANTI NUCLEAR ANTIBODY (ANA) - IFA, HEp2 *by IFA (IMMUNO FLUORESCENT ASSAY)*	NEGATIVE (-ve)		NEGATIVE (-ve)

INTERPRETATION:

1.Anti Nuclear antibody (ANA) in dilutions is recommended for all positive results and follow up

2.Immunofluorescence microscopy using human cellular extracts like HEp-2 cells is a sensitive test for detection of serum antibodies that react specifically with various cellular proteins and nucleic acids

3.Test conducted on Serum

INTERPRETATION GUIDELINES : (Sample screening Dilution - 1:100):

Negative : No Immunofluorescence

+ : Weak Positive (1:100)

++ : Moderate Positive (1:320)

+++ : Strong Positive (1:1000)

++++ : Very strong Positive (1:3200)

COMMENTS:

Anti Nuclear antibody (ANA / ANF) is a group of autoantibodies directed against constituents of cell nuclei including DNA, RNA & various nuclear proteins. These autoantibodies are found with high frequency in patients with connective tissue disorders specially SLE. Since positive ANA results have been reported in healthy individuals, these reactivities are not by themselves diagnostic but must be correlated with other laboratory and clinical findings.

PATTERN	DISEASE ASSOCIATION
NUCLEAR	
Homogenous	SLE & other connective tissue disorders, Drug induced SLE
Peripheral	SLE & other connective tissue disorders
Speckled Coarse	Mixed connective Tissue Disorders (MCTD), Scleroderma-Polymyositis Overlap Syndrome, Raynauds Phenomenon, Psoariasis, Sjogrens Syndrome, Systemic Sclerosis.

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Test Name	Value	Unit	Biological Reference interval
Speckled Fine	SLE,Sjogrens syndrome,Scleroderma,Myositis,MCTD		
NUCLEAR DOTS			
Few	Auto-immune & Viral disease- Primary Biliay Cirrhosis & Chronic Active Hepatitis, Rarely Collagen Vascular disease		
Multiple	Primary Biliary Cirrhosis (>30%)		
Centromere	CREST syndrome, Progresive Systemic Sclerosis		
NUCLEOLAR			
Homogeneous	Scleroderma, Myositis, Raynauds Phenomena, SLE & Rheumatoid arthiritis		
Clumpy	Systemic sclerosis & Scleroderma		
CYTOPLASMIC			
Mitochondrial	Primary Biliary Cirrhosis,Scleroderma & Overlap syndrome		
Ribosomal	SLE (10-20%)		

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Test Name	Value	Unit	Biological Reference interval

ANTI SMOOTH MUSCLE ANTIBODY (ASMA) - WITH REFLEX TO TITRES: IFA

ANTI SMOOTH MUSCLE ANTIBODY (ASMA) - IFA *by IFA (IMMUNO FLUORESCENT ASSAY)*	NEGATIVE (-ve)		NEGATIVE (-ve)

INTERPRETATION:

1.Smooth muscle autoantibodies (SMA) are found in approximately 3% of normal adult caucasians.
2.High titres (>=1:160) of SMA are found in approximately 97% of patients with autoimmune chronic active hepatitis. SMA are found less frequently in uveitis, drug induced hepatitis, alcoholic liver disease, primary pulmonary hypertension and transiently in acute hepatitis and other viral infections including infectious mononucleosis.
3.Low titer antibodies may be found in the sera of patients with viral infections, malignancies and in the normal population.
4.The presence of SMA is not predictive of the development of liver disease.
5.The absence of ASMA indicates non autoimmune forms of chronic hepatitis.

*** End Of Report ***

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