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NAME	**: Mr. HARISH AHUJA**		
AGE/ GENDER	: 48 YRS/MALE	**PATIENT ID**	: 1792613
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012503150058**
REFERRED BY	: FORTIS HOSPITAL (MOHALI)	**REGISTRATION DATE**	: 15/Mar/2025 05:24 PM
BARCODE NO.	: 01527147	**COLLECTION DATE**	: 15/Mar/2025 05:26PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 15/Mar/2025 06:42PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	5.2	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	102.54	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):		
REFERENCE GROUP	**GLYCOSYLATED HEMOGLOGIB (HBAIC) in %**	
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.4	
Diagnosing Diabetes	>= 6.5	
Therapeutic goals for glycemic control	**Age > 19 Years**	
	Goals of Therapy:	< 7.0
	Actions Suggested:	>8.0
	Age < 19 Years	
	Goal of therapy:	<7.5

COMMENTS:

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients.

2.Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAIc. Converse is true for a diabetic previously under good control but now poorly controlled.

3.Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropiate.

4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications

5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution , given the pathological processes including anemia,increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.

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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIVER FUNCTION TEST (COMPLETE)

Test Name	Value	Unit	Biological Reference interval
BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	0.83	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.17	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.66	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	21.6	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	28.2	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.77	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	73.71	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM *by SZASZ, SPECTROPHTOMETRY*	17.52	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	**8.03^{H}**	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	4.24	gm/dL	3.50 - 5.50
GLOBULIN: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**3.79^{H}**	gm/dL	2.30 - 3.50
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.12	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
CREATININE			
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETRY*	1.05	mg/dL	0.40 - 1.40

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Test Name	Value	Unit	Biological Reference interval

CALCIUM

CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	9.73	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

PHOSPHOROUS

PHOSPHOROUS: SERUM *by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY*	2.72	mg/dL	2.5 - 4.5

INTERPREATION:-

1.Eighty-eight percent of the phosphorus contained in the body is localized in bone in the form of hydroxyapatite. The remainder is involved in intermediary carbohydrate metabolism and in physiologically important substances such as phospholipids, nucleic acids, and adenosine triphosphate (ATP).
2.Phosphorus occurs in blood in the form of inorganic phosphate and organically bound phosphoric acid. The small amount of extracellular organic phosphorus is found exclusively in the form of phospholipids.
3.Serum phosphate concentrations are dependent on meals and variation in the secretion of hormones such as parathyroid hormone (PTH) and may vary widely.

DECREASED (HYPOPHOSPHATEMIA):-

1.Shift of phosphate from extracellular to intracellular.
2.Renal phosphate wasting.
3.Loss from the gastrointestinal tract.
4.Loss from intracellular stores.

INCREASED (HYPERPHOPHATEMIA):-

1.Inability of the kidneys to excrete phosphate.
2.Increased intake or a shift of phosphate from the tissues into the extracellular fluid.

SIGNIFICANCE:-

1.Phosphate levels may be used in the diagnosis and management of a variety of disorders including bone, parathyroid and renal disease.
2.Hypophosphatemia is relatively common in hospitalized patients. Levels less than 1.5 mg/dL may result in muscle weakness, hemolysis of red cells, coma, and bone deformity and impaired bone growth.
3.The most acute problem associated with rapid elevations of serum phosphate levels is hypocalcemia with tetany, seizures, and hypotension. Soft tissue calcification is also an important long-term effect of high phosphorus levels.
4.Phosphorus levels less than 1.0 mg/dL are potentially life-threatening and are considered a critical value.

NOTE: Phosphorus has a very strong biphasic circadian rhythm. Values are lowest in the morning, peak first in the late afternoon and peak again in the late evening. The second peak is quite elevated and results may be outside the reference range

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Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.95	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	6.41	µgm/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	1.36	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

TSH levels are subject to circadian 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 concentrations. TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4)and triiodothyronine (T3).Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction(hyperthyroidism) of T4 and/or T3.

CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

LIMITATIONS:-

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG),and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin , salicylates).
3. Serum T4 levels in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.
4. TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothyroidism , pregnancy , phenytoin therapy.

TRIIODOTHYRONINE (T3)		THYROXINE (T4)		THYROID STIMULATING HORMONE (TSH)	
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range (µIU/mL)
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 – 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 – 17.04	3 Days – 6 Months	0.70 - 8.40
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 – 16.16	6 – 12 Months	0.70 - 7.00

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Test Name			Value	Unit	Biological Reference interval
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20	11 – 19 Years	0.50 – 5.50
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35– 5.50
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (µIU/mL)					
1st Trimester			0.10 – 2.50		
2nd Trimester			0.20 – 3.00		
3rd Trimester			0.30 – 4.10		

INCREASED TSH LEVELS:

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

DECREASED TSH LEVELS:

1.Toxic multi-nodular goiter & Thyroiditis.
2.Over replacement of thyroid hormone in treatment of hypothyroidism.
3.Autonomously functioning Thyroid adenoma
4.Secondary pituitary or hypothalamic hypothyroidism
5.Acute psychiatric illness
6.Severe dehydration.
7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
8.Pregnancy: 1st and 2nd Trimester

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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

HEPATITIS C VIRUS (HCV) ANTIBODY: TOTAL

HEPATITIS C ANTIBODY (HCV) TOTAL: SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.19	S/CO	NEGATIVE: < 1.00 POSITIVE: > 1.00
HEPATITIS C ANTIBODY (HCV) TOTAL RESULT *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	NON - REACTIVE		

INTERPRETATION:-

RESULT (INDEX)	REMARKS
< 1.00	NON - REACTIVE/NOT - DETECTED
> =1.00	REACTIVE/ASYMPTOMATIC/INFECTIVE STATE/CARRIER STATE.

Hepatitis C (HCV) is an RNA virus of Favivirus group transmitted via blood transfusions, transplantation, injection drug abusers, accidental needle punctures in healthcare workers, dialysis patients and rarely from mother to infant. 10 % of new cases show sexual transmission. As compared to HAV & HBV , chronic infection with HCV occurs in 85 % of infected individuals. In high risk population, the predictive value of Anti HCV for HCV infection is > 99% whereas in low risk populations it is only 25 %.

USES:

1. Indicator of past or present infection, but does not differentiate between Acute/ Chronic/Resolved Infection.
2. Routine screening of low and high prevelance population including blood donors.

NOTE:

1. False positive results are seen in Auto-immune disease, Rheumatoid Factor, HYpergammaglobulinemia, Paraproteinemia, Passive antibody transfer, Anti-idiotypes and Anti-superoxide dismutase.
2. False negative results are seen in early Acute infection, Immunosuppression and Immuno—incompetence.
3. *HCV-RNA PCR recommended in all reactive results to differentiate between past and present infection.*

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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)*	3.99	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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REFERRED BY	: FORTIS HOSPITAL (MOHALI)	**REGISTRATION DATE**	: 15/Mar/2025 05:24 PM
BARCODE NO.	: 01527147	**COLLECTION DATE**	: 15/Mar/2025 05:26PM
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

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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Test Name	Value	Unit	Biological Reference interval

HEPATITIS B SURFACE ANTIGEN (HBsAg) ULTRA

HEPATITIS B SURFACE ANTIGEN (HBsAg): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	0.26	S/CO	NEGATIVE: < 1.0 POSITIVE: > 1.0
HEPATITIS B SURFACE ANTIGEN (HBsAg) RESULT *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	NON REACTIVE		

INTERPRETATION:

RESULT IN INDEX VALUE	REMARKS
< 1.30	NEGATIVE (-ve)
>=1.30	POSITIVE (+ve)

Hepatitis B Virus (HBV) is a member of the Hepadna virus family causing infection of the liver with extremely variable clinical features. Hepatitis B is transmitted primarily by body fluids especially serum and also spread effectively sexually and from mother to baby. In most individuals HBV hepatitis is self limiting, but 1-2 % normal adolescent and adults develop Chronic Hepatitis. Frequency of chronic HBV infection is 5-10% in immunocompromised patients and 80 % neonates. The initial serological marker of acute infection is HBsAg which typically appears 2-3 months after infection and disappears 12-20 weeks after onset of symtoms. Persistence of HBsAg for more than 6 months indicates carrier state or Chronic Liver disease.

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Test Name	Value	Unit	Biological Reference interval

IMMUNOGLOBIN IgE

IMMUNOGLOBIN-E (IgE): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	5.23	IU/mL	0.00 - 100.00

INTERPRETATION:

COMMENTS:

1.IgE antibodies mediate allergic diseases by sensitizing mast cells and basophils to release histamine and other inflammatory mediators on exposure to allergens.
2.Total IgE is represents the sum of all the specific IgE, which inturn includes many groups of specific IgE & allergen specific IgE is just one such group amongst them.
3.Total IgE determination constitutes a screening method of atopic diseases, although within range values of total IgE do not exclude the existence of atopy and high values of total IgE are not pathognomonic of atopy by themselves.
4.Antigen-specific IgE is the next step in the in vitro identification of the responsible allergen. There are more than 400 characterized known allergens available for in vitro diagnostic tests and testing to be selected based on symptoms, clinical & environmental details.
5.In adults, Total IgE values between 100 to 1000 UI/ml may not correlate with allergen specific IgE, where the patients may be just sensitized to different allergen or often the cause for high IgE could be non-atopic.
6.Specific IgE results obtained with the different methods vary significantly, hence followup testing to be performed using one laboratory only.
7.The probability of finding an increased level of IgE in serum in a patient with allergic disease varies directly with the number of different allergens to which the patient is sensitized.
8.A normal level of IgE in serum does not eliminate the possibility of allergic disease; this occurs if there is sensitivity to a limited number of allergens and limited end organ involvement.

INCREASED:

1.Atopic/Non Atopic Allergy
2.Parasitic Infection.
3.IgE Myeloma
4.Allergic bronchopulmonary aspergillosis.
5.The rare hyper IgE syndrome.
6.Immunodeficiency States and Autoimmune states

USES:

1.Evaluation of children with strong family history of allergies and early clinical signs of disease ·
2.Evaluation of children and adults suspected of having allergic respiratory disease to establish the diagnosis and define the allergens
3.To confirm clinical expression of sensitivity to foods in patients with Anaphylactic sensitivity or with Asthma, Angioedema or Cutaneous disease
4.To evaluate sensitivity to insect venom allergens particularly as an aid in defining venom specificity in those cases in which skin tests are equivocal
5.To confirm the presence of IgE antibodies to certain occupational allergens

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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)*	**5.7[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.*

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Test Name	Value	Unit	Biological Reference interval

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	**89**^{L}	pg/mL	190.0 - 890.0

INTERPRETATION:-

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol Igestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.
NOTE:A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

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CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

Test Name	Value	Unit	Biological Reference interval
PHYSICAL EXAMINATION			
QUANTITY RECIEVED	10	ml	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
COLOUR	PALE YELLOW		PALE YELLOW
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
TRANSPARANCY	CLEAR		CLEAR
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SPECIFIC GRAVITY	>=1.030		1.002 - 1.030
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
CHEMICAL EXAMINATION			
REACTION	ACIDIC		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
PROTEIN	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SUGAR	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
pH	<=5.0		5.0 - 7.5
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
BILIRUBIN	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
NITRITE	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.			
UROBILINOGEN	Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
KETONE BODIES	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
BLOOD	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
ASCORBIC ACID	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
MICROSCOPIC EXAMINATION			
RED BLOOD CELLS (RBCs)	NEGATIVE (-ve)	/HPF	0 - 3

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Test Name	Value	Unit	Biological Reference interval
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS	2-3	/HPF	0 - 5
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
EPITHELIAL CELLS	0-2	/HPF	ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
CRYSTALS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
CASTS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
BACTERIA	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
OTHERS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
TRICHOMONAS VAGINALIS (PROTOZOA)	ABSENT		ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			

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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. Immunofluorescence microscopy using human cellular extracts like Hep-2 cells is sensitive for detection of serum antibodies that react specifically with various cellular proteins and nucleic acid.
2. Test conducted on serum
3. Patients are reported as per international consensus ANA Patterns (ICAP)

INTERNATIONAL GUIDELINES FOR GRADING	
GRADE	**REMARKS**
Negative (-ve)	No fluorescence
1+	Minimum fluorescence
2+	Mildly positive
3+	Significantly positive
4+	Strongly positive

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 (ICAP)	ICAP CODE	ANTIGEN ASSOCIATION	DISEASE ASSOCIATION
NUCLEAR PATTERNS			
Homogenous	AC-1	dsDNA, nucleosomes, histones	SLE, Drug-induced lupus, Juvenile idiopathic

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Test Name		Value	Unit	Biological Reference interval
			arthritis	
Speckled	AC-2,4,5	hnRNP, U1RNP, Sm, SS-A/Ro (Ro 80), SS-B/La, RNA polymerase III, Mi-2, Ku	MCTD, SLE, DM, SSc/PM overlap	
Dense fine speckled	AC-2	DFS70/LEDGF	Rare in SLE, Sjogren's syndrome, SSc	
Fine speckled	AC-4	SS-A/Ro (Ro 80), T1F1ß, SS-B/La,Mi-2,T1F1γ, Ku, RNA helicase A, replication protein A	Sjogren's syndrome, SLE, DM,SSc/PM overlap	
Large/Coarse speckled	AC-5	hnRNP, U1RNP, Sm, RNA polymerase III	MCTD, SLE, SSc	
Centromere	AC-3	CENP-A/B	Limited cutaneous SSc, PBC	
Discrete nuclear dots	AC-6,7			
Multiple nuclear dots	Ac-6	Sp-100, PML proteins, MJ/NXP-2	PBC, SARD, PM/DM	
Few nuclear dots	Ac-7	P80-coilin, SMN	Sjogren's syndrome, SLE, SSc, PM, asymptomatic individuals	
Nucleolar	AC-8,9,10			
Nucleolar homogenous	AC-8	PM/Scl-75, PM/Scl-100, Thi/To,B23/nucleophosmin, nucleolin, No55/SC65	SSc, SSc/PM overlap	
Nucleolar clumpy	AC-9	U3-smoRNP/fibrillarin	SSc	
Nucleolar punctate	Ac-10	RNA polymerase 1, hUBF/NOR-90	SSc, Sjogren's syndrome	
Nuclear envelope	AC-11,12			
Smooth nuclear envelope	AC-11	Lamin A,B,C or lamin associated proteins	SLE, Sjogren's syndrome, Seronegative arthritis	

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Test Name		Value	Unit	Biological Reference interval
Punctate nuclear envelope	AC-12	Nuclear pore complex proteins (gp210)	PBC	
Pleomorphic	AC-13,14			
PCNA-like	AC-13	PCNA	SLE, other conditions	
CENP-F like	AC-14	CENP-F	Cancer, other conditions	
CYTOPLASMIC PATTERNS				
Fibrillar	AC-15,16,17			
Linear/actin	AC-15	Actin, non-muscle myosin, MCTD	MCTD, Chronic active hepatitis,Liver cirrhosis, Myasthenia gravis,Crohn's disease, PBC, Long term hemodialysis, rare in SARD other than MCTD	
Filamentous/microtubules	AC-16	Vimentin, cytokeratins	Infections or inflammatory conditions, Long term hemodialysis, Alcoholic liver disease, SARD, Psoriasis, healthy controls	
Segmental	AC-17	Alpha-actin, vinuculin, tropomyosin	Myasthenia gravis, Crohn's disease, Ulcerative colitis	
Speckled	AC-18,19,20			
Discrete dots/GW body like	AC-18	SGW182, Su/Ago2,	PBC, SARD, neurological and autoimmune conditions	
Dense fine speckled	AC-19	PL-7, PL-12, ribosomal P proteins	Anti-synthetase syndrome, PM/DM, SLE, Juvenile SLE, Neuropsychiatric SLE	

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Test Name		Value	Unit	Biological Reference interval
Fine speckled	AC-20	Jo-1/histidyl-Trna synthetase		Anti-synthetase syndrome, PM/DM, limited SSc, Idiopathic pleural effusion
Reticular/AMA (Mitochondrial)	AC-21	PDC-E2/M2, BCOADC-E2 OGDC-E2, E1α subunit of PDC, E3BP/proteinX		Common in PBC, SSc, rare in other SARD
Polar/ Golgi like	AC-22	Giantin/macrogolgin, golgin-97, golgin-245		Rare in Sjogren's syndrome, SLE, RA, MCTD,GPA, Idiopathic cerebellar ataxia, Paraneoplastic cerebellar degeneration,viral infections
Rods and rings	AC-23	IMPDH2, others		HCV patients post IFN/Ribavirin therapy,rare in SLE, Hashimoto's and healthy controls
MITOTIC PATTERNS				
Centrosome	AC-24	Pericentrin, ninein, Cep250, Cep110		Rare in SSc, Raynaud's phenomenon, infections (viral and mycoplasma)
Spindle fibres	AC-25	HsEg5		Rare in Sjogren's syndrome, SLE, other SARD
NuMA like	AC-26	Centrophilin		Sjogren's syndrome, SLE, other
Intracellular bridges	AC-27	Aurora kinase B, CENP-E,MSA-2, KIF-14, MKLP-1		Rare in SSc, Raynaud's phenomenon, malignancy

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COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012503150058**
REFERRED BY	: FORTIS HOSPITAL (MOHALI)	**REGISTRATION DATE**	: 15/Mar/2025 05:24 PM
BARCODE NO.	: 01527147	**COLLECTION DATE**	: 15/Mar/2025 05:26PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 16/Mar/2025 02:54PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name		Value	Unit	Biological Reference interval
Mitotic chromosome coat	AC-28	Modified histone H3, MCA-1	Rare in Discoid lupus erythematous, Chronic lymphocytic leukemia, Sjogren's syndrome, and Polymyalgia rheumatica	

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MD (Pathology)
CEO & Consultant Pathologist

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REFERRED BY	: FORTIS HOSPITAL (MOHALI)	**REGISTRATION DATE**	: 15/Mar/2025 05:24 PM
BARCODE NO.	: 01527147	**COLLECTION DATE**	: 15/Mar/2025 05:26PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 19/Mar/2025 02:42PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

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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