



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961

✉ pkrjainhealthcare@gmail.com

NAME : Mrs. KULWINDER KAUR
AGE/ GENDER : 49 YRS/FEMALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12505242
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1647624
REG. NO./LAB NO. : 122410190006
REGISTRATION DATE : 19/Oct/2024 10:19 AM
COLLECTION DATE : 19/Oct/2024 03:15PM
REPORTING DATE : 19/Oct/2024 01:43PM

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIPID PROFILE : BASIC

CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	202.67 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	72.96	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM by SELECTIVE INHIBITION	70.11	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	130.97 ^H	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	132.56 ^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	14.59	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	491.3	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.89	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM	1.87	RATIO	LOW RISK: 0.50 - 3.0




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by CALCULATED, SPECTROPHOTOMETRY

MODERATE RISK: 3.10 - 6.0

HIGH RISK: > 6.0

TRIGLYCERIDES/HDL RATIO: SERUM

1.04^L

RATIO

3.00 - 5.00

by CALCULATED, SPECTROPHOTOMETRY

INTERPRETATION:

1. Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL.
5. Additional testing for Apolipoprotein B, hsCRP, Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement



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CALCIUM

CALCIUM: SERUM	10.56	mg/dL	8.50 - 10.60
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by ARSENAZO III, SPECTROPHOTOMETRY

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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PHOSPHOROUS

PHOSPHOROUS: SERUM 2.81 mg/dL 2.30 - 4.70

by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY

INTERPRETATION:-

- 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 (HYPERPHOSPHATEMIA):-


- 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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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.32	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	6.25	µgm/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	2.75	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRA SENSITIVE

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:-

- 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.
- 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).
- 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.
- 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	Reference Range (ng/mL)	Age	Reference 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




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Test Name		Value		Unit	Biological Reference interval
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 - 12 Months	0.70 - 7.00
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 (μ U/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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INTACT PARATHYROID HORMONE (PTH)

INTACT PARATHYROID HORMONE (PTH): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	43.8	pg/mL	9.5 - 75.0
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Intrepretation:-

Parathyroid hormone (PTH) is produced and secreted by the parathyroid glands, which are located along the posterior aspect of the thyroid gland. The serum calcium level regulates PTH secretion via negative feedback through the parathyroid calcium sensing receptor (CASR). Decreased calcium levels stimulate PTH release. Secreted PTH interacts with its specific type II G-protein receptor, causing rapid increases in renal tubular reabsorption of calcium and decreased phosphorus reabsorption. It also participates in long-term calciostatic functions by enhancing mobilization of calcium from bone and increasing renal synthesis of 1,25-dihydroxy vitamin D, which, in turn, increases intestinal calcium absorption.

The assay is useful for:

- Differential diagnosis of hypercalcemia
- Diagnosis of primary, secondary, and tertiary hyperparathyroidism
- Diagnosis of hypoparathyroidism
- Monitoring end-stage renal failure patients for possible renal osteodystrophy

Interpretation of results:

- An (appropriately) low PTH level and high phosphorus level in a hypercalcemic patient suggests that the hypercalcemia is not caused by PTH or PTH-like substances.
- An (appropriately) low PTH level with a low phosphorus level in a hypercalcemic patient suggests the diagnosis of paraneoplastic hypercalcemia.
- A low or normal PTH in a patient with hypocalcemia suggests hypoparathyroidism.

Low serum calcium and high PTH levels in a patient with normal renal function suggest resistance to PTH action (pseudohypoparathyroidism type 1a, 1b, 1c, or 2) or, very rarely, bio-ineffective PTH.

Elevated PTH value with a normal serum calcium in many cases in India is due to secondary hyperparathyroidism, primary cause being Vitamin D deficiency.



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IMMUNOPATHOLOGY/SEROLOGY

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA	10.34	IU/mL	NEGATIVE: < 20.0 POSITIVE: > 20.0
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by ELISA (ENZYME LINKED IMMUNOASSAY)

INTERPRETATION:

1. Anti-transglutaminase antibodies (ATA) are autoantibodies against the transglutaminase protein.
2. Antibodies to tissue transglutaminase are found in patients with several conditions, including coeliac disease, juvenile diabetes, inflammatory bowel disease, and various forms of arthritis.
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



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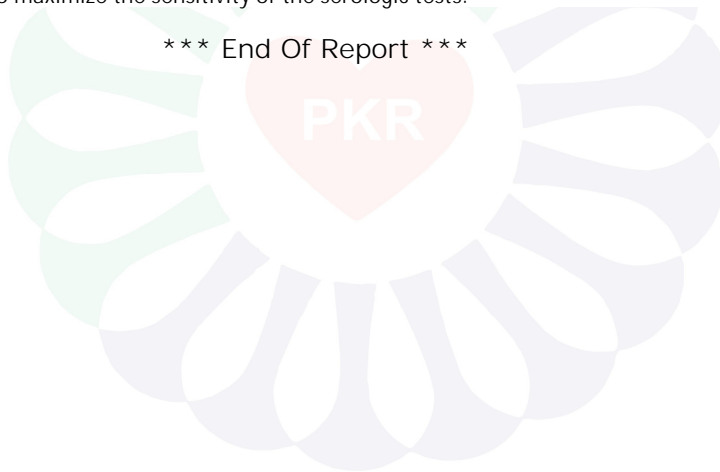
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increased probability of having celiac disease and in whom a small intestinal biopsy is recommended.
2.Affected individuals who have been on a gluten-free diet prior to testing may have a negative result.
3.For individuals who test negative, IgA deficiency should be considered. If total IgA is normal and tissue transglutaminase (tTG)-IgA is negative 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.

*** End Of Report ***




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