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NAME	**: Mrs. RAJWINDER KAUR**		
AGE/ GENDER	: 34 YRS/FEMALE	**PATIENT ID**	: 1558927
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012407240021**
REFERRED BY	:	**REGISTRATION DATE**	: 24/Jul/2024 09:34 AM
BARCODE NO.	: 01513722	**COLLECTION DATE**	: 24/Jul/2024 09:39AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 24/Jul/2024 10:34AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

CLINICAL CHEMISTRY/BIOCHEMISTRY

LIPID PROFILE : BASIC

Test Name	Value	Unit	Biological Reference interval
CHOLESTEROL TOTAL: SERUM *by CHOLESTEROL OXIDASE PAP*	195.03	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM *by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)*	143.84	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*	39.54	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	126.72	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***	**155.49^{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*	28.77	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	533.9	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM ***by CALCULATED, SPECTROPHOTOMETRY***	**4.93^{H}**	**RATIO**	**LOW RISK: 3.30 - 4.40** **AVERAGE RISK: 4.50 - 7.0** **MODERATE RISK: 7.10 - 11.0** **HIGH RISK: > 11.0**

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Test Name	Value	Unit	Biological Reference interval
LDL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**3.2^{H}**	**RATIO**	**LOW RISK: 0.50 - 3.0** **MODERATE RISK: 3.10 - 6.0** **HIGH RISK: > 6.0**
TRIGLYCERIDES/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	3.64	RATIO	3.00 - 5.00

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 atherogeniclipoproteins such as LDL , VLDL, IDL, Lpa, 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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IRON PROFILE

Test Name	Value	Unit	Biological Reference interval
IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	**25.19^{L}**	**µg/dL**	**50.0 - 170.0**
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM *by FERROZINE, SPECTROPHOTOMETERY*	**361.37^{H}**	**µg/dL**	**150.0 - 336.0**
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM *by SPECTROPHOTOMETERY*	386.56	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM *by CALCULATED, SPECTROPHOTOMETERY (FERENE)*	**6.52^{L}**	**%**	**15.0 - 50.0**
TRANSFERRIN: SERUM *by SPECTROPHOTOMETERY (FERENE)*	274.46	mg/dL	200.0 - 350.0

INTERPRETATION:-

VARIABLES	*ANEMIA OF CHRONIC DISEASE*	*IRON DEFICIENCY ANEMIA*	*THALASSEMIA α/β TRAIT*
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:
1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia,anemia of chronic disease and thalassemia syndromes.
2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BINDING CAPACITY (TIBC):
1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:
1.Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.

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Test Name	Value	Unit	Biological Reference interval

FERRITIN

FERRITIN: SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**4.35[L]**	**ng/mL**	**10.0 - 290.0**

INTERPRETATION:

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy.

DECREASED:

1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.
2. Hypothyroidism.
3. Vitamin-C deficiency.

INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

1. Hemochromatosis or hemosiderosis.
2. Wilson Disease.

INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

1. Transfusion overload
2. Excess dietary Iron
3. Porphyria Cutanea tada
4. Ineffective erythropoiesis.

INCREASED FERRITIN WITHOUT IRON OVERLOAD:

1. Liver disorders (NASH) or viral hepatitis (B/C).
2. Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
3. Leukaemia, hodgkin's disease.
4. Alcohol excess.
5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma, synthesis of ferritin by tumour cells.
6. Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can thererfore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive proteins to rule out any inflammatory conditions.

2. Patients with iron deficiency anaemia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.

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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)*	0.06	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	9.18	IU/mL	NEGATIVE: < 20.0 POSITIVE: > 20.0

by ELISA (ENZYME LINKED IMMUNOASSAY)

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.

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Test Name	Value	Unit	Biological Reference interval

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.

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Test Name	Value	Unit	Biological Reference interval

HEPATITIS B SURFACE ANTIGEN (HBsAg) ULTRA

HEPATITIS B SURFACE ANTIGEN (HBsAg): SERUM	0.19	S/CO	NEGATIVE: < 1.0 POSITIVE: > 1.0
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
HEPATITIS B SURFACE ANTIGEN (HBsAg) RESULT	NON REACTIVE		
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			

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

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	39.4	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	198	pg/mL	190.0 - 890.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

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.

*** End Of Report ***

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