NAME	**: Mrs. PARAMJIT**		
AGE/ GENDER	: 67 YRS/FEMALE	**PATIENT ID**	: 1558190
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 042407230008**
REFERRED BY	: NAGPAL HOSPITAL (SHAHBAD)	**REGISTRATION DATE**	: 23/Jul/2024 02:10 PM
BARCODE NO.	: A0465034	**COLLECTION DATE**	: 23/Jul/2024 03:36PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	**REPORTING DATE**	: 23/Jul/2024 04:22PM
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*	**285.74 H**	**mg/dL**	**OPTIMAL: < 200.0** **BORDERLINE HIGH: 200.0 - 239.0** **HIGH CHOLESTEROL: > OR = 240.0**
TRIGLYCERIDES: SERUM *by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)*	**283.96 H**	**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*	56.51	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**172.44 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*	**229.23 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*	**56.79 H**	**mg/dL**	**0.00 - 45.00**
TOTAL LIPIDS: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**855.44 H**	**mg/dL**	**350.00 - 700.00**
CHOLESTEROL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**5.06 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***	**3.05^{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***	**5.02^{H}**	**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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Test Name	Value	Unit	Biological Reference interval

CREATININE PHOSPHOKINASE (CPK-NAC) TOTAL

CREATININE PHOSPHO KINASE (CPK-NAC) *by SPECTROPHOTOMETERY*	61.77	IU/L	24 - 190

Interpretation:-

1.Serum creatine kinase (CK) activity is greatly elevated, at some time during the course of the disease, in all types of muscular dystrophy, and especially so in Duchenne type, in which levels up to 50 times the upper limit of normal may be encountered.

2.In progressive muscular dystrophy, enzyme activity in serum is highest in infancy and childhood (7-10 years of age) and may be elevated long before the disease is clinically apparent.

3.Quite high values of CK are noted in viral myositis, polymyositis, and similar muscle diseases.

4.However, in neurogenic Parkinsonism, serum enzyme activity is normal. Very high activity is also encountered in malignant hyperthermia.

Significance:-

1.An early rise in CK is also seen after an acute MI, with values peaking at 12 to 24 hours and falling back to normal in 3 to 4 days.

2.Although total CK activity has been used as a diagnostic test for MI, it has been replaced by the troponin T and I immunoassays, and is no longer the laboratory test choice for diagnosing and monitoring acute infarctions.

3.Serum CK activity may increase in patients with acute cerebrovascular disease or neurosurgical intervention and with cerebral ischemia.

4.Serum CK activity also demonstrates an inverse relationship with thyroid activity. About 60% of hypothyroid subjects show an average elevation of CK activity 5-fold over the upper reference limit; elevation of as high as 50-fold may also be found.

Note: Exercise and muscle trauma (contact sports, traffic accidents, intramuscular injections, surgery, convulsions, wasp or bee stings, and burns) can elevate serum creatine kinase values.

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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

ANTI NUCLEAR ANTIBODY/FACTOR (ANA/ANF)

ANTI NUCLEUR ANTIBODIES (ANA): SERUM *by ELISA (ENZYME LINKED IMMUNOASSAY)*	0.63	INDEX VALUE	NEGATIVE: < 1.0 BORDERLINE: 1.0 - 1.20 POSITIVE: > 1.20

INTERPRETATION:-

1.For diagnostic purposes, ANA value should be used as an adjuvant to other clinical and laboratory data available.
2.Measurement of antinuclear antibodies (ANAs) in serum is the most commonly performed screening test for patients suspected of having a systemic rheumatic disease, also referred to as connective tissue disease.
3.ANAs occur in patients with a variety of autoimmune diseases, both systemic and organ-specific. They are particularly common in the systemic rheumatic diseases, which include lupus erythematosus (LE), discoid LE, drug-induced LE, mixed connective tissue disease, Sjogren syndrome scleroderma (systemic sclerosis), CREST (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia) syndrome, polymyositis/dermatomyositis, and rheumatoid arthritis.

NOTE:

1.The diagnosis of a systemic rheumatic disease is based primarily on the presence of compatible clinical signs and symptoms.
The results of tests for autoantibodies including ANA and specific autoantibodies are ancillary. Additional diagnostic criteria include consistent histopathology or specific radiographic findings. Although individual systemic rheumatic diseases are relatively uncommon, a great many patients present with clinical findings that are compatible with a systemic rheumatic disease ANA screening may be useful for ruling out the disease.
2.Secondary, disease specific auto antibodies maybe ordered for patients who are screen positive as ancillary aids for the diagnosis of specific auto-immune disorders.

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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)*	74.7	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	874	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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