

KOS Diagnostic Lab (A Unit of KOS Healthcare)



Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist

IU/L

24 - 190

NAME : Mrs. DARSHAN GUPTA

AGE/ GENDER : 78 YRS/FEMALE **PATIENT ID** : 1589758

COLLECTED BY : SURJESH REG. NO./LAB NO. : 012408240019

REFERRED BY **REGISTRATION DATE** : 24/Aug/2024 09:31 AM BARCODE NO. :01515605 **COLLECTION DATE** : 24/Aug/2024 09:46AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 24/Aug/2024 10:44AM

46.31

CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit **Biological Reference interval**

CLINICAL CHEMISTRY/BIOCHEMISTRY CREATININE PHOSPHOKINASE (CPK-NAC) TOTAL

CREATININE PHOSPHO KINASE (CPK-NAC)

by SPECTROPHOTOMETERY

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.

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 Although total CK activity has been used as a diagnostic test for MI, it has been replaced by the troponin T and I immunoassays, and is no longer the laboratory test choice for diagnosing and monitoring acute infarctions.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



CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY) DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST



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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 52.3 ng/mL

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

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.

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 > 2000^H 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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