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 CEO & Consultant Pathologist

NAME	: Mr. VIVEK MAHAJAN	PATIENT ID	: 1563071
AGE/ GENDER	: 38 YRS/MALE	REG. NO./LAB NO.	: 012407270055
COLLECTED BY	:	REGISTRATION DATE	: 27/Jul/2024 07:53 PM
REFERRED BY	:	COLLECTION DATE	: 27/Jul/2024 08:21PM
BARCODE NO.	: 01513934	REPORTING DATE	: 27/Jul/2024 10:47PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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ENDOCRINOLOGY

ANGIOTENSIN CONVERTING ENZYME (ACE): SERUM

ANGIOTENSIN CONVERTING ENZYME (ACE): SERUM	13.1	U/L	8.0 - 52.0
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by FURYACRYLOYLPHENYLALANYLGLYCYLGLYCINE (FAPPG)

INTERPRETATION

1. Angiotensin converting Enzyme (ACE) also known as kinase II, is present in many cells types such as neuronal cells, renal proximal tubular cells, and mostly in endothelial cells.
2. Angiotensin converting enzyme (ACE) modulates peripheral vascular resistance as well as renal and cardiovascular function. It is responsible for conversion of Angiotensin I to Angiotensin II as well as inactivation of bradykinin
3. It is attached to endothelial surface membrane by an anchor peptide and can be cleaved to be released into the blood circulation as soluble enzyme. Serum ACE activity is significantly elevated in patients with untreated active disease.
4. Majority of ACE is tissue bound (> 90%) found predominantly in lungs & testes
5. It has been established as an important diagnostic parameter in Sarcoidosis. Spontaneous or induced remission of sarcoidosis has been seen, by decreasing serum ACE values.

FACTORS AFFECTING ACE LEVELS:

1. Smoking – ACE activity is 30% lower in smokers
2. Thyroid hormone- Stimulates ACE synthesis
3. Postmenopausal estrogen replacement – ACE activity is 20% lower

INCREASED LEVELS:

1. Sarcoidosis – ACE levels are used in the diagnosis and monitoring of this disease and are directly related to the number of organs affected and activity of granulomas. Mature granulomas produce less ACE than developing ones. ACE is more likely to be elevated with pulmonary involvement than with purely hilar adenopathy.
2. Pulmonary causes like Emphysema, Asthma, Small cell carcinoma & Squamous cell carcinoma, Idiopathic pulmonary fibrosis
3. Renal diseases – patients on hemodialysis show high ACE levels as compared to patients who are not on dialysis, chronic renal failure
4. Other causes – Multiple sclerosis, Addison's disease, Hyperthyroidism, Diabetes Alcoholic hepatitis & cirrhosis & Peptic ulcer, histoplasmosis, hodgekins disease, gauchers disease, leprosy, amyloidosis, tuberculosis
5. Elevated ACE is thought to be a risk factor for myocardial infarction & cardiomyopathy.
6. ACE inhibitors have found wide spread application in treatment of systemic hypertension and Congestive Heart Failure (CHF). Monitoring of ACE may be beneficial to determine the optimum low dose of ACE inhibitor.

DECREASED LEVELS

1. Chronic liver disease.
2. Anorexia nervosa
3. Hypothyroidism

To be correlated clinically




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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM
 by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

16.2^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
PREFERRED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

- 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.
- 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.
- 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 hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

- Lack of sunshine exposure.
- Inadequate intake, malabsorption (celiac disease)
- Depressed Hepatic Vitamin D 25- hydroxylase activity
- Secondary to advanced Liver disease
- Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:

- 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 hyperphosphatemia.

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 interfere with Vitamin D absorption.




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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM 296 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 lgestion
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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