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NAME	**: Mrs. ANJALI**		
AGE/ GENDER	: 36 YRS/FEMALE	**PATIENT ID**	: 1551264
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012407160056**
REFERRED BY	:	**REGISTRATION DATE**	: 16/Jul/2024 05:14 PM
BARCODE NO.	: 01513269	**COLLECTION DATE**	: 16/Jul/2024 05:18PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 16/Jul/2024 06:21PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

ALKALINE PHOSPHATASE (ALP)

ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	119	U/L	40.0 - 150.0

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Test Name	Value	Unit	Biological Reference interval

URIC ACID

URIC ACID: SERUM	3.9	mg/dL	2.50 - 6.80

by URICASE - OXIDASE PEROXIDASE

INTERPRETATION:-

1.GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.
2.Uric Acid is the end product of purine metabolism . Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

1.Idiopathic primary gout.
2.Excessive dietary purines (organ meats,legumes,anchovies, etc).
3.Cytolytic treatment of malignancies especially leukemais & lymphomas.
4.Polycythemai vera & myeloid metaplasia.
5.Psoriasis.
6.Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCREATION (BY KIDNEYS)

1.Alcohol ingestion.
2.Thiazide diuretics.
3.Lactic acidosis.
4.Aspirin ingestion (less than 2 grams per day).
5.Diabetic ketoacidosis or starvation.
6.Renal failure due to any cause etc.

DECREASED:-

(A).DUE TO DIETARY DEFICIENCY

1.Dietary deficiency of Zinc, Iron and molybdenum.
2.Fanconi syndrome & Wilsons disease.
3.Multiple sclerosis .
4.Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCREATION

1.Drugs:-Probenecid , sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosterroids and ACTH, anti-coagulants and estrogens etc.

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Test Name	Value	Unit	Biological Reference interval
CALCIUM			
CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	9.12	mg/dL	8.50 - 10.60

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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Test Name	Value	Unit	Biological Reference interval

PHOSPHOROUS

PHOSPHOROUS: SERUM	2.31	mg/dL	2.30 - 4.70

by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY

INTERPREATION:-

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 (HYPERPHOPHATEMIA):-

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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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)*	55.7	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.*

*** End Of Report ***

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