Dr. Vinay Chopra
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Dr. Yugam Chopra
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NAME	**: Mrs. FARHA**		
AGE/ GENDER	: 51 YRS/FEMALE	**PATIENT ID**	: 1795951
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012503180032**
REFERRED BY	:	**REGISTRATION DATE**	: 18/Mar/2025 11:11 AM
BARCODE NO.	: 01527334	**COLLECTION DATE**	: 18/Mar/2025 11:18AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 18/Mar/2025 01:41PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

URIC ACID

URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	5.39	mg/dL	2.50 - 6.80

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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CALCIUM

CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	9.05	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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ENDOCRINOLOGY

FOLLICLE STIMULATING HORMONE (FSH)

FOLLICLE STIMULATING HORMONE (FSH): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	87.1	mIU/ml	FEMALE FOLLICULAR PHASE: 2.0 - 15.0 FEAMLE LUTEAL PHASE: 2.0 - 12.0 FEMALE 0VULATORY PHASE: 2.0 - 25.0 MENOPAUSAL: >40.0 PREGNANCY: 0.0 - 12.0 PRIMARY OVARIAN FAILURE: 40.0 - 150.0 MALE: 2.0 - 15.0

INTERPRETATION:

1. Gonadotropin-releasing hormone from the hypothalamus controls the secretion of the gonadotropins, follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the anterior pituitary.
2. The menstrual cycle is divided by a midcycle surge of both FSH and LH into a follicular phase and a luteal phase.
3. FSH appears to control gametogenesis in both males and females.

The test is useful in the following settings:

1. An adjunct in the evaluation of menstrual irregularities.
2. Evaluating patients with suspected hypogonadism.
3. Predicting ovulation
4. Evaluating infertility
5. Diagnosing pituitary disorders
6. In both males and females, primary hypogonadism results in an elevation of basal follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels.

FSH and LH LEVELS ELEVATED IN:

1. Primary gonadal failure
2. Complete testicular feminization syndrome.
3. Precocious puberty (either idiopathic or secondary to a central nervous system lesion)
4. Menopause (postmenopausal FSH levels are generally >40 IU/L)
5. Primary ovarian hypofunction in females
6. Primary hypogonadism in males

NOTE:

1. Normal or decreased FSH is seen in polycystic ovarian disease in females
2. FSH and LH are both decreased in failure of the pituitary or hypothalamus.

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IMMUNOPATHOLOGY/SEROLOGY

RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM

RHEUMATOID (RA) FACTOR QUANTITATIVE: SERUM *by NEPHLOMETRY*	2.36	IU/mL	NEGATIVE: < 18.0 BORDERLINE: 18.0 - 25.0 POSITIVE: > 25.0

INTERPRETATION:-

RHEUMATOID FACTOR (RA):

1. Rheumatoid factors (RF) are antibodies that are directed against the Fc fragment of IgG altered in its tertiary structure.
2. Over 75% of patients with rheumatoid arthritis (RA) have an IgM antibody to IgG immunoglobulin. This autoantibody (RF) is diagnostically useful although it may not be etiologically related to RA.
3. Inflammatory Markers such as ESR & C-Reactive protein (CRP) are normal in about 60 % of patients with positive RA.
4. The titer of RF correlates poorly with disease activity, but those patients with high titers tend to have more severe disease course.
5. The test is useful for diagnosis and prognosis of rheumatoid arthritis.

RHEUMATOID ARTHIRITIS:

1. Rheumatoid Arthiritis is a systemic autoimmune disease that is multi-functional in origin and is characterized by chronic inflammation of the membrane lining (synovium) joints which ledas to progressive joint destruction and in most cases to disability and reduction of quality life.
2. The disease spredas from small to large joints, with greatest damage in early phase.
3. The diagnosis of RA is primarily based on clinical, radiological & immunological features.The most frequent serological test is the measurement of RA factor.

CAUTION (FALSE POSTIVE):-

1. RA factor is not specific for Rheumatoid arthiritis, as it is often present in healthy individuals with other autoimmune diseases and chronic infections.
2. Non rheumatoid and rheumatoid arthritis (RA) populations are not clearly separate with regard to the presence of rheumatoid factor (RF) (15% of RA patients have a nonreactive titer and 8% of nonrheumatoid patients have a positive titer).
3. Patients with various nonrheumatoid diseases,characterized by chronic inflammation may have positive tests for RF. These diseases include systemic lupus erythematosus, polymyositis, tuberculosis, syphilis, viral hepatitis, infectious mononucleosis, and influenza.
4. Anti-CCP have been discovered in joints of patients with RA, but not in other form of joint disease.Anti-CCP2 is HIGHLY SENSITIVE (71%) & more specific (98%) than RA factor.
5. Upto 30 % of patients with Seronegative Rheumatoid arthiritis also show Anti-CCP antibodies.
6. The positive predictive value of Anti-CCP antibodies for Rheumatoid Arthiritis is far greater than Rheumatoid factor.

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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	34.763	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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