



A PIONEER DIAGNOSTIC CENTRE

NAME : Mr. MOHIT

AGE/ GENDER : 19 YRS/MALE **PATIENT ID** : 1577919

COLLECTED BY : 122408120004 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 12/Aug/2024 09:30 AM BARCODE NO. : 12504111 **COLLECTION DATE** : 12/Aug/2024 09:32AM CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE REPORTING DATE : 12/Aug/2024 01:11PM

CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

Test Name Value Unit **Biological Reference interval**

CLINICAL CHEMISTRY/BIOCHEMISTRY

CALCIUM

10.55 CALCIUM: SERUM mg/dL 8.50 - 10.60

by ARSENAZO III, SPECTROPHOTOMETRY

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.



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

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





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■ 0171-2532620, 8222896961 ■ pkrjainhealthcare@gmail.com

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ELECTROLYTES COMPLETE PROFILE

SODIUM: SERUM	139.3	mmol/L	135.0 - 150.0
by ISE (ION SELECTIVE ELECTRODE)			
POTASSIUM: SERUM	4.3	mmol/L	3.50 - 5.00
by ISE (ION SELECTIVE ELECTRODE)			
CHLORIDE: SERUM	104.48	mmol/L	90.0 - 110.0
by ISE (ION SELECTIVE ELECTRODE)			

INTERPRETATION:-

CLIENT CODE.

SODIUM:-

Sodium is the major cation of extra-cellular fluid. Its primary function in the body is to chemically maintain osmotic pressure & acid base balance & to transmit nerve impulse.

HYPONATREMIA (LOW SODIUM LEVEL) CAUSES:-

- 1. Low sodium intake.
- 2. Sodium loss due to diarrhea & vomiting with adequate water and iadequate salt replacement.
- 3. Diuretics abuses.
- 4. Salt loosing nephropathy.
- 5. Metabolic acidosis.
- 6. Adrenocortical issuficiency.
- 7. Hepatic failure.

HYPERNATREMIA (INCREASED SODIUM LEVEL) CAUSES:-

- 1. Hyperapnea (Prolonged)
- 2. Diabetes insipidus
- 3. Diabetic acidosis
- 4. Cushings syndrome
- 5.Dehydration

POTASSIUM:-

Potassium is the major cation in the intracellular fluid. 90% of potassium is concentrated within the cells. When cells are damaged, potassium is released in the blood.

HYPOKALEMIA (LOW POTASSIUM LEVELS):-

- 1. Diarrhoea, vomiting & malabsorption.
- 2. Severe Burns.
- 3. Increased Secretions of Aldosterone

HYPERKALEMIA (INCREASED POTASSIUM LEVELS):-

- 1.Oliguria
- 2. Renal failure or Shock
- 3. Respiratory acidosis



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4. Hemolysis of blood

CLIENT CODE.



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Test Name Value Unit **Biological Reference interval**

MAGNESIUM

REPORTING DATE

MAGNESIUM: SERUM 2.4 mg/dL 1.6 - 2.6

by XYLIDYL BLUE, SPECTROPHOTMETRY

INTERPRETATION:-

CLIENT CODE.

1. Magnesium along with potassium is a major intracellular cation.

2. Magnesium is a cofactor of many enzyme systems. All adenosine triphosphate (ATP)-dependent enzymatic reactions require magnesium as a cofactor. 3.Approximately 70% of magnesium ions are stored in bone. The remainder is involved in intermediary metabolic processes; about 70% is present in free form while the other 30% is bound to proteins (especially albumin), citrates, phosphate, and other complex formers. The serum magnesium level is kept constant within very narrow limits. Regulation takes place mainly via the kidneys, primarily via the ascending loop of Henle.

INCREASD (HYPERMAGNESIA):-Conditions that interfere with glomerular filtration result in retention of magnesium and hence elevation of serum concentrations.

- 1.Acute and chronic renal failure.
- 2.magnesium overload.
- 3. Magnesium release from the intracellular space.
- 4.Mild-to-moderate hypermagnesemia may prolong atrioventricular conduction time. Magnesium toxicity may result in central nervous system (CNS) depression, cardiac arrest, and respiratory arrest.

DECREASED (HYPOMAGNESIA):-

- 1.Chronic alcoholism.
- 2.Childhood malnutrition.
- 3. Malabsorption.
- 4. Acute pancreatitis
- 5. Hypothyroidism.
- 6.Chronic glomerulonephritis.
- 7. Aldosteronism.
- 8. Prolonged intravenous feeding.

NOTE:-

Numerous studies have shown a correlation between magnesium deficiency and changes in calcium-, potassium-, and phosphate-homeostasis which are associated with cardiac disorders such as ventricular arrhythmias that cannot be treated by conventional therapy, increased sensitivity to digoxin, coronary artery spasms, and sudden death. Additional concurrent symptoms include neuromuscular and neuropsychiatric disorders.

*** End Of Report ***

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