

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	MD (Pathology	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologist		n <b>Chopra</b> 9 (Pathology) t Pathologist
NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Mrs. SATYA DEVI : 70 YRS/FEMALE : : : 01518251 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD	R R C( R	ATIENT ID EG. NO./LAB NO. EGISTRATION DATE DLLECTION DATE EPORTING DATE	: 1633766 : 012410030046 : 03/Oct/2024 05:55 PM : 03/Oct/2024 05:57PM : 03/Oct/2024 07:12PM
Test Name		Value	Unit	Biological Reference interval
	CLIN	JICAL CHEMIST	RY/BIOCHEMISTR	Y
		CALC		
parathyroid gland, ou 2. Calcium levels may 3.The calcium conter and <1% is present ir 4. In serum, calcium present as free or ior <b>NOTE:</b> -Calcium ions a addition, calcium ior	al) estimation is used for the di r gastrointestinal tract. y also reflect abnormal vitamin it of an adult is somewhat over in the extra-osseous intracellula is bound to a considerable exte nized calcium. affect the contractility of the hear is play an important role in bloc	D or protein levels. 1 kg (about 2% of the r space or extracellul nt to proteins (appro art and the skeletal n	e body weight).Of this, 9 ar space (ECS). ximately 40%), 10% is ir nusculature, and are ess	8.50 - 10.60 isorders including diseases of bone, kidney, 9% is present as calcium hydroxyapatite in bones in the form of inorganic complexes, and 50% is ential for the function of the nervous system. In
I.Due to the absence 2. Chronic renal failu and skeletal resistance	V CALCIUM LEVELS) CAUSES :- e or impaired function of the pa ire is also frequently associated ce to the action of parathyroid h istic symptom of hypocalcemia	with hypocalcemia on ormone (PTH).	due to decreased vitami	in-D synthesis as well as hyperphosphatemia
1.Increased mobiliza 2.Primary hyperpara 3.Bone metastasis of	CREASE CALCIUM LEVELS) CAUSE tion of calcium from the skeleta thyroidism (pHPT) carcinoma of the breast, prosta calcemia may result in cardiac a	al system or increase ate, thyroid gland, or		





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Test Name		Value	Unit	Biological Reference interval
		ELECTROLYTES	COMPLETE PROFILE	
sodium: serum		142.4	mmol/L	135.0 - 150.0
by ISE (ION SELECTIVE	E ELECTRODE)			
POTASSIUM: SERUM by ise (ION SELECTIVE		4.09	mmol/L	3.50 - 5.00
CHLORIDE: SERUM		106.8	mmol/L	90.0 - 110.0
	, _, _, _, _, _,			
balance & to transmit HYPONATREMIA (LOV	ation of extra-cellular nerve impulse. / SODIUM LEVEL) CAUSI		in the body is to chemical	ly maintain osmotic pressure & acid base
INTERPRETATION:- SODIUM:- Sodium is the major of balance & to transmit HYPONATREMIA (LOV 1. Low sodium intake. 2. Sodium loss due to 3. Diuretics abuses. 4. Salt loosing nephro 5. Metabolic acidosis 6. Adrenocortical issu 7.Hepatic failure.	ation of extra-cellular nerve impulse. <b>/ SODIUM LEVEL) CAUSI</b> diarrhea & vomiting wi pathy. ificiency . <b>REASED SODIUM LEVEL</b>	S:-	in the body is to chemical dequate salt replacement.	lly maintain osmotic pressure & acid base



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Test Name	Value	Unit	Biological Reference interval

4.Hemolysis of blood





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Test Name		Value	Unit	Biological Reference interval
		MAGNE	SIUM	
MAGNESIUM: SERU		1.98	mg/dL	1.6 - 2.6

## **INTERPRETATION:-**

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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