

Dr. Vinay Chopra  
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Chairman & Consultant Pathologist

Dr. Yugam Chopra  
MD (Pathology)  
CEO & Consultant Pathologist

NAME : Mr. BRIJPAL WALIA  
AGE/ GENDER : 61 YRS/MALE  
COLLECTED BY :  
REFERRED BY :  
BARCODE NO. : 01521964  
CLIENT CODE. : KOS DIAGNOSTIC LAB  
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1690444  
REG. NO./LAB NO. : 012412040037  
REGISTRATION DATE : 04/Dec/2024 12:26 PM  
COLLECTION DATE : 04/Dec/2024 12:30PM  
REPORTING DATE : 04/Dec/2024 03:16PM

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

UREA

UREA: SERUM  
by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)  
Rechecked

69.58<sup>H</sup> mg/dL 10.00 - 50.00



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CREATININE

CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY Rechecked	2.32 <sup>H</sup>	mg/dL	0.40 - 1.40
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<b>BARCODE NO.</b>	: 01521964	<b>REPORTING DATE</b>	: 04/Dec/2024 03:03PM
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### ELECTROLYTES COMPLETE PROFILE

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	144.7	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.59	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	108.53	mmol/L	90.0 - 110.0

#### INTERPRETATION:-

##### 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 inadequate salt replacement.
3. Diuretics abuses.
4. Salt loosing nephropathy.
5. Metabolic acidosis.
6. Adrenocortical insufficiency .
7. Hepatic failure.

##### HYPERNATREMIA (INCREASED SODIUM LEVEL) CAUSES:-

1. Hyperapnea (Prolonged)
2. Diabetes insipidus
3. Diabetic acidosis
4. Cushing's 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



  
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## ENDOCRINOLOGY

### INTACT PARATHYROID HORMONE (PTH)

INTACT PARATHROID HORMONE (PTH): SERUM	<b>212.1<sup>H</sup></b>	pg/mL	9.5 - 75.0
<i>by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)</i>			

#### Intrepretation:-

Parathyroid hormone (PTH) is produced and secreted by the parathyroid glands, which are located along the posterior aspect of the thyroid gland. The serum calcium level regulates PTH secretion via negative feedback through the parathyroid calcium sensing receptor (CASR). Decreased calcium levels stimulate PTH release. Secreted PTH interacts with its specific type II G-protein receptor, causing rapid increases in renal tubular reabsorption of calcium and decreased phosphorus reabsorption. It also participates in long-term calciostatic functions by enhancing mobilization of calcium from bone and increasing renal synthesis of 1,25-dihydroxy vitamin D, which, in turn, increases intestinal calcium absorption.

The assay is useful for:

- Differential diagnosis of hypercalcemia
- Diagnosis of primary, secondary, and tertiary hyperparathyroidism
- Diagnosis of hypoparathyroidism
- Monitoring end-stage renal failure patients for possible renal osteodystrophy

Interpretation of results:

- An (appropriately) low PTH level and high phosphorus level in a hypercalcemic patient suggests that the hypercalcemia is not caused by PTH or PTH-like substances.
- An (appropriately) low PTH level with a low phosphorus level in a hypercalcemic patient suggests the diagnosis of paraneoplastic hypercalcemia.
- A low or normal PTH in a patient with hypocalcemia suggests hypoparathyroidism.

Low serum calcium and high PTH levels in a patient with normal renal function suggest resistance to PTH action (pseudohypoparathyroidism type 1a, 1b, 1c, or 2) or, very rarely, bio-ineffective PTH.

Elevated PTH value with a normal serum calcium in many cases in India is due to secondary hyperparathyroidism, primary cause being Vitamin D deficiency.



  
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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	<b>23.192<sup>L</sup></b>	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
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#### 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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## CLINICAL PATHOLOGY

### URINE ROUTINE & MICROSCOPIC EXAMINATION

#### PHYSICAL EXAMINATION

QUANTITY RECEIVED	10	ml	
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
COLOUR	AMBER YELLOW		PALE YELLOW
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
TRANSPARANCY	CLEAR		CLEAR
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
SPECIFIC GRAVITY	1.01		1.002 - 1.030
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			


#### CHEMICAL EXAMINATION


REACTION	ACIDIC		
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
PROTEIN	Negative		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
SUGAR	2+		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
pH	<=5.0		5.0 - 7.5
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
BILIRUBIN	Negative		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
NITRITE	Negative		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
UROBILINOGEN	Normal	EU/dL	0.2 - 1.0
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
KETONE BODIES	Negative		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
BLOOD	Negative		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
ASCORBIC ACID	NEGATIVE (-ve)		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			

#### MICROSCOPIC EXAMINATION

RED BLOOD CELLS (RBCs)	NEGATIVE (-ve)	/HPF	0 - 3
<i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>			



  
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PUS CELLS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	3-4	/HPF	0 - 5
EPITHELIAL CELLS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	2-3	/HPF	ABSENT
CRYSTALS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	ABSENT		ABSENT



  
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**MICROALBUMIN/CREATININE RATIO - RANDOM URINE**

MICROALBUMIN: RANDOM URINE by SPECTROPHOTOMETRY	11.55	mg/L	0 - 25
CREATININE: RANDOM URINE by SPECTROPHOTOMETRY	69.63	mg/dL	20 - 320
MICROALBUMIN/CREATININE RATIO - RANDOM URINE by SPECTROPHOTOMETRY	16.59	mg/g	0 - 30

**INTERPRETATION:-**

PHYSIOLOGICALLY NORMAL:	mg/L	0 - 30
MICROALBUMINURIA:	mg/L	30 - 300
GROSS PROTEINURIA:	mg/L	> 300

Long standing un-treated Diabetes and Hypertension can lead to renal dysfunction.

2. Diabetic nephropathy or kidney disease is the most common cause of end stage renal disease(ERSD) or kidney failure.

3. Presence of Microalbuminuria is an early indicator of onset of compromised renal function in these patients.

4. Microalbuminuria is the condition when urinary albumin excretion is between 30-300 mg & above this it is called as macroalbuminuria, the presence of which indicates serious kidney disease.

5. Microalbuminuria is not only associated with kidney disease but of cardiovascular disease in patients with diabetes & hypertension.

6. Microalbuminuria reflects vascular damage & appear to be a marker of early arterial disease & endothelial dysfunction.

**NOTE:-** IF A PATIENT HAS = 1+ PROTEINURIA (30 mg/dl OR 300 mg/L) BY URINE DIPSTICK (URINE ANALYSIS), OVERT PROTEINURIA IS PRESENT AND TESTING FOR MICROALBUMIN IS INAPPROPRIATE. IN SUCH A CASE, URINE PROTEIN:CREATININE RATIO OR 24 HOURS TOTAL URINE MICROPROTEIN IS APPROPRIATE.

\*\*\* End Of Report \*\*\*



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