

Dr. Vinay Chopra  
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Chairman & Consultant Pathologist

Dr. Yugam Chopra  
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CEO & Consultant Pathologist

NAME : Mr. BERINDER SINGH VIRK  
AGE/ GENDER : 67 YRS/MALE  
COLLECTED BY :  
REFERRED BY :  
BARCODE NO. : A1260144  
CLIENT CODE. : KOS DIAGNOSTIC SHAHBAD  
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1661571  
REG. NO./LAB NO. : 042412190003  
REGISTRATION DATE : 19/Dec/2024 01:17 PM  
COLLECTION DATE : 19/Dec/2024 02:28PM  
REPORTING DATE : 19/Dec/2024 04:48PM

Test Name	Value	Unit	Biological Reference interval
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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	53.22 <sup>H</sup>	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	1.21	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	24.87	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	20.55 <sup>H</sup>	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	43.98	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	7.31	mg/dL	3.60 - 7.70



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**INTERPRETATION:**

Normal range for a healthy person on normal diet: 12 - 20

To Differentiate between pre- and postrenal azotemia.

**INCREASED RATIO (>20:1) WITH NORMAL CREATININE:**

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased glomerular filtration rate.

2. Catabolic states with increased tissue breakdown.

3. GI hemorrhage.

4. High protein intake.

5. Impaired renal function plus .

6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushings syndrome, high protein diet, burns, surgery, cachexia, high fever).

7. Urine reabsorption (e.g. ureterocolostomy)

8. Reduced muscle mass (subnormal creatinine production)

9. Certain drugs (e.g. tetracycline, glucocorticoids)

**INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:**

1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).

2. Prerenal azotemia superimposed on renal disease.

**DECREASED RATIO (<10:1) WITH DECREASED BUN :**

1. Acute tubular necrosis.

2. Low protein diet and starvation.

3. Severe liver disease.

4. Other causes of decreased urea synthesis.

5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).

6. Inherited hyperammonemias (urea is virtually absent in blood).

7. SIADH (syndrome of inappropriate antidiuretic hormone) due to tubular secretion of urea.

8. Pregnancy.

**DECREASED RATIO (<10:1) WITH INCREASED CREATININE:**

1. Phenacimide therapy (accelerates conversion of creatine to creatinine).

2. Rhabdomyolysis (releases muscle creatinine).

3. Muscular patients who develop renal failure.

**INAPPROPRIATE RATIO:**

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).

2. Cephalosporin therapy (interferes with creatinine measurement).



*[Signature]*

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### ELECTROLYTES COMPLETE PROFILE

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	145.4	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	5.25 <sup>H</sup>	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	109.05	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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### CLINICAL PATHOLOGY

#### MICROALBUMIN/CREATININE RATIO - RANDOM URINE

MICROALBUMIN: RANDOM URINE by SPECTROPHOTOMETRY	60.9 <sup>H</sup>	mg/L	0 - 25
CREATININE: RANDOM URINE by SPECTROPHOTOMETRY	19.89 <sup>L</sup>	mg/dL	20 - 320
MICROALBUMIN/CREATININE RATIO - RANDOM URINE by SPECTROPHOTOMETRY	306.18 <sup>H</sup>	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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