

KOS Diagnostic Lab (A Unit of KOS Healthcare)



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

NAME : Mr. MUKESH CHOPRA

AGE/ GENDER : 64 YRS/MALE **PATIENT ID** : 1531990

COLLECTED BY : REG. NO./LAB NO. : 012409040023

 REFERRED BY
 : 04/Sep/2024 09:39 AM

 BARCODE NO.
 : 01516279
 COLLECTION DATE
 : 04/Sep/2024 09:40AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 04/Sep/2024 11:00AM

CLIENT ADDRESS: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

KIDNEY FUNCTION TEST (BASIC)

35.86	mg/dL	10.00 - 50.00
1.45 ^H	mg/dL	0.40 - 1.40
16.76	mg/dL	7.0 - 25.0
11.56	RATIO	10.0 - 20.0
24.73	RATIO	
6.97	mg/dL	3.60 - 7.70
	3	
	1.45^H 16.76 11.56	1.45 ^H mg/dL 16.76 mg/dL 11.56 RATIO 24.73 RATIO



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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 (pia (PLIN) rises dispreparties toly more than

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 inappropiate antidiuretic harmone) 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

INAPPROPIATE 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).



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ELECTROLYTES PROFILE: SODIUM AND POTASSIUM

SODIUM: SERUM 136.7 mmol/L 135.0 - 150.0

by ISE (ION SELECTIVE ELECTRODE)

POTASSIUM: SERUM 4.49 mmol/L 3.50 - 5.00

by ISE (ION SELECTIVE ELECTRODE)

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 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
- 4. Hemolysis of blood



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CLINICAL PATHOLOGY URINE ROUTINE & MICROSCOPIC EXAMINATION

PHYSICAL EXAMINATION

QUANTITY RECIEVED	10	ml
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		

COLOUR AMBER YELLOW PALE YELLOW

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

TRANSPARANCY HAZY CLEAR

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SPECIFIC GRAVITY

1.002 - 1.030

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

CHEMICAL EXAMINATION

REACTION ACIDIC

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

PROTEIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SUGAR

Negative

NEGATIVE (-ve)

UGAR Negative NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

pH 5.5 5.0 - 7.5

BILIRUBIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NITRITE Negative NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.

UROBILINOGEN Normal EU/dL 0.2 - 1.0 by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

KETONE BODIES Negative NEGATIVE (-ve)

BLOOD Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

ASCORBIC ACID NEGATIVE (-ve) NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

MICROSCOPIC EXAMINATION



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RED BLOOD CELLS (RBCs) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3	
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	5-7	/HPF	0 - 5	
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	ABSENT	
CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT		ABSENT	

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End Of Report



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