P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	**: Mrs. SUNEHRI DEVI**		
AGE/ GENDER	: 60 YRS/FEMALE	**PATIENT ID**	: 1674745
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122411180002**
REFERRED BY	:	**REGISTRATION DATE**	: 18/Nov/2024 08:42 AM
BARCODE NO.	: 12505701	**COLLECTION DATE**	: 18/Nov/2024 09:08AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 18/Nov/2024 11:20AM
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Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

PHOSPHOROUS

PHOSPHOROUS: SERUM *by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY*	3.28	mg/dL	2.30 - 4.70

INTERPREATION:-

1.Eighty-eight percent of the phosphorus contained in the body is localized in bone in the form of hydroxyapatite. The remainder is involved in intermediary carbohydrate metabolism and in physiologically important substances such as phospholipids, nucleic acids, and adenosine triphosphate (ATP).
2.Phosphorus occurs in blood in the form of inorganic phosphate and organically bound phosphoric acid. The small amount of extracellular organic phosphorus is found exclusively in the form of phospholipids.
3.Serum phosphate concentrations are dependent on meals and variation in the secretion of hormones such as parathyroid hormone (PTH) and may vary widely.

DECREASED (HYPOPHOSPHATEMIA):-

1.Shift of phosphate from extracellular to intracellular.
2.Renal phosphate wasting.
3.Loss from the gastrointestinal tract.
4.Loss from intracellular stores.

INCREASED (HYPERPHOPHATEMIA):-

1.Inability of the kidneys to excrete phosphate.
2.Increased intake or a shift of phosphate from the tissues into the extracellular fluid.

SIGNIFICANCE:-

1.Phosphate levels may be used in the diagnosis and management of a variety of disorders including bone, parathyroid and renal disease.
2.Hypophosphatemia is relatively common in hospitalized patients. Levels less than 1.5 mg/dL may result in muscle weakness, hemolysis of red cells, coma, and bone deformity and impaired bone growth.
3.The most acute problem associated with rapid elevations of serum phosphate levels is hypocalcemia with tetany, seizures, and hypotension. Soft tissue calcification is also an important long-term effect of high phosphorus levels.
4.Phosphorus levels less than 1.0 mg/dL are potentially life-threatening and are considered a critical value.

NOTE: Phosphorus has a very strong biphasic circadian rhythm. Values are lowest in the morning, peak first in the late afternoon and peak again in the late evening. The second peak is quite elevated and results may be outside the reference range

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KIDNEY FUNCTION TEST (BASIC)

Test Name	Value	Unit	Biological Reference interval
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	**83.66**^{H}	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	**2.25**^{H}	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETERY*	**39.09**^{H}	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	17.37	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	37.18	RATIO	
URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	6.14	mg/dL	2.50 - 6.80

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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 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 *by ISE (ION SELECTIVE ELECTRODE)*	135.8	mmol/L	135.0 - 150.0
POTASSIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	4.86	mmol/L	3.50 - 5.00

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

*** End Of Report ***

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