



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961 ✉ pkrjainhealthcare@gmail.com

TEST PERFORMED AT: KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	: Mr. SUMIT	PATIENT ID	: 1704653
AGE/ GENDER	: 40 YRS/MALE	REG. NO./LAB NO.	: 122412200017
COLLECTED BY	:	REGISTRATION DATE	: 20/Dec/2024 05:01 PM
REFERRED BY	:	COLLECTION DATE	: 20/Dec/2024 08:25PM
BARCODE NO.	: 12506238	REPORTING DATE	: 21/Dec/2024 02:38AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE		
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE RANDOM (R)

GLUCOSE RANDOM (R): PLASMA <i>by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)</i>	144.98^H	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > OR = 200.0
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INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A random plasma glucose level below 140 mg/dl is considered normal.
2. A random glucose level between 140 - 200 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A random glucose level of above 200 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.



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ALBUMIN

ALBUMIN: SERUM
by BROMOCRESOL GREEN

3.33^L

gm/dL

3.50 - 5.50



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Test Name	Value	Unit	Biological Reference interval
CALCIUM			
CALCIUM: SERUM <i>by ARSENAZO III, SPECTROPHOTOMETRY</i>	5.68 ^L	mg/dL	8.50 - 10.60

INTERPRETATION:-

1. Serum calcium (total) estimation is used for the diagnosis and monitoring of a wide range of disorders including diseases of bone, kidney, parathyroid gland, or gastrointestinal tract.
2. Calcium levels may also reflect abnormal vitamin D or protein levels.
3. The calcium content of an adult is somewhat over 1 kg (about 2% of the body weight). Of this, 99% is present as calcium hydroxyapatite in bones and <1% is present in the extra-osseous intracellular space or extracellular space (ECS).
4. In serum, calcium is bound to a considerable extent to proteins (approximately 40%), 10% is in the form of inorganic complexes, and 50% is present as free or ionized calcium.

NOTE:-Calcium ions affect the contractility of the heart and the skeletal musculature, and are essential for the function of the nervous system. In addition, calcium ions play an important role in blood clotting and bone mineralization.

HYPOCALCEMIA (LOW CALCIUM LEVELS) CAUSES :-


1. Due to the absence or impaired function of the parathyroid glands or impaired vitamin-D synthesis.
2. Chronic renal failure is also frequently associated with hypocalcemia due to decreased vitamin-D synthesis as well as hyperphosphatemia and skeletal resistance to the action of parathyroid hormone (PTH).
3. **NOTE:-** A characteristic symptom of hypocalcemia is latent or manifest tetany and osteomalacia.


HYPERCALCEMIA (INCREASE CALCIUM LEVELS) CAUSES:-

1. Increased mobilization of calcium from the skeletal system or increased intestinal absorption.
2. Primary hyperparathyroidism (pHPT)
3. Bone metastasis of carcinoma of the breast, prostate, thyroid gland, or lung.

NOTE:-Severe hypercalcemia may result in cardiac arrhythmia.




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PHOSPHOROUS

PHOSPHOROUS: SERUM	7.39^H	mg/dL	2.5 - 4.5
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by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY

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 (HYPERPHOSPHATEMIA):-


- 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)


UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	345.79^H	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	13.46^H	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	161.58^H	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	12	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	25.69	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	14.73^H	mg/dL	3.60 - 7.70

NOTE 2 ADVICE

RESULT RECHECKED TWICE
KINDLY CORRELATE CLINICALLY




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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).



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IRON PROFILE

IRON: SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	32.1^L	µg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	165.45	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM <i>by SPECTROPHOTOMETRY</i>	197.55^L	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY (FERENE)</i>	16.25	%	15.0 - 50.0
TRANSFERRIN: SERUM <i>by SPECTROPHOTOMETRY (FERENE)</i>	140.26^L	mg/dL	200.0 - 350.0

INTERPRETATION:-

VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:

- 1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia,anemia of chronic disease and thalassemia syndromes.
2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BINDING CAPACITY (TIBC):

- 1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:

- 1.Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.



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CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

PHYSICAL EXAMINATION

QUANTITY RECEIVED	10	ml	
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
COLOUR	PALE YELLOW		PALE YELLOW
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
TRANSPARANCY	HAZY		CLEAR
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
SPECIFIC GRAVITY	1.02		1.002 - 1.030
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			

CHEMICAL EXAMINATION

REACTION	ACIDIC		
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
PROTEIN	3+		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
SUGAR	Negative		NEGATIVE (-ve)
<i>by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY</i>			
pH	5.5		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	TRACE		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)	1-2	/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

*** End Of Report ***



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