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| **NAME** | **: Mrs. PARKASHO DEVI** | | |
| **AGE/ GENDER** | : 60 YRS/FEMALE | **PATIENT ID** | : 1631930 |
| **COLLECTED BY** | : | **REG. NO./LAB NO.** | **: 012410020007** |
| **REFERRED BY** | : | **REGISTRATION DATE** | : 02/Oct/2024 08:09 AM |
| **BARCODE NO.** | : 01518147 | **COLLECTION DATE** | : 02/Oct/2024 08:13AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 02/Oct/2024 10:45AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **CLINICAL CHEMISTRY/BIOCHEMISTRY** | | | |
| **KIDNEY FUNCTION TEST (COMPLETE)** | | | |
| **UREA: SERUM** *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)* | **132.03$^{H}$** | **mg/dL** | **10.00 - 50.00** |
| **CREATININE: SERUM** *by ENZYMATIC, SPECTROPHOTOMETERY* | **7.44$^{H}$** | **mg/dL** | **0.40 - 1.20** |
| **BLOOD UREA NITROGEN (BUN): SERUM** *by CALCULATED, SPECTROPHOTOMETRY* | **61.7$^{H}$** | **mg/dL** | **7.0 - 25.0** |
| **BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM** *by CALCULATED, SPECTROPHOTOMETRY* | **8.29$^{L}$** | **RATIO** | **10.0 - 20.0** |
| UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY* | 17.75 | RATIO | |
| URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE* | 4.56 | mg/dL | 2.50 - 6.80 |
| CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY* | 8.97 | mg/dL | 8.50 - 10.60 |
| **PHOSPHOROUS: SERUM** *by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY* | **5.24$^{H}$** | **mg/dL** | **2.30 - 4.70** |
| **ELECTROLYTES** | | | |
| SODIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)* | 137.6 | mmol/L | 135.0 - 150.0 |
| POTASSIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)* | 4.61 | mmol/L | 3.50 - 5.00 |
| CHLORIDE: SERUM *by ISE (ION SELECTIVE ELECTRODE)* | 103.2 | mmol/L | 90.0 - 110.0 |
| **ESTIMATED GLOMERULAR FILTERATION RATE** | | | |
| ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM *by CALCULATED* | 5.8 | | |
| **NOTE 2** | RESULT RECHECKED TWICE | | |
| **ADVICE** | **KINDLY CORRELATE CLINICALLY** | | |

***INTERPRETATION:***
To differentiate between pre- and post renal azotemia.

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**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 haemorrhage.
4. High protein intake.
5. Impaired renal function plus
6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
7. Urine reabsorption (e.g. ureter colostomy)
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).

**ESTIMATED GLOMERULAR FILTERATION RATE:**

| CKD STAGE | DESCRIPTION | GFR ( mL/min/1.73m2 ) | ASSOCIATED FINDINGS |
|---|---|---|---|
| G1 | Normal kidney function | >90 | No proteinuria |
| G2 | Kidney damage with normal or high GFR | >90 | Presence of Protein , Albumin or cast in urine |
| G3a | Mild decrease in GFR | 60 -89 | |
| G3b | Moderate decrease in GFR | 30-59 | |

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| Test Name | | Value | Unit | Biological Reference interval |
|---|---|---|---|---|
| G4 | Severe decrease in GFR | 15-29 | | |
| G5 | Kidney failure | <15 | | |

COMMENTS:
1. Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
3. In patients, with eGFR creatinine between 45-59 ml/min/1.73 m2 (G3) and without any marker of Kidney damage, It is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fullfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
***7. A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).***
***ADVICE:***
KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated

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## ENDOCRINOLOGY

### INTACT PARATHYROID HORMONE (PTH)

| | | | |
|---|---|---|---|
| **INTACT PARATHROID HORMONE (PTH): SERUM**<br>***by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*** | **235.6$^{H}$** | **pg/mL** | **9.5 - 75.0** |

**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<br>*by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)* | 30.7 | ng/mL | DEFICIENCY: < 20.0<br>INSUFFICIENCY: 20.0 - 30.0<br>SUFFICIENCY: 30.0 - 100.0<br>TOXICITY: > 100.0 |

***INTERPRETATION:***

| | | |
|---|---|---|
| **DEFICIENT:** | < 20 | ng/mL |
| **INSUFFICIENT:** | 21 - 29 | ng/mL |
| **PREFFERED RANGE:** | 30 - 100 | ng/mL |
| **INTOXICATION:** | > 100 | ng/mL |

1.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.
2.25-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.
3.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 harmone (PTH).
4.Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

**DECREASED:**
1.Lack of sunshine exposure.
2.Inadequate intake, malabsorption (celiac disease)
3.Depressed Hepatic Vitamin D 25- hydroxylase activity
4.Secondary to advanced Liver disease
5.Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

**INCREASED:**
1. 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 hyperphophatemia.

**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 interefere with Vitamin D absorption.*

*** End Of Report ***

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