# 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** | **: Miss. VINNI CHOPRA** | | |
| **AGE/ GENDER** | : 20 YRS/FEMALE | **PATIENT ID** | : 1435573 |
| **COLLECTED BY** | : | **REG. NO./LAB NO.** | **: 122502110012** |
| **REFERRED BY** | : | **REGISTRATION DATE** | : 11/Feb/2025 09:53 AM |
| **BARCODE NO.** | : 12506950 | **COLLECTION DATE** | : 11/Feb/2025 10:16AM |
| **CLIENT CODE.** | : P.K.R JAIN HEALTHCARE INSTITUTE | **REPORTING DATE** | : 11/Feb/2025 03:29PM |
| **CLIENT ADDRESS** | : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **CLINICAL CHEMISTRY/BIOCHEMISTRY** | | | |
| **KIDNEY FUNCTION TEST (BASIC)** | | | |
| UREA: SERUM<br>*by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)* | 16.82 | mg/dL | 10.00 - 50.00 |
| CREATININE: SERUM<br>*by ENZYMATIC, SPECTROPHOTOMETERY* | 0.88 | mg/dL | 0.40 - 1.20 |
| BLOOD UREA NITROGEN (BUN): SERUM<br>*by CALCULATED, SPECTROPHOTOMETERY* | 7.86 | mg/dL | 7.0 - 25.0 |
| BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETERY* | **8.93**$^{L}$ | RATIO | 10.0 - 20.0 |
| UREA/CREATININE RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETERY* | 19.11 | RATIO | |
| URIC ACID: SERUM<br>*by URICASE - OXIDASE PEROXIDASE* | 4.65 | 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 COMPLETE PROFILE** | | | |
| SODIUM: SERUM<br>*by ISE (ION SELECTIVE ELECTRODE)* | 140.2 | mmol/L | 135.0 - 150.0 |
| POTASSIUM: SERUM<br>*by ISE (ION SELECTIVE ELECTRODE)* | 4.48 | mmol/L | 3.50 - 5.00 |
| CHLORIDE: SERUM<br>*by ISE (ION SELECTIVE ELECTRODE)* | 105.15 | 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 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

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| Test Name | Value | Unit | Biological Reference interval |
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| 4.Hemolysis of blood | | | |

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **LITHIUM** | | | |
| LITHIUM: SERUM<br>*by ISE (ION SELECTIVE ELECTRODE)* | 0.98 | mmol/L | 0.30 - 1.50 |

***INTERPRETATION:***

| | | |
|---|---|---|
| Therapeutic | mmol/L | 0.6 - 1.2 |
| Potentially toxic | mmol/L | 1.5 - 2.5 |
| Severely toxic | mmol/L | > 2.5 |

1.Serum Lithium levels are useful for monitoring therapy of patients with bipolar disorders, including recurrent episodes of mania and depression and for evaluation of toxicity

2.Lithium alters the intraneuronal metabolism of catecholamines by an unknown mechanism. It is used to suppress the manic phase of manic-depressive psychosis.

3.Lithium is distributed throughout the total water spaces of the body and is excreted primarily by the kidney.

4.Toxicity from lithium salts leads to ataxia, slurred speech, and confusion.

5.Since the concentration of lithium in the serum varies with the time after the dose, blood for lithium determination should be drawn at a standard time, preferably 8 to 12 hours after the last dose (trough values).

***NOTE:*-Values above 1.6 mmol/L (trough values) are generally considered toxic and are indicative of dose modulation.**

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## ENDOCRINOLOGY

### THYROID FUNCTION TEST: TOTAL

| | | | |
|---|---|---|---|
| TRIIODOTHYRONINE (T3): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 1.35 | ng/mL | 0.35 - 1.93 |
| THYROXINE (T4): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 8.35 | µgm/dL | 4.87 - 12.60 |
| THYROID STIMULATING HORMONE (TSH): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 2.47 | µIU/mL | 0.35 - 5.50 |

**3rd GENERATION, ULTRASENSITIVE**

***INTERPRETATION:***

*TSH levels are subject to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50%.Hence time of the day has influence on the measured serum TSH concentrations.* TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4)and triiodothyronine (T3).Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction(hyperthyroidism) of T4 and/or T3.

| CLINICAL CONDITION | T3 | T4 | TSH |
|---|---|---|---|
| Primary Hypothyroidism: | Reduced | Reduced | Increased (Significantly) |
| Subclinical Hypothyroidism: | Normal or Low Normal | Normal or Low Normal | High |
| Primary Hyperthyroidism: | Increased | Increased | Reduced (at times undetectable) |
| Subclinical Hyperthyroidism: | Normal or High Normal | Normal or High Normal | Reduced |

**LIMITATIONS:-**

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG),and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin , salicylates).
3. Serum T4 levels in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.
4. TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothyroidism , pregnancy , phenytoin therapy.

| TRIIODOTHYRONINE (T3) | | THYROXINE (T4) | | THYROID STIMULATING HORMONE (TSH) | |
|---|---|---|---|---|---|
| Age | Refferance Range (ng/mL) | Age | Refferance Range ( µg/dL) | Age | Reference Range ( µIU/mL) |
| 0 - 7 Days | 0.20 - 2.65 | 0 - 7 Days | 5.90 – 18.58 | 0 - 7 Days | 2.43 - 24.3 |
| 7 Days - 3 Months | 0.36 - 2.59 | 7 Days - 3 Months | 6.39 - 17.66 | 7 Days - 3 Months | 0.58 - 11.00 |
| 3 - 6 Months | 0.51 - 2.52 | 3 - 6 Months | 6.75 – 17.04 | 3 Days – 6 Months | 0.70 - 8.40 |
| 6 - 12 Months | 0.74 - 2.40 | 6 - 12 Months | 7.10 – 16.16 | 6 – 12 Months | 0.70 - 7.00 |

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| 1 - 10 Years | 0.92 - 2.28 | 1 - 10 Years | 6.00 - 13.80 | 1 – 10 Years | 0.60 - 5.50 |
| 11- 19 Years | 0.35 - 1.93 | 11 - 19 Years | 4.87- 13.20 | 11 – 19 Years | 0.50 – 5.50 |
| > 20 years (Adults) | 0.35 - 1.93 | > 20 Years (Adults) | 4.87 - 12.60 | > 20 Years (Adults) | 0.35– 5.50 |
| RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY ( µIU/mL) | | | | | |
| 1st Trimester | | | 0.10 – 2.50 | | |
| 2nd Trimester | | | 0.20 – 3.00 | | |
| 3rd Trimester | | | 0.30 – 4.10 | | |

**INCREASED TSH LEVELS:**

1.Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.
2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
3.Hashimotos thyroiditis
4.DRUGS: Amphetamines, iodine containing agents & dopamine antagonist.
5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

**DECREASED TSH LEVELS:**

1.Toxic multi-nodular goiter & Thyroiditis.
2.Over replacement of thyroid hormone in treatment of hypothyroidism.
3.Autonomously functioning Thyroid adenoma
4.Secondary pituitary or hypothalamic hypothyroidism
5.Acute psychiatric illness
6.Severe dehydration.
7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
8.Pregnancy: 1st and 2nd Trimester

*** End Of Report ***

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