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| | | | |
|---|---|---|---|
| **NAME** | **: Mr. VINOD KALIA** | | |
| **AGE/ GENDER** | : 70 YRS/MALE | **PATIENT ID** | : 1724173 |
| **COLLECTED BY** | : | **REG. NO./LAB NO.** | **: 012501300002** |
| **REFERRED BY** | : | **REGISTRATION DATE** | : 30/Jan/2025 07:38 AM |
| **BARCODE NO.** | : 01524633 | **COLLECTION DATE** | : 30/Jan/2025 07:43AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 30/Jan/2025 09:45AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## HAEMATOLOGY

### ERYTHROCYTE SEDIMENTATION RATE (ESR)

| | | | |
|---|---|---|---|
| ERYTHROCYTE SEDIMENTATION RATE (ESR)<br>*by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY* | 12 | mm/1st hr | 0 - 20 |

**INTERPRETATION:**

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

**CONDITION WITH LOW ESR**

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis) , and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

**NOTE:**

1. ESR and C - reactive protein (C-RP) are both markers of inflammation.
2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
3. **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.**
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it

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| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 30/Jan/2025 10:51AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## CLINICAL CHEMISTRY/BIOCHEMISTRY

### GLUCOSE FASTING (F) AND POST PRANDIAL (PP)

| | | | |
|---|---|---|---|
| GLUCOSE FASTING (F): PLASMA<br>*by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)* | **121.88$^{H}$** | mg/dL | NORMAL: < 100.0<br>PREDIABETIC: 100.0 - 125.0<br>DIABETIC: > 0R = 126.0 |

***INTERPRETATION:***

**IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:**

1. A fasting plasma glucose below 100 mg/dL and post-prandial plasma glucose level below 140 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl and post-prandial plasma glucose level between 140 – 200 mg/dL is considered as glucose intolerant or pre diabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dL and post-prandial plasma glucose level 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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| **BARCODE NO.** | : 01524633 | **COLLECTION DATE** | : 30/Jan/2025 07:43AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 30/Jan/2025 11:03AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## ENDOCRINOLOGY

### THYROID FUNCTION TEST: TOTAL

| | | | |
|---|---|---|---|
| TRIIODOTHYRONINE (T3): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 1.115 | ng/mL | 0.35 - 1.93 |
| THYROXINE (T4): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 8.08 | µgm/dL | 4.87 - 12.60 |
| THYROID STIMULATING HORMONE (TSH): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 1.965 | 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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| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | | | Value | Unit | Biological Reference interval |
|---|---|---|---|---|---|
| 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

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| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 30/Jan/2025 01:29PM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## ANGIOTENSIN CONVERTING ENZYME (ACE): SERUM

| | | | |
|---|---|---|---|
| ANGIOTENSIN CONVERTNG ENZYME (ACE): SERUM<br>*by FURYLACRYLOYLPHENYLALANYGLYCYLGLYCINE (FAPPG)* | **54.5$^{H}$** | U/L | 8.0 - 52.0 |

***INTERPRETATION***

1.Angiotensin converting Enzyme (ACE) also known as kinase II, is present in many cells types such as neuronal cells, renal proximal tubular cells, and mostly in endothelial cells.
2.Angiotensin converting enzyme ( ACE) modulates peripheral vascular resistance as well as renal and cardiovascular function. It is responsible for conversion of Angiotensin I to Angiotensin II as well as inactivation of bradykinin
3.It is attached to endothelial surface membrane by an anchor peptide and can be cleaved to be released into the blood circulation as soluble enzyme. Serum ACE activity is significantly elevated in patients with untreated active disease.
4.Majority of ACE is tissue bound (> 90%) found predominantly in lungs & testes
5.It has been established as an important diagnostic parameter in Sarcoidosis. Spontaneous or induced remission of sarcoidosis has been seen, by decreasing serum ACE values.

**FACTORS AFFECTING ACE LEVELS:**

1.Smoking – ACE activity is 30% lower in smokers
2.Thyroid hormone- Stimulates ACE synthesis
3.Postmenopausal estrogen replacement – ACE activity is 20% lower

**INCREASED LEVELS:**

1.Sarcoidosis – ACE levels are used in the diagnosis and monitoring of this disease and are directly related to the number of organs affected and activity of granulomas. Mature granulomas produce less ACE than developing ones. ACE is more likely to be elevated with pulmonary involvement than with purely hilar adenopathy.
2.Pulmonary causes like Emphysema, Asthma, Small cell carcinoma & Squamous cell carcinoma, Idiopathic pulmonary fibrosis
3.Renal diseases – patients on hemodialysis show high ACE levels as compared to patients who are not on dialysis, chronic renal failure
4.Other causes – Multiple sclerosis, Addison's disease, Hyperthyroidism, Diabetes Alcoholic hepatitis &cirrohosis & Peptic ulcer,histoplasmosis, hodgkins disease, gauchers disease, leprosy, amyloidosis, tuberculosis
5.Elevated ACE is thought to be a risk factor for myocardial infarction & cardiomyopathy.
7.ACE inhibitors have found wide spread application in treatment of systemic hypertension and Congestive Heart Failure (CHF). Monitoring of ACE may be beneficial to determine the optimum low dose of ACE inhibitor.

**DECREASED LEVELS**

1.Chronic liver disease.
2.Anorexia nervosa
3.Hypothyroidism

*To be correlated clinically*

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| **BARCODE NO.** | : 01524633 | **COLLECTION DATE** | : 30/Jan/2025 07:43AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 31/Jan/2025 06:02AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## IMMUNOPATHOLOGY/SEROLOGY

### ANTI NUCLEAR ANTIBODY/FACTOR (ANA/ANF)

| | | | |
|---|---|---|---|
| ANTI NUCLEUR ANTIBODIES (ANA): SERUM<br>*by ELISA (ENZYME LINKED IMMUNOASSAY)* | **1.46$^{H}$** | INDEX VALUE | NEGATIVE: < 1.0<br>BORDERLINE: 1.0 - 1.20<br>POSITIVE: > 1.20 |

***INTERPRETATION:-***

1.For diagnostic purposes, ANA value should be used as an adjuvant to other clinical and laboratory data available.
2.Measurement of antinuclear antibodies (ANAs) in serum is the most commonly performed screening test for patients suspected of having a systemic rheumatic disease, also referred to as connective tissue disease.
3.ANAs occur in patients with a variety of autoimmune diseases, both systemic and organ-specific. They are particularly common in the systemic rheumatic diseases, which include lupus erythematosus (LE), discoid LE, drug-induced LE, mixed connective tissue disease, Sjogren syndrome, scleroderma (systemic sclerosis), CREST (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia) syndrome, polymyositis/dermatomyositis, and rheumatoid arthritis.

**NOTE:**

1.The diagnosis of a systemic rheumatic disease is based primarily on the presence of compatible clinical signs and symptoms.
The results of tests for autoantibodies including ANA and specific autoantibodies are ancillary. Additional diagnostic criteria include consistent histopathology or specific radiographic findings. Although individual systemic rheumatic diseases are relatively uncommon, a great many patients present with clinical findings that are compatible with a systemic rheumatic disease ANA screening may be useful for ruling out the disease.
2.Secondary, disease specific auto antibodies maybe ordered for patients who are screen positive as ancillary aids for the diagnosis of specific auto-immune disorders.

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| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 30/Jan/2025 11:11AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## C-REACTIVE PROTEIN (CRP)

| | | | |
|---|---|---|---|
| C-REACTIVE PROTEIN (CRP) QUANTITATIVE: SERUM<br>*by NEPHLOMETRY* | 0.3 | mg/L | 0.0 - 6.0 |

***INTERPRETATION:***

1. C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.
2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant rejection, and to monitor these inflammatory processes.
4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,
5. Elevated values are consistent with an acute inflammatory process.

**NOTE:**

1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
2. Oral contraceptives may increase CRP levels.

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

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## VITAMINS

### VITAMIN B12/COBALAMIN

| | | | |
|---|---|---|---|
| VITAMIN B12/COBALAMIN: SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | **> 2000$^{H}$** | pg/mL | 190.0 - 890.0 |

***INTERPRETATION:-***

| INCREASED VITAMIN B12 | DECREASED VITAMIN B12 |
|---|---|
| 1.Ingestion of Vitamin C | 1.Pregnancy |
| 2.Ingestion of Estrogen | 2.DRUGS:Aspirin, Anti-convulsants, Colchicine |
| 3.Ingestion of Vitamin A | 3.Ethanol Igestion |
| 4.Hepatocellular injury | 4. Contraceptive Harmones |
| 5.Myeloproliferative disorder | 5.Haemodialysis |
| 6.Uremia | 6. Multiple Myeloma |

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.
**NOTE:**A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

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| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 30/Jan/2025 10:31AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## CLINICAL PATHOLOGY

### BENCE JONES PROTEINS (QUALITATIVE) - RANDOM URINE

| | | | |
|---|---|---|---|
| **URINE FOR BENCE JONES PROTEINS** | NEGATIVE | | NEGATIVE |

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| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 30/Jan/2025 02:47PM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **SPECIAL INVESTIGATIONS** | | | |
| **PROTEIN ELECTROPHORESIS: SERUM** | | | |
| TOTAL PROTEINS: SERUM *by MIGRATION GEL ELECTROPHORESIS* | 6.84 | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM *by MIGRATION GEL ELECTROPHORESIS* | 4.12 | gm/dL | 3.50 - 5.50 |
| A : G RATIO: SERUM *by MIGRATION GEL ELECTROPHORESIS* | 1.52 | RATIO | 1.00 - 2.00 |
| ALPHA 1 GLOBULIN *by MIGRATION GEL ELECTROPHORESIS* | 0.24 | gm/dL | 0.11 - 0.40 |
| ALPHA 2 GLOBULIN *by MIGRATION GEL ELECTROPHORESIS* | 0.8 | gm/dL | 0.43 - 1.03 |
| BETA GLOBULIN *by MIGRATION GEL ELECTROPHORESIS* | 0.75 | mg/dL | 0.53 - 1.40 |
| GAMMA GLOBULIN *by MIGRATION GEL ELECTROPHORESIS* | 0.93 | gm/dL | 0.75 - 1.80 |
| **INTERPRETATION** | Protein electrophoresis shows normal pattern. Kindly correlate clinically. | | |

***INTERPRETATION:***

1.Serum protein electrophoresis is commonly used to identify patients with multiple myeloma and disorders of serum proteins.
2.Electrophoresis is a method of separating proteins based on their physical properties. the pattern of serum protein electrophoresis results depends on the frations of 2 types of protein : albumin and globulin (alpha 1 alpha2, beta and gamma.)
3.A homogeneous spike-like peak in a focal region of the gamma-globulin zone indicates a monoclonal gammopathy.
4.Monoclonal gammopathies are associated with a clonal process that is malignant or potentially malignant, including multiple myeloma, Waldenstrom macroglobulinemia, solitary plasmacytoma, smoldering multiple myeloma, monoclonal gammopathy of undetermined significance, plasma cell leukemia, heavy chain disease, and amyloidosis.
5.M-protein (in the gamma region) level greater than 3 g/dL should be interpreted along with other radiologic and haematological findings to arrive at a diagnosis of Multiple myeloma and must not be considered in isolation.
6.Occasionally M protein may appear as a narrow spike in the beta or alpha2 regions also.
7.Up to one fifth of patients with Myeloma may have an M-protein spike of less than 1 g /dL.
8.Hypogammaglobulinemia on serum protein electrophoresis occurs in about 10% of patients with multiple myeloma who do not have a serum M-protein spike.
9.Most of these patients have a large amount of Bence Jones protein (monoclonal free kappa or lambda chain) in their urine, wherein urine protein electrophoresis should be performed. Monoclonal gammopathy is present in up to 8 percent of healthy geriatric patients.

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Chairman & Consultant Pathologist

Dr. Yugam Chopra
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CEO & Consultant Pathologist

| | | | |
|---|---|---|---|
| **NAME** | **: Mr. VINOD KALIA** | | |
| **AGE/ GENDER** | : 70 YRS/MALE | **PATIENT ID** | : 1724173 |
| **COLLECTED BY** | : | **REG. NO./LAB NO.** | **: 012501300002** |
| **REFERRED BY** | : | **REGISTRATION DATE** | : 30/Jan/2025 07:38 AM |
| **BARCODE NO.** | : 01524633 | **COLLECTION DATE** | : 30/Jan/2025 07:43AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 30/Jan/2025 02:47PM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

**NOTE:**
The following conditions require serum immunofixation to confirm monoclonality or to differentiate monoclonal and polyclonal disoders.
1.A well defined "M" band.
2.Faint band .
3.Chronic inflammatory pattern (decreased albumin, increased alpha, increased gamma fractions)
4.Isolated increase in any region with an otherwise normal pattern.
5.Shouldering of albumin peak along anodal or cathodal side may be seen with lipoproteins, drugs, bilirubin or radiological contrast.

*** End Of Report ***

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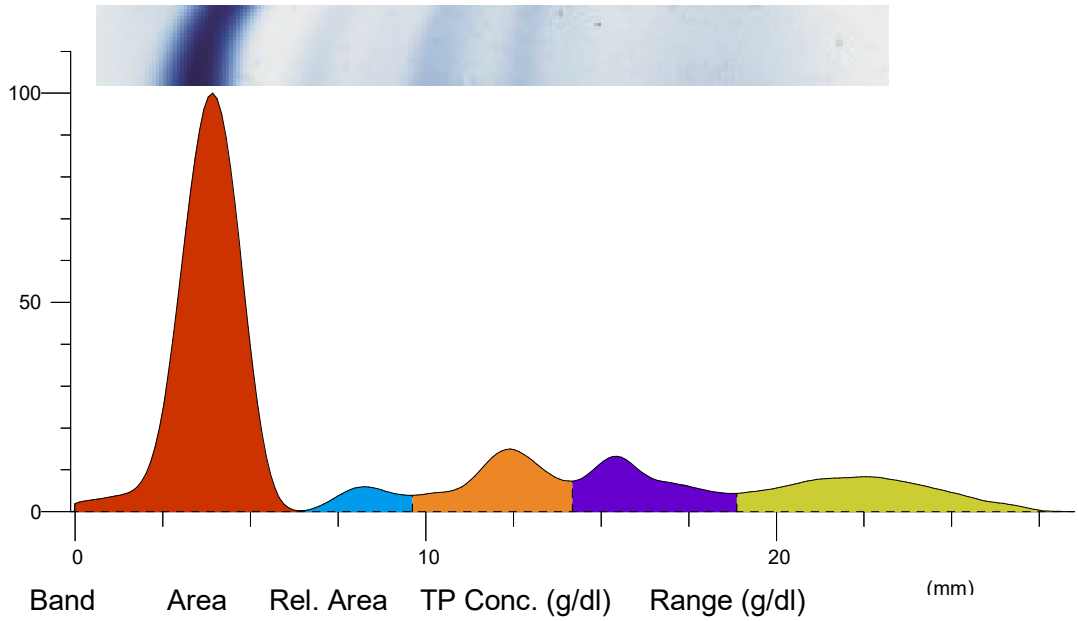
# KOS Diagnostic Lab

**(A Unit of KOS Healthcare)**

## PROTEIN ELECTROPHORESIS

| | | | |
|---|---|---|---|
| **NAME** | **VINOD KALIA** | **BARCODE ID** | **01524633** |
| **AGE/SEX** | **70 YRS/M** | **DATE** | **30-01-2025** |

**Chemistry Results** **TP 6.84(g/dl)**




| Index | Band | Area | Rel. Area | TP Conc. (g/dl) | Range (g/dl) |
|---|---|---|---|---|---|
| 1 | Albumin | 1.148 | 60.30% | 4.12 | 3.50 ... 5.00 |
| 2 | Alpha 1 | 0.066 | 3.49% | 0.24 | 0.11 ... 0.40 |
| 3 | Alpha 2 | 0.224 | 11.76% | 0.80 | 0.43 ... 1.03 |
| 4 | Beta | 0.207 | 10.90% | 0.75 | 0.53 ... 1.40 |
| 5 | Gamma | 0.258 | 13.55% | 0.93 | 0.75 ... 1.80 |
| Total | | 1.903 | | 6.84 | |

Ratio A/G 1.52

**Comment:-**

Protein electrophoresis shows normal pattern. Kindly correlate clinically.

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**MD (Pathology and Microbiology)**

**Dr Yugam Chopra**
**MD (Pathology**