

# **KOS Diagnostic Lab**

(A Unit of KOS Healthcare)



Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist

**NAME** : Mr. DEEPAK MEHTA

**AGE/ GENDER** : 74 YRS/MALE **PATIENT ID** : 1651142

**COLLECTED BY** : 012410230046 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 23/Oct/2024 12:29 PM BARCODE NO. :01519427 **COLLECTION DATE** : 23/Oct/2024 12:30PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 23/Oct/2024 01:47PM

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

**Value** Unit **Biological Reference interval Test Name** 

# **CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE RANDOM (R)**

GLUCOSE RANDOM (R): PLASMA NORMAL: < 140.00 342.21<sup>H</sup> mg/dL

by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) PREDIABETIC: 140.0 - 200.0

DIABETIC: > 0R = 200.0

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-prnadial 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.



CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST





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**CREATININE** 

CREATININE: SERUM
by ENZYMATIC, SPECTROPHOTOMETRY

2.19<sup>H</sup> mg/dL 0.40 - 1.40

Rechecked



DR.VINAY CHOPRA
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#### **URIC ACID**

URIC ACID: SERUM 6.96 mg/dL 3.60 - 7.70

by URICASE - OXIDASE PEROXIDASE

## **INTERPRETATION:-**

1.GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint

2.Uric Acid is the end product of purine metabolism. Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

#### INCREASED:-

## (A).DUE TO INCREASED PRODUCTION:-

1.Idiopathic primary gout.

2. Excessive dietary purines (organ meats, legumes, anchovies, etc).

3. Cytolytic treatment of malignancies especially leukemais & lymphomas.

4. Polycythemai vera & myeloid metaplasia.

5. Psoriasis.

6. Sickle cell anaemia etc.

#### (B).DUE TO DECREASED EXCREATION (BY KIDNEYS)

1. Alcohol ingestion.

2. Thiazide diuretics.

3.Lactic acidosis.

4. Aspirin ingestion (less than 2 grams per day ).

5. Diabetic ketoacidosis or starvation.

6.Renal failure due to any cause etc.

### DECREASED:-

#### (A).DUE TO DIETARY DEFICIENCY

1. Dietary deficiency of Zinc, Iron and molybdenum.

2. Fanconi syndrome & Wilsons disease.

3. Multiple sclerosis

4. Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

## (B) DUE TO INCREASED EXCREATION

1.Drugs:-Probenecid, sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosterroids and ACTH, anti-coagulants and estrogens etc.



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End Of Report



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