

(A Unit of KOS Healthcare)



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

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

**NAME** : Mrs. SUMAN PURI

**AGE/ GENDER** : 65 YRS/FEMALE **PATIENT ID** : 1642411

**COLLECTED BY** : SURJESH REG. NO./LAB NO. :012410140019

REFERRED BY **REGISTRATION DATE** : 14/Oct/2024 09:08 AM BARCODE NO. :01518862 **COLLECTION DATE** : 14/Oct/2024 09:25AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 14/Oct/2024 09:47AM

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

Test Name Value Unit **Biological Reference interval** 

## **HAEMATOLOGY HAEMOGLOBIN (HB)**

HAEMOGLOBIN (HB) 12.0 - 16.0 11.3<sup>L</sup> qm/dL

by CALORIMETRIC

**INTERPRETATION:-**

Hemoglobin is the protein molecule in red blood cells that carries oxygen from the lungs to the bodys tissues and returns carbon dioxide from the tissues back to the lungs.

A low hemoglobin level is referred to as ANEMIA or low red blood count.

ANEMIA (DECRESED HAEMOGLOBIN):

- 1) Loss of blood (traumatic injury, surgery, bleeding, colon cancer or stomach ulcer)
- 2) Nutritional deficiency (iron, vitamin B12, folate)
- 3) Bone marrow problems (replacement of bone marrow by cancer)
- 4) Suppression by red blood cell synthesis by chemotherapy drugs
- 5) Kidney failure
- 6) Abnormal hemoglobin structure (sickle cell anemia or thalassemia).

### POLYCYTHEMIA (INCREASED HAEMOGLOBIN):

- 1) People in higher altitudes (Physiological)
- 2) Smoking (Secondary Polycythemia)
- 3) Dehydration produces a falsely rise in hemoglobin due to increased haemoconcentration
- 4) Advanced lung disease (for example, emphysema)
- 5) Certain tumors
- 6) A disorder of the bone marrow known as polycythemia rubra vera,
- 7) Abuse of the drug erythropoetin (Epogen) by athletes for blood doping purposes (increasing the amount of oxygen available to the body by chemically raising the production of red blood cells).

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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# **CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE FASTING (F)**

97.89 GLUCOSE FASTING (F): PLASMA mg/dL NORMAL: < 100.0

by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

INTERPRETATION
IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.

2. A fasting plasma glucose level between 100 - 125 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 fasting plasma glucose level of above 125 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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**URIC ACID** 

URIC ACID: SERUM 7.51<sup>H</sup> mg/dL 2.50 - 6.80

by URICASE - OXIDASE PEROXIDASE

NTERPRETATION:-

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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### **ENDOCRINOLOGY**

## THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 0.744 μIU/mL 0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

#### **INTERPRETATION:**

AGE	REFFERENCE RANGE (μIU/mL)
0 – 5 DAYS	0.70 - 15.20
6 Days – 2 Months	0.70 - 11.00
3 – 11 Months	0.70 - 8.40
1 – 5 Years	0.70 - 7.00
6 – 10 Years	0.60 - 5.50
11 - 15	0.50 - 5.50
> 20 Years (Adults)	0.27 - 5.50
PREC	GNANCY
1st Trimester	0.10 - 3.00
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

NOTE:-TSH levels are subjected to circardian 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 concentration.

**USE**:- TSH controls biosynthesis and release of thyroid harmones T4 & T3. It is a sensitive measure of thyroid function, especially useful in early or subclinical hypothyroidism, before the patient develops any clinical findings or goitre or any other thyroid function abnormality.

#### INCREASED LEVELS:

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

#### **DECREASED LEVELS:**

- 1.Toxic multi-nodular goitre & Thyroiditis.
- 2. Over replacement of thyroid harmone in treatment of hypothyroidism.
- 3. Autonomously functioning Thyroid adenoma
- 4. Secondary pituatary or hypothalmic hypothyroidism
- 5. Acute psychiatric illness
- 6.Severe dehydration.



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7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

8. Pregnancy: 1st and 2nd Trimester

### LIMITATIONS:

1.TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.

2. Autoimmune disorders may produce spurious results.

\*\*\* End Of Report \*\*



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