

(A Unit of KOS Healthcare)



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

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

NAME : Mrs. RAMAN SEHGAL

AGE/ GENDER : 69 YRS/FEMALE **PATIENT ID** : 1635989

: SURJESH **COLLECTED BY** : 012410060020 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 06/Oct/2024 10:32 AM BARCODE NO. :01518401 **COLLECTION DATE** : 06/Oct/2024 10:44AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE :06/Oct/2024 12:32PM

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

Test Name Value Unit **Biological Reference interval**

HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): % 6.7^H 4.0 - 6.4

WHOLE BLOOD

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE

145.59^H mg/dL 60.00 - 140.00 by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

INTERPRETATION:

AS PER AMERICAN D	IABETES ASSOCIATION (ADA):	
REFERENCE GROUP	GLYCOSYLATED HEMOGLOGIB (HBAIC) in %	
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.4	
Diagnosing Diabetes	>= 6.5	
Therapeutic goals for glycemic control	Age > 19 Years	
	Goals of Therapy:	< 7.0
	Actions Suggested:	>8.0
	Age < 19 Years	
	Goal of therapy:	<7.5

COMMENTS:

- 1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients. 2. Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.
- 3. Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be
- 4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications 5. Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.
- 6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution, given the pathological processes including anemia,increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7. Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells



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CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE FASTING (F)

78.63 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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CEO & Consultant Pathologist

NAME : Mrs. RAMAN SEHGAL

by ENZYMATIC, SPECTROPHOTOMETRY

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CREATININE

CREATININE: SERUM 1.81^H mg/dL 0.40 - 1.20



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URIC ACID

URIC ACID: SERUM 6.49 mg/dL 2.50 - 6.80

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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ELECTROLYTES COMPLETE PROFILE

SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	140.3	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.56	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	105.23	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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4.Hemolysis of blood

End Of Report *



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