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 Chairman & Consultant Pathologist

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<b>NAME</b>	: Mr. JAGDISH KUMAR	<b>PATIENT ID</b>	: 1662783
<b>AGE/ GENDER</b>	: 60 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 012411060021
<b>COLLECTED BY</b>	: SURJESH	<b>REGISTRATION DATE</b>	: 06/Nov/2024 10:05 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 06/Nov/2024 10:24AM
<b>BARCODE NO.</b>	: 01520209	<b>REPORTING DATE</b>	: 06/Nov/2024 03:08PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
<b>CLIENT ADDRESS</b>	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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### HAEMATOLOGY

#### GLYCOSYLATED HAEMOGLOBIN (HbA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	7.3 <sup>H</sup>	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	162.81 <sup>H</sup>	mg/dL	60.00 - 140.00

#### INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):		
REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HbA1C) 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:

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- 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 HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- 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, targeting a goal of < 7.0% may not be appropriate.
- High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shortens RBC life span like acute blood loss, hemolytic anemia falsely lowers HbA1c results.
- 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 glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.





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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	<b>189.99<sup>H</sup></b>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
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#### 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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UREA  
UREA: SERUM 95.02<sup>H</sup> mg/dL 10.00 - 50.00  
by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)



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CREATININE

CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	2.17 <sup>H</sup>	mg/dL	0.40 - 1.40
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### URIC ACID

URIC ACID: SERUM	<b>10.07<sup>H</sup></b>	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 leukemias & lymphomas.
4. Polycythemia vera & myeloid metaplasia.
5. Psoriasis.
6. Sickle cell anaemia etc.

##### (B).DUE TO DECREASED EXCRETION (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 EXCRETION

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

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





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