



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961 ✉ pkrajainhealthcare@gmail.com

NAME : Mr. SANJEEV
AGE/ GENDER : 48 YRS/MALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12506631
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1731218
REG. NO./LAB NO. : 122501220016
REGISTRATION DATE : 22/Jan/2025 12:08 PM
COLLECTION DATE : 22/Jan/2025 12:13PM
REPORTING DATE : 22/Jan/2025 03:47PM

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HbA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): **8.8^H** % 4.0 - 6.4
by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)
ESTIMATED AVERAGE PLASMA GLUCOSE **205.86^H** mg/dL 60.00 - 140.00
by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

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

KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	30.51	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	1.32	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	14.26	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	10.8	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	23.11	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	3.4 ^L	mg/dL	3.60 - 7.70




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INTERPRETATION:

Normal range for a healthy person on normal diet: 12 - 20

To Differentiate between pre- and postrenal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased glomerular filtration rate.
2. Catabolic states with increased tissue breakdown.
3. GI hemorrhage.
4. High protein intake.
5. Impaired renal function plus .
6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushings syndrome, high protein diet, burns, surgery, cachexia, high fever).
7. Urine reabsorption (e.g. ureterocolostomy)
8. Reduced muscle mass (subnormal creatinine production)
9. Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
2. Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN :

1. Acute tubular necrosis.
2. Low protein diet and starvation.
3. Severe liver disease.
4. Other causes of decreased urea synthesis.
5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
6. Inherited hyperammonemias (urea is virtually absent in blood).
7. SIADH (syndrome of inappropriate antidiuretic hormone) due to tubular secretion of urea.
8. Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:


1. Phenacimide therapy (accelerates conversion of creatine to creatinine).
2. Rhabdomyolysis (releases muscle creatinine).
3. Muscular patients who develop renal failure.


INAPPROPRIATE RATIO:

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
2. Cephalosporin therapy (interferes with creatinine measurement).

*** End Of Report ***




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