Dr. Vinay Chopra
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Chairman & Consultant Pathologist

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

NAME	**: Mr. NARINDER KUMAR**		
AGE/ GENDER	: 66 YRS/MALE	**PATIENT ID**	: 1667841
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012502040001**
REFERRED BY	:	**REGISTRATION DATE**	: 04/Feb/2025 06:03 AM
BARCODE NO.	: 01524903	**COLLECTION DATE**	: 04/Feb/2025 06:04AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 04/Feb/2025 06:51AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	**6.9**^{H}	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	**151.33**^{H}	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES 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 HbAIc. 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 appropiate.

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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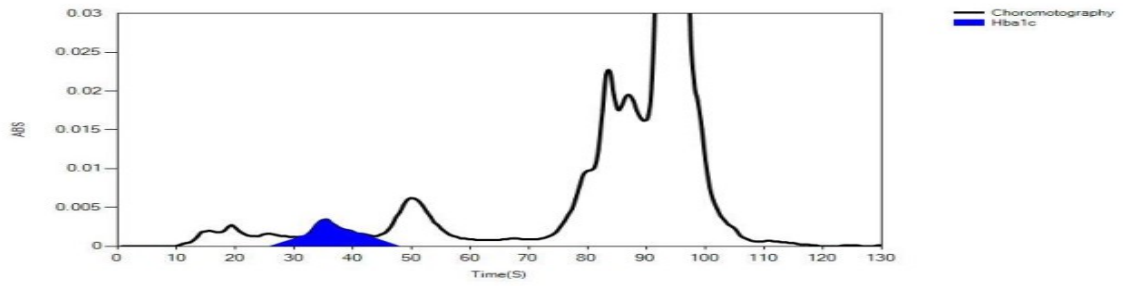
Test Name	Value	Unit	Biological Reference interval

LIFOTRONIC Graph Report

Name :	Case :	Patient Type :		Test Date : 04/02/2025 09:12:45
Age :	Department :	Sample Type : Whole Blood EDTA		Sample Id : 01524903
Gender :				Total Area : 8175

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	69	2202	7130	82.1
HbA1c	37	63	602	6.9
La1c	26	34	241	2.8
HbF	18	16	25	0.3
Hba1b	14	27	102	1.2
Hba1a	11	20	75	0.9



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 04/Feb/2025 12:15PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
CLINICAL CHEMISTRY/BIOCHEMISTRY			
KIDNEY FUNCTION TEST (BASIC)			
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	41.51	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	1.32	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETERY*	19.4	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	14.7	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	31.45	RATIO	
URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	3.82	mg/dL	3.60 - 7.70

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Test Name	Value	Unit	Biological Reference interval

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 inappropiate antidiuretic harmone) 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.

INAPPROPIATE 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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