



# P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

**A PIONEER DIAGNOSTIC CENTRE**

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

**NAME** : Mr. KRISHAN LAL  
**AGE/ GENDER** : 78 YRS/MALE  
**COLLECTED BY** :  
**REFERRED BY** :  
**BARCODE NO.** : 12505328  
**CLIENT CODE.** : P.K.R JAIN HEALTHCARE INSTITUTE  
**CLIENT ADDRESS** : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

**PATIENT ID** : 1368574  
**REG. NO./LAB NO.** : 122410240015  
**REGISTRATION DATE** : 24/Oct/2024 12:23 PM  
**COLLECTION DATE** : 24/Oct/2024 01:04PM  
**REPORTING DATE** : 24/Oct/2024 04:20PM

Test Name	Value	Unit	Biological Reference interval
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## HAEMATOLOGY

### GLYCOSYLATED HAEMOGLOBIN (HbA1c)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): **7.2<sup>H</sup>** % 4.0 - 6.4  
by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)  
ESTIMATED AVERAGE PLASMA GLUCOSE **159.94<sup>H</sup>** 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.



  
DR. VINAY CHOPRA  
CONSULTANT PATHOLOGIST  
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

  
DR. YUGAM CHOPRA  
CONSULTANT PATHOLOGIST  
MBBS, MD (PATHOLOGY)





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA  
by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)

88.6

mg/dL

NORMAL: < 100.0  
PREDIABETIC: 100.0 - 125.0  
DIABETIC: > OR = 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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MBBS, MD (PATHOLOGY)





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<b>LIPID PROFILE : BASIC</b>			
CHOLESTEROL TOTAL: SERUM <i>by CHOLESTEROL OXIDASE PAP</i>	125.57	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM <i>by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)</i>	98.16	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM <i>by SELECTIVE INHIBITION</i>	72.74	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	33.2	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	52.83	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	19.63	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	<b>349.3<sup>L</sup></b>	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.73	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0



  
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LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.46 <sup>L</sup>	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.35 <sup>L</sup>	RATIO	3.00 - 5.00

**INTERPRETATION:**

1. Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL.
5. Additional testing for Apolipoprotein B, hsCRP, Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement



  
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## KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	44.24	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	0.91	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	20.67	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	22.71 <sup>H</sup>	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	48.62	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	4.34	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).



  
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## ENDOCRINOLOGY

### THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 5.21  $\mu$ IU/mL 0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

#### INTERPRETATION:

AGE	REFERENCE RANGE ( $\mu$ IU/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
PREGNANCY	
1st Trimester	0.10 - 3.00
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

**NOTE:-** TSH levels are subjected to circadian 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 hormones 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 hormone in treatment of hypothyroidism.
- 3.Autonomously functioning Thyroid adenoma
- 4.Secondary pituitary or hypothalamic hypothyroidism
- 5.Acute psychiatric illness
- 6.Severe dehydration.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.



  
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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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