



# P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

**A PIONEER DIAGNOSTIC CENTRE**

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

**NAME** : Mr. GURJOT SINGH  
**AGE/ GENDER** : 22 YRS/MALE **PATIENT ID** : 1720744  
**COLLECTED BY** : **REG. NO./LAB NO.** : 122501100004  
**REFERRED BY** : **REGISTRATION DATE** : 10/Jan/2025 11:19 AM  
**BARCODE NO.** : 12506472 **COLLECTION DATE** : 10/Jan/2025 11:21AM  
**CLIENT CODE.** : P.K.R JAIN HEALTHCARE INSTITUTE **REPORTING DATE** : 10/Jan/2025 12:47PM  
**CLIENT ADDRESS** : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

Test Name	Value	Unit	Biological Reference interval
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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.71	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.28	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.43	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	20.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	15.83	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.31	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	83.88	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	9.87	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.49	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.26	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.23 <sup>L</sup>	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.91	RATIO	1.00 - 2.00

#### INTERPRETATION


**NOTE:-** To be correlated in individuals having SGOT and SGPT values higher than Normal Reference Range.


**USE:-** Differential diagnosis of diseases of hepatobiliary system and pancreas.

#### INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5



  
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HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS			> 1.3 (Slightly Increased)


**DECREASED:**


1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestasis: 0.8 (normal or slightly decreased).

**PROGNOSTIC SIGNIFICANCE:**

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6



  
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### KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	44.36	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	0.72	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	20.73	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	28.79 <sup>H</sup>	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	61.61	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	3.29 <sup>L</sup>	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 TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	6.66	ng/mL	0.47 - 9.80
by CMA (CHEMILUMINESCENT PARTICLE IMMUNOASSAY)			

### INTERPRETATION:

- Testosterone is secreted in females by the ovary and formed indirectly from androstenedione in adrenal glands.
- In males it is secreted by the testes. It circulates in blood bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form.
- The bioavailable fraction includes the free form and that "weakly bound" to albumin (40% of the total in men and 20% of the total in women) and bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone.
- The total testosterone bound to SHBG fluctuates since SHBG levels are affected by medication, disease, sex steroids and insulin.

### CLINIC USE:

- Assessment of testicular functions in males
- Management of hirsutism and virilization in females

### INCREASED LEVELS:


- Precocious puberty (Males)
- Androgen resistance
- Testotoxicosis
- Congenital Adrenal Hyperplasia
- Polycystic ovarian disease
- Ovarian tumors


### DECREASED LEVELS:

- Delayed puberty (Males)
- Gonadotropin deficiency
- Testicular defects
- Systemic diseases

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



  
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