



# P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

**A PIONEER DIAGNOSTIC CENTRE**

☎ 0171-2532620, 8222896961 ✉ [pkrajainhealthcare@gmail.com](mailto:pkrajainhealthcare@gmail.com)

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	: Mrs. KANIKA ANAND	PATIENT ID	: 1563732
AGE/ GENDER	: 34 YRS/FEMALE	REG. NO./LAB NO.	: 122407290004
COLLECTED BY	:	REGISTRATION DATE	: 29/Jul/2024 08:57 AM
REFERRED BY	:	COLLECTION DATE	: 29/Jul/2024 09:40AM
BARCODE NO.	: 12503866	REPORTING DATE	: 29/Jul/2024 01:09PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE		
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>	0.78	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.25	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.53	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	72.15 <sup>H</sup>	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	115.75 <sup>H</sup>	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.62	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	123.42	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	41.99	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	7.05	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.47	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.58	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.73	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



*[Signature]*

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INTRAHEPATIC CHOLESTATIS	> 1.5		
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)		


**DECREASED:**


- Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
- 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)	17.82	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	0.51	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	8.33	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	16.33	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	34.94	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	4.84	mg/dL	2.50 - 6.80



  
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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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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

**VITAMIN D (25-HYDROXY VITAMIN D3): SERUM**  
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

12.3<sup>L</sup>

ng/mL

**DEFICIENCY:** < 20.0  
**INSUFFICIENCY:** 20.0 - 30.0  
**SUFFICIENCY:** 30.0 - 100.0  
**TOXICITY:** > 100.0

#### INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFERRED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

- Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
- 25-OH--Vitamin D represents the main body reservoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
- Vitamin D plays a primary role in the maintenance of calcium homeostasis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

#### DECREASED:

- Lack of sunshine exposure.
- Inadequate intake, malabsorption (celiac disease)
- Depressed Hepatic Vitamin D 25- hydroxylase activity
- Secondary to advanced Liver disease
- Osteoporosis and Secondary Hyperparathyroidism (Mild to Moderate deficiency)
- Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

#### INCREASED:

- Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphosphatemia.

**CAUTION:** Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

**NOTE:**-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interfere with Vitamin D absorption.

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



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