



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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

NAME	: Mr. VINAYAK	PATIENT ID	: 1392713
AGE/ GENDER	: 26 YRS/MALE	REG. NO./LAB NO.	: 122502170007
COLLECTED BY	:	REGISTRATION DATE	: 17/Feb/2025 11:02 AM
REFERRED BY	:	COLLECTION DATE	: 17/Feb/2025 11:23AM
BARCODE NO.	: 12507053	REPORTING DATE	: 17/Feb/2025 12:46PM
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>	0.55	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>	0.27	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	28.44	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	33.76	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.84	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	87.8	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	17.78	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.46	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.49	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.97^L	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.28^H	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)	
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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)	25.87	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	1.23	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	12.09	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	9.83 ^L	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	21.03	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	3.93	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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IMMUNOPATHOLOGY/SEROLOGY

C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: 3.2 mg/L 0.0 - 6.0
SERUM

by NEPHLOMETRY

INTERPRETATION:

1. C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.
2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant rejection, and to monitor these inflammatory processes.
4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,
5. Elevated values are consistent with an acute inflammatory process.

NOTE:

1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
2. Oral contraceptives may increase CRP levels.



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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM 51.9 ng/mL
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)
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.



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CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

PHYSICAL EXAMINATION

QUANTITY RECEIVED	20	ml	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
COLOUR	PALE YELLOW		PALE YELLOW
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
TRANSPARANCY	CLEAR		CLEAR
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SPECIFIC GRAVITY	1.01		1.002 - 1.030
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			


CHEMICAL EXAMINATION


REACTION	NEUTRAL		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
PROTEIN	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SUGAR	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
pH	7		5.0 - 7.5
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
BILIRUBIN	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
NITRITE	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
UROBILINOGEN	NOT DETECTED	EU/dL	0.2 - 1.0
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
KETONE BODIES	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
BLOOD	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
ASCORBIC ACID	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			

MICROSCOPIC EXAMINATION

RED BLOOD CELLS (RBCs)	NEGATIVE (-ve)	/HPF	0 - 3
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by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS	3-4	/HPF	0 - 5
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
EPITHELIAL CELLS	2-3	/HPF	ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
CRYSTALS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
CASTS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
BACTERIA	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
OTHERS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
TRICHOMONAS VAGINALIS (PROTOZOA)	ABSENT		ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			

*** End Of Report ***




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