

(A Unit of KOS Healthcare)



Dr. Vinay Chopra
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NAME : Mrs. REKHA BHARDWAJ

AGE/ GENDER : 62 YRS/FEMALE PATIENT ID : 1658808

COLLECTED BY : REG. NO./LAB NO. : 012411020051

 REFERRED BY
 : 02/Nov/2024 02:09 PM

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 : 01519937
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 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 02/Nov/2024 03:20 PM

CLIENT ADDRESS: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.26	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.08	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.18	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	13.85	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	18.44	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.75	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para nitrophenyl phosphatase by amino methyl propanol	106.48	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	31.46	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	5.81 ^L	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	3.82	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.99 ^L	gm/dL	2.30 - 3.50
A: GRATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.92	RATIO	1.00 - 2.00

INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance 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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Test Name	Value	Unit	Biological Reference interval

REPORTING DATE

HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS > 1.3 (Slightly Increased)

CLIENT CODE.

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)

2. Extra Hepatic cholestatis: 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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Test Name	Value	Unit	Biological Reference interval			
KIDNEY FUNCTION TEST (BASIC)						
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	25.46	mg/dL	10.00 - 50.00			
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	1.13	mg/dL	0.40 - 1.20			
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETERY	11.9	mg/dL	7.0 - 25.0			
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETERY	10.53	RATIO	10.0 - 20.0			
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETERY	22.53	RATIO				
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	7.88 ^H	mg/dL	2.50 - 6.80			



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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 (pia (PLIN) rises dispreparties toly more than

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).



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Value Unit **Biological Reference interval Test Name**

IMMUNOPATHOLOGY/SEROLOGY ANTI CYCLIC CITRULLINATED PEPTIDE CCP2 (HIGHLY SENSITIVE)

ANTI CYCLIC CITRULLINATED PEPTIDE (CCP) 0.00 - 5.002.9 AU/mL

ANTIBODY: SERUM

by CMIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:

1. ANTI-CCP antibodies are potentially important surrogate marker for diagnosis and prognosis in rheumatoid arthritis (RA). 2. Anti-CCP is of two types: Anti-CCP1 & Anti-CCP2. 3. Anti-CCP2 is HIGHLY SENSITIVE (71%) & more specific (98%) than Anti-CCP1.

4. Anti-CCP2 predict the eventual development in Rheumatoid Arthritis (RA), when found in undifferentiated arthritis
5. Anti-CCP2 may be detected in healthy individual's years before onset of clinical Rheumatoid Arthritis as well as to differentiate elderly onset Rheumatoid Arthritis from Polymyalgia Rheumatoic & Erosive SLE.
6. The positive predictive value of Anti-CCP antibodies for Rheumatoid Arthritis is far greater than Rheumatoid factor. Up to 30% patients with

seronegative Rheumatoid Arthritis also show Anti CCP antibodies RHEUMATOID ARTHIRITIS:

1. Rheumatoid Arthritis is a systemic autoimmune disease that is multi-functional in origin and is characterized by chronic inflammation of the membrane lining (synovium) joints which leads to progressive joint destruction and in most cases to disability and reduction of quality life.

2. The disease spreads from small to large joints, with greatest damage in early phase.

3. The diagnosis of RA is primarily based on clinical, radiological & immunological features. The most frequent serological test is the measurement of RA factor

4. RA factor is not specific for rheumatoid arthritis, as it is often present in healthy individuals with other autoimmune diseases and chronic

5. ANTI-CCP have been discovered in joints of patients with RA, but not in other form of joint disease.



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C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: 9.35H 0.0 - 6.0mg/L

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.

*** End Of Report



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