



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	: Mr. DAYA RANI	PATIENT ID	: 1576479
AGE/ GENDER	: 61 YRS/MALE	REG. NO./LAB NO.	: 122408100020
COLLECTED BY	:	REGISTRATION DATE	: 10/Aug/2024 11:09 AM
REFERRED BY	:	COLLECTION DATE	: 10/Aug/2024 11:12AM
BARCODE NO.	: 12504096	REPORTING DATE	: 10/Aug/2024 01:34PM
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>	0.85	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.3	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.55	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	49.5 ^H	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	31.57	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.57	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	123.46	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	15.82	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.78	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.32	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.46	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.76	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



Chopra

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

Chopra

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





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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)	33.51	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	1.39	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	15.66	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	11.27 ^L	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	24.11	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	4.13	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 0.723 μ IU/mL 0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

AGE	REFERENCE RANGE (μ 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.



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7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

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.




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IMMUNOPATHOLOGY/SEROLOGY HEPATITIS C VIRUS (HCV) ANTIBODIES SCREENING

HEPATITIS C ANTIBODY (HCV) TOTAL
RESULT

NON - REACTIVE

by IMMUNOCHROMATOGRAPHY

INTERPRETATION:

1.Anti HCV total antibody assay identifies presence IgG antibodies in the serum . It is a useful screening test with a specificity of nearly 99%.
2.It becomes positive approximately 24 weeks after exposure. The test can not isolate an active ongoing HCV infection from an old infection that has been cleared. All positive results must be confirmed for active disease by an HCV PCR test .

FALSE NEGATIVE RESULTS SEEN IN:

- 1.Window period
- 2.Immunocompromised states.



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ANTI CYCLIC CITRULLINATED PEPTIDE CCP2 (HIGHLY SENSITIVE)


ANTI CYCLIC CITRULLINATED PEPTIDE (CCP) ANTIBODY: SERUM by CMIA (CHEMILUMINESCENCE IMMUNOASSAY)	< 0.5	AU/mL	0.00 - 5.00
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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 Rheumatic & 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 ARTHRITIS:**
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 infections.
 5. ANTI-CCP have been discovered in joints of patients with RA, but not in other form of joint disease.




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SJOGRENS SYNDROME ANTIBODY PROFILE (SS-A/ANTI - RO), (SS-B/ANTI - LA) - IgG

SS-A/RO ANTIBODY IgG QUANTITATIVE by EIA (ENZUME IMMUNO ASSAY)	<0.1	Index	< 1.0
SS-A/RO ANTIBODY IgG RESULT by EIA (ENZUME IMMUNO ASSAY)	NON - REACTIVE		NEGATIVE (-ve)
SS-B/LA ANTIBODY IgG QUANTITATIVE by EIA (ENZUME IMMUNO ASSAY)	<0.1	Index	< 1.0
SS-B/LA ANTIBODY IgG RESULT by EIA (ENZUME IMMUNO ASSAY)	NON - REACTIVE		NEGATIVE (-ve)


INTERPRETATION:


RESULT IN RU/mL	REMARKS
< 15	Negative
15 - 25	Weak Positive
> 25	Moderate Positive
>50	Strong Positive

COMMENTS:

Sjogren's syndrome is a chronic progressive inflammatory autoimmune disease marked by dryness of eyes, mouth and other mucous membranes. It occurs more frequently in women than men with an increasing prevalence throughout adult life. It is of two types – Primary Sjogren's syndrome (not associated with another connective tissue disease) and Secondary Sjogren's syndrome (associated with Rheumatoid arthritis or other autoimmune disorders). In Primary Sjogren's syndrome, Anti SS-B / La antibodies are seen in 90% whereas SS-A / Ro are seen in 60-70% of patients.




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HEPATITIS B SURFACE ANTIGEN (HBsAg) SCREENING

HEPATITIS B SURFACE ANTIGEN (HBsAg) NON - REACTIVE
RESULT

by IMMUNOCHROMATOGRAPHY

INTERPRETATION:-

1.HBsAG is the first serological marker of HBV infection to appear in the blood (approximately 30-60 days after infection and prior to the onset of clinical disease). It is also the last viral protein to disappear from blood and usually disappears by three months after infection in self limiting acute Hepatitis B viral infection.

2.Persistence of HBsAg in blood for more than six months implies chronic infection. It is the most common marker used for diagnosis of an acute Hepatitis B infection but has very limited role in assessing patients suffering from chronic hepatitis.

FALSE NEGATIVE RESULT SEEN IN:

- 1.Window period.
- 2.Infection with HBsAg mutant strains
- 3.Hepatitis B Surface antigen (HBsAg) is the earliest indicator of HBV infection. Usually it appears in 27 - 41 days (as early as 14 days).
- 4.Appears 7 - 26 days before biochemical abnormalities. Peaks as ALT rises. Persists during the acute illness. Usually disappears 12- 20 weeks after the onset of symptoms / laboratory abnormalities in 90% of cases.
- 5.Is the most reliable serologic marker of HBV infection. Persistence > 6 months defines carrier state. May also be found in chronic infection.Hepatitis B vaccination does not cause a positive HBsAg. Titers are not of clinical value.

NOTE:-

- 1.All reactive HBsAG Should be reconfirmed with neutralization test(HBsAg confirmatory test).
- 2.Anti - HAV IgM appears at the same time as symptoms in > 99% of cases, peaks within the first month, becomes nondetectable in 12 months (usually 6 months). Presence confirms diagnosis of recent acute infection.



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RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM

RHEUMATOID (RA) FACTOR QUANTITATIVE: SERUM by NEPHLOMETRY	81.85 ^H	IU/mL	NEGATIVE: < 18.0 BORDERLINE: 18.0 - 25.0 POSITIVE: > 25.0
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INTERPRETATION:-

RHEUMATOID FACTOR (RA):

1. Rheumatoid factors (RF) are antibodies that are directed against the Fc fragment of IgG altered in its tertiary structure.
2. Over 75% of patients with rheumatoid arthritis (RA) have an IgM antibody to IgG immunoglobulin. This autoantibody (RF) is diagnostically useful although it may not be etiologically related to RA.
3. Inflammatory Markers such as ESR & C-Reactive protein (CRP) are normal in about 60 % of patients with positive RA.
4. The titer of RF correlates poorly with disease activity, but those patients with high titers tend to have more severe disease course.
5. The test is useful for diagnosis and prognosis of rheumatoid arthritis.

RHEUMATOID ARTHRITIS:

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.

CAUTION (FALSE POSTIVE):-

1. RA factor is not specific for Rheumatoid arthritis, as it is often present in healthy individuals with other autoimmune diseases and chronic infections.
2. Non rheumatoid and rheumatoid arthritis (RA) populations are not clearly separate with regard to the presence of rheumatoid factor (RF) (15% of RA patients have a nonreactive titer and 8% of nonrheumatoid patients have a positive titer).
3. Patients with various nonrheumatoid diseases, characterized by chronic inflammation may have positive tests for RF. These diseases include systemic lupus erythematosus, polymyositis, tuberculosis, syphilis, viral hepatitis, infectious mononucleosis, and influenza.
4. Anti-CCP have been discovered in joints of patients with RA, but not in other form of joint disease. Anti-CCP2 is HIGHLY SENSITIVE (71%) & more specific (98%) than RA factor.
5. Upto 30 % of patients with Seronegative Rheumatoid arthritis also show Anti-CCP antibodies.
6. The positive predictive value of Anti-CCP antibodies for Rheumatoid Arthritis is far greater than Rheumatoid factor.

*** End Of Report ***



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

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

