



	<b>Dr. Vinay Cho</b> MD (Pathology & Chairman & Cons		Dr. Yugam MD ( CEO & Consultant	Pathology)
NAME	: Mrs. KAMLESH KALRA			
AGE/ GENDER	: 68 YRS/FEMALE	PATI	ENT ID	: 1620153
COLLECTED BY	:	REG.	NO./LAB NO.	: 012409210004
REFERRED BY	:	REGI	STRATION DATE	: 21/Sep/2024 07:15 AM
BARCODE NO.	: 01517370	COLL	ECTION DATE	: 21/Sep/2024 07:51AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 21/Sep/2024 01:12PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
	CLINI	CAL CHEMISTRY	BIOCHEMISTRY	
	KIC	<b>DNEY FUNCTION TE</b>	ST (COMPLETE)	
JREA: SERUM		25.91	mg/dL	10.00 - 50.00
by UREASE - GLUTAM CREATININE: SERUN	ATE DEHYDROGENASE (GLDH)	0.87	ma/dl	0.40 - 1.20
by ENZYMATIC, SPEC		0.07	mg/dL	0.40 - 1.20
	GEN (BUN): SERUM	12.11	mg/dL	7.0 - 25.0
	<i>ectrophotometry</i> )GEN (BUN)/CREATININE	13.92	RATIO	10.0 - 20.0
RATIO: SERUM	JOEN (DON)/CREATININE	13.72	KATIO	10.0 - 20.0
by CALCULATED, SPE				
UREA/CREATININE F by CALCULATED, SPE		29.78	RATIO	
URIC ACID: SERUM		4.85	mg/dL	2.50 - 6.80
by URICASE - OXIDAS	SE PEROXIDASE		-	0.50, 10, (0,
CALCIUM: SERUM by arsenazo III, spe	ECTROPHOTOMETRY	9.14	mg/dL	8.50 - 10.60
PHOSPHOROUS: SER		3.1	mg/dL	2.30 - 4.70
	DATE, SPECTROPHOTOMETRY			
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	(E ELECTRODE)	136.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUM	1	3.75	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV	/E ELECTRODE)	100.00	1.0	00.0.110.0
CHLORIDE: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	102.38	mmol/L	90.0 - 110.0
	RULAR FILTERATION RATE			
ESTIMATED GLOME	RULAR FILTERATION RATE	72.5		
(eGFR): SERUM				
by CALCULATED INTERPRETATION:				

INTERPRETATION:

To differentiate between pre- and post renal 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



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

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

 KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana

 KOS Molecular Lab: Ilnd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana

 0171-2643898, +91 99910 43898
 care@koshealthcare.com
 www.koshealthcare.com







	Dr. Vinay Ch MD (Pathology & Chairman & Cor	k Microbiology)	<b>Pr. Yugam</b> MD ( Consultant I	Pathology)	
NAME	: Mrs. KAMLESH KALRA				
AGE/ GENDER	: 68 YRS/FEMALE	PATIENT ID		: 1620153	
COLLECTED BY		<b>REG. NO./LAB</b>	NO	:012409210004	
					414
REFERRED BY	:	REGISTRATIO		: 21/Sep/2024 07:15	
BARCODE NO.	:01517370	COLLECTION I		: 21/Sep/2024 07:51	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING D	ATE	: 21/Sep/2024 01:12	PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT			
Test Name		Value	Unit	Biological R	Reference interval
9. Certain drugs (e.g.	ass (subnormal creatinine produteracycline, glucocorticoids)	uction)			
<ol> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>Prezenal azotemia</li> <li>DECREASED RATIO (&lt;         <ol> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> </ol> </li> <li>DECREASED RATIO (&lt;         <ol> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>MAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin ther</li> </ol> </li> </ol>	superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. urea rather than creatinine diffi- monemias (urea is virtually abse- of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATINII py (accelerates conversion of cr- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). rapy (interferes with creatinine r JLAR FILTERATION RATE:	nore than creatinine) (e.g. obstruent uses out of extracellular fluid). ent in blood). none) due to tubular secretion of <b>NE:</b> eatine to creatinine).	urea. methodolog	jes,resulting in normal	ratio when dehydratio
Postrenal azotemia Prerenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis ( Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Nhenacimide thera Rhabdomyolysis (r Nuscular patients NAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin ther ESTIMATED GLOMERL OKD STAGE	a (BUN rises disproportionately r superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. e. creased urea synthesis. furea rather than creatinine diffi monemias (urea is virtually absor- of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATINII py (accelerates conversion of cr- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION	nore than creatinine) (e.g. obstruents uses out of extracellular fluid). ent in blood). none) due to tubular secretion of <b>NE:</b> eatine to creatinine). ncrease in creatinine with certain neasurement).	urea. methodolog	ies,resulting in normal OCIATED FINDINGS	ratio when dehydratio
Postrenal azotemia     Prerenal azotemia     Cecreased RATIO (<         Acute tubular necr         Acute tubular necr         Acute tubular necr         Acute tubular necr         Severe liver disease         Other causes of de         Acute dialysis (         Acute tubular necr         SIADH (syndrome of         SIADH (syndrome of	a (BUN rises disproportionately r superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. and starvation. e. creased urea synthesis. furea rather than creatinine diffi monemias (urea is virtually absort of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATINII py (accelerates conversion of cre eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION Normal kidney func	nore than creatinine) (e.g. obstru- uses out of extracellular fluid). ent in blood). none) due to tubular secretion of <b>NE:</b> eatine to creatinine). ncrease in creatinine with certain neasurement). GFR (mL/min/1.73m2 stion >90 ith >90	urea. methodolog	ies,resulting in normal OCIATED FINDINGS No proteinuria	ratio when dehydratio
Postrenal azotemia     Prerenal azotemia     CEREASED RATIO (<         Acute tubular necr         Acute tubular necr         Low protein diet ar         Severe liver disease         Other causes of de         Repeated dialysis (         SIADH (syndrome of         SIADH (syndrome of         SIADH (syndrome of         SIADH syndrome of         SIADH syndrom         SIADH syndr	a (BUN rises disproportionately r superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. and starvation. e. creased urea synthesis. furea rather than creatinine diffi monemias (urea is virtually absort finappropiate antidiuretic harm 10:1) WITH INCREASED CREATINII py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION Normal kidney func Kidney damage wi normal or high Gf	nore than creatinine) (e.g. obstru- uses out of extracellular fluid). ent in blood). none) due to tubular secretion of <b>NE:</b> eatine to creatinine). ncrease in creatinine with certain neasurement). GFR (mL/min/1.73m2 ith >90 FR	urea. methodolog	ies,resulting in normal OCIATED FINDINGS No proteinuria	ratio when dehydratio
Postrenal azotemia     Prerenal azotemia     Cecreased RATIO (<         Acute tubular necr         Acute tubular necr         Low protein diet ar         Severe liver disease         Other causes of de         Severe liver disease         Severe liver d	(BUN rises disproportionately r superimposed on renal disease. (0:1) WITH DECREASED BUN : osis. nd starvation. creased urea synthesis. (urea rather than creatinine diffi monemias (urea is virtually absor- finappropiate antidiuretic harm (0:1) WITH INCREASED CREATINII py (accelerates conversion of cr- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r <u>JLAR FILTERATION RATE:</u> <u>DESCRIPTION</u> <u>Normal kidney func</u> <u>Kidney damage wi</u> <u>normal or high Gf</u> <u>Mild decrease in G</u>	nore than creatinine) (e.g. obstru- uses out of extracellular fluid). ent in blood). none) due to tubular secretion of <b>NE:</b> eatine to creatinine). ncrease in creatinine). <u>GFR (mL/min/1.73m2</u> ith >90 ith >90 FR	urea. methodolog	ies,resulting in normal OCIATED FINDINGS No proteinuria	ratio when dehydratio
Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr         Low protein diet ar         Severe liver disease         Other causes of de         Severe liver disease         Severe liver	a (BUN rises disproportionately r superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. and starvation. e. creased urea synthesis. furea rather than creatinine diffi monemias (urea is virtually absort finappropiate antidiuretic harm 10:1) WITH INCREASED CREATINII py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION Normal kidney func Kidney damage wi normal or high Gf	nore than creatinine) (e.g. obstru- uses out of extracellular fluid). ent in blood). none) due to tubular secretion of <b>NE:</b> eatine to creatinine). ncrease in creatinine). <u>GFR (mL/min/1.73m2</u> ition >90 ith >90 FR 60 -89 n GFR 30-59	urea. methodolog	ies,resulting in normal OCIATED FINDINGS No proteinuria	ratio when dehydratio



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

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









	<b>Dr. Vinay Chopra</b> MD (Pathology & Micro Chairman & Consultant	obiology) ME	m Chopra D (Pathology) ht Pathologist
NAME	: Mrs. KAMLESH KALRA		
AGE/ GENDER	: 68 YRS/FEMALE	PATIENT ID	: 1620153
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 012409210004
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 21/Sep/2024 07:15 AM
BARCODE NO.	:01517370	COLLECTION DATE	: 21/Sep/2024 07:51AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 21/Sep/2024 01:12PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	LA CANTT	
Г			
Test Name		Value Unit	Biological Reference interval

COMMENTS:

Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
 eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
 In patients, with eGFR creatinine between 45-59 ml/min/1.73 m2 (G3) and without any marker of Kidney damage, It is recommended to measure of CFD with the commended to measure

3. In patients, with eGFR cleaning between 45-59 minimit 1.73 m2 (G3) and without any marker of Kidney damage, it is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fulfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
7. A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).

ADVICE:

KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated



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

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





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	MD	Vinay Chopra (Pathology & Microbiology) rman & Consultant Pathologi		(Pathology)
NAME	: Mrs. KAMLESH	KALRA		
GE/ GENDER	: 68 YRS/FEMALE		PATIENT ID	: 1620153
COLLECTED BY	:		REG. NO./LAB NO.	: 012409210004
REFERRED BY	:		<b>REGISTRATION DATE</b>	: 21/Sep/2024 07:15 AM
BARCODE NO.	:01517370		COLLECTION DATE	: 21/Sep/2024 07:51AM
CLIENT CODE.	: KOS DIAGNOSTI	C LAB	REPORTING DATE	: 21/Sep/2024 11:35AM
CLIENT ADDRESS	: 6349/1, NICHOL	SON ROAD, AMBALA CANT	ſ	
Test Name		Value	Unit	Biological Reference interval
		IMMUNOPATH	IOLOGY/SEROLOGY	
	ANTI	CYCLIC CITRULLINATED		SENSITIVE)
ANTI CYCLIC CITRULI			AU/mL	0.00 - 5.00
ANTIBODY: SERUM		<i>.</i>		
by CMIA (CHEMILUMIN INTERPRETATION:	IESCENCE IMMUNOASS	AY)		
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 predict t 5. Anti-CCP2 may be Rheumatoid Arthritis	types: Anti-CCP1 & Ai Y <b>SENSITIVE (71%) &amp;</b> the eventual develop detected in healthy i from Polymyalgia Rł	more specific (98%) than An ment in Rheumatoid Arthriti ndividual's years before ons neumatic & Erosive SLE.	F <b>i-CCP1.</b> s (RA), when found in undiff et of clinical Rheumatoid Ar	erentiated arthritis thritis as well as to differentiate elderly onset
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Am ment in Rheumatoid Arthriti ndividual's years before ons heumatic & Erosive SLE. Pantibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint de o joints, with greatest damagon clinical, radiological & im d arthritis, as it is often pres	<b>Fi-CCP1.</b> s (RA), when found in undiffer et of clinical Rheumatoid Ar <b>Arthritis is far greater than</b> Iti-functional in origin and is estruction and in most case by in early phase. Imunological features. The is ent in healthy individuals w	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint de o joints, with greatest damagon on clinical, radiological & im	<b>Fi-CCP1.</b> s (RA), when found in undiffer et of clinical Rheumatoid Ar <b>Arthritis is far greater than</b> Iti-functional in origin and is estruction and in most case by in early phase. Imunological features. The is ent in healthy individuals w	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Am ment in Rheumatoid Arthriti ndividual's years before ons heumatic & Erosive SLE. Pantibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint de o joints, with greatest damagon clinical, radiological & im d arthritis, as it is often pres	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint d e joints, with greatest damagon clinical, radiological & im d arthritis, as it is often press s of patients with RA, but no	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint d e joints, with greatest damagon clinical, radiological & im d arthritis, as it is often press s of patients with RA, but no	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint d e joints, with greatest damagon clinical, radiological & im d arthritis, as it is often press s of patients with RA, but no	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint d e joints, with greatest damagon clinical, radiological & im d arthritis, as it is often press s of patients with RA, but no	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint d e joints, with greatest damagon clinical, radiological & im d arthritis, as it is often press s of patients with RA, but no	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint d e joints, with greatest damagon clinical, radiological & im d arthritis, as it is often press s of patients with RA, but no	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint d e joints, with greatest damagon clinical, radiological & im d arthritis, as it is often press s of patients with RA, but no	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic
2. Anti-CCP is of two t 3. Anti-CCP2 is HIGHL 4. Anti-CCP2 may be Rheumatoid Arthritis 5. The positive predic seronegative Rheuma RHEUMATOID ARTHIR 1. Rheumatoid Arthrit membrane lining (syr 2. The disease spread 3. The diagnosis of R measurement of RA fa 4. RA factor is not spin nfections.	types: Anti-CCP1 & Ai Y SENSITIVE (71%) & the eventual develop detected in healthy i from Polymyalgia Ri tive value of Anti-CC atoid Arthritis also sh CTIS: itis is a systemic auto novium) joints which ds from small to large A is primarily based actor. ecific for rheumatoid	hti-CCP2. more specific (98%) than Ami ment in Rheumatoid Arthriti ndividual's years before ons eumatic & Erosive SLE. P antibodies for Rheumatoid how Anti CCP antibodies bimmune disease that is mu leads to progressive joint d e joints, with greatest damagon clinical, radiological & im d arthritis, as it is often press s of patients with RA, but no	<b>Fi-CCP1.</b> s (RA), when found in undiffiet of clinical Rheumatoid Ar <b>Farthritis is far greater than</b> ti-functional in origin and is estruction and in most case ge in early phase. munological features. The is ent in healthy individuals w t in other form of joint disea	erentiated arthritis thritis as well as to differentiate elderly onset <b>Rheumatoid factor. Up to 30% patients with</b> as characterized by chronic inflammation of the ess to disability and reduction of quality life. most frequent serological test is the ith other autoimmune diseases and chronic

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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

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

 KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana

 KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana

 0171-2643898, +91 99910 43898
 care@koshealthcare.com
 www.koshealthcare.com

