P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

0171-2532620, 8222896961 pkrjainhealthcare@gmail.com

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	**: Mrs. SARITA**		
AGE/ GENDER	: 28 YRS/FEMALE	**PATIENT ID**	: 1566145
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122408010009**
REFERRED BY	:	**REGISTRATION DATE**	: 01/Aug/2024 11:01 AM
BARCODE NO.	: 12503933	**COLLECTION DATE**	: 01/Aug/2024 11:03AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 01/Aug/2024 01:39PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
CLINICAL CHEMISTRY/BIOCHEMISTRY			
KIDNEY FUNCTION TEST (BASIC)			
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	18.62	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	0.48	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETERY*	8.7	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	18.13	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	38.79	RATIO	
URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	5.9	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 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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ENDOCRINOLOGY

ANGIOTENSIN CONVERTING ENZYME (ACE): SERUM

ANGIOTENSIN CONVERTNG ENZYME (ACE): SERUM	11.5	U/L	8.0 - 52.0

by FURYLACRYLOYLPHENYLALANYGLYCYLGLYCINE (FAPPG)

INTERPRETATION

1.Angiotensin converting Enzyme (ACE) also known as kinase II, is present in many cells types such as neuronal cells, renal proximal tubular cells, and mostly in endothelial cells.
2.Angiotensin converting enzyme (ACE) modulates peripheral vascular resistance as well as renal and cardiovascular function. It is responsible for conversion of Angiotensin I to Angiotensin II as well as inactivation of bradykinin
3.It is attached to endothelial surface membrane by an anchor peptide and can be cleaved to be released into the blood circulation as soluble enzyme. Serum ACE activity is significantly elevated in patients with untreated active disease.
4.Majority of ACE is tissue bound (> 90%) found predominantly in lungs & testes
5.It has been established as an important diagnostic parameter in Sarcoidosis. Spontaneous or induced remission of sarcoidosis has been seen, by decreasing serum ACE values.

FACTORS AFFECTING ACE LEVELS:

1.Smoking – ACE activity is 30% lower in smokers
2.Thyroid hormone- Stimulates ACE synthesis
3.Postmenopausal estrogen replacement – ACE activity is 20% lower

INCREASED LEVELS:

1.Sarcoidosis – ACE levels are used in the diagnosis and monitoring of this disease and are directly related to the number of organs affected and activity of granulomas. Mature granulomas produce less ACE than developing ones. ACE is more likely to be elevated with pulmonary involvement than with purely hilar adenopathy.
2.Pulmonary causes like Emphysema, Asthma, Small cell carcinoma & Squamous cell carcinoma, Idiopathic pulmonary fibrosis
3.Renal diseases – patients on hemodialysis show high ACE levels as compared to patients who are not on dialysis, chronic renal failure
4.Other causes – Multiple sclerosis, Addison's disease, Hyperthyroidism, Diabetes Alcoholic hepatitis &cirrohosis & Peptic ulcer,histoplasmosis, hodgkins disease, gauchers disease, leprosy, amyloidosis, tuberculosis
5.Elevated ACE is thought to be a risk factor for myocardial infarction & cardiomyopathy.
7.ACE inhibitors have found wide spread application in treatment of systemic hypertension and Congestive Heart Failure (CHF). Monitoring of ACE may be beneficial to determine the optimum low dose of ACE inhibitor.

DECREASED LEVELS

1.Chronic liver disease.
2.Anorexia nervosa
3.Hypothyroidism

To be correlated clinically

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