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 Chairman & Consultant Pathologist

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NAME	: Mr. ASHWANI AGGARWAL	PATIENT ID	: 1716640
AGE/ GENDER	: 60 YRS/MALE	REG. NO./LAB NO.	: 012501050045
COLLECTED BY	:	REGISTRATION DATE	: 05/Jan/2025 03:45 PM
REFERRED BY	:	COLLECTION DATE	: 05/Jan/2025 03:54PM
BARCODE NO.	: 01523491	REPORTING DATE	: 05/Jan/2025 04:19PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY
CREATININE PHOSPHOKINASE-MB (CPK-MB)

CPK-MB - SERUM	2.98	ng/mL	0.0 - 5.0
<i>by EFIA (ENZYM FLUORESCENT IMMUNOASSAY)</i>			

Interpretation:-

1. Alternative name of Creatine Kinase (CK) is Creatine phospho-kinase (CPK).
2. Creatine Kinase (CK) is a dimeric enzyme composed of two types of monomer sub-units (i.e. M-Muscular & B-Brain), which combine to form three distinct CK isoenzymes.
 - a). CK-BB (CK-I), is produced primarily by brain, lungs and smooth muscles, and enters the blood only on injury to these organs like cerebrovascular accidents or pulmonary infarctions.
 - b). CK-MB (CK-II), is produced primarily by heart muscle;
 - c). CK-MM (CK-III), is produced primarily by skeletal muscle.
- 3). Normally very little CK is found circulating in the blood. Elevated levels indicate damage to either muscle or brain possibly from a myocardial infarction, muscle disease, or stroke.
- 4). CK levels are reduced in first half of pregnancy, and increased in second half of pregnancy.

Increased:-

Physiological:-

1. Strenuous physical activity.
2. New Born.

Pathological :-

1. Myocardial & pulmonary infarction
2. Accident and recent surgery.
3. Drugs:- Statins.
4. Convulsions & brain tumour.
5. Myopathies
6. Malignant hyperthermia
7. Hypothyroidism & Hyperthyroidism

5). CK-MB (CK-II) levels increase significantly 4-6 hours following a myocardial infarction and peak at around 12-24 hours after the infarct. The levels return to normal, in case of no further myocardial damage, after 24 to 48 hours. Hence the increased levels of CK-MB along with elevated levels of total CK is a good indicator of myocardial infarction.

6). For diagnosis of MI with high sensitivity and specificity, serial sampling over a period of 8 to 12 hours is required. For accurate diagnosis of myocardial infarction, CK-MB activity along with total CK should be measured. If the total CK activity is raised and CK-MB contributes more than 6% of the total activity, then myocardial infarction is considered highly probable.




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IMMUNOPATHOLOGY/SEROLOGY

TROPONIN I ULTRASENSITIVE (QUANTITATIVE)

TROPONIN I ULTRASENSITIVE (QUANTITATIVE)	0.02	ng/mL	< 0.50
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by ELFA (ENZYME LINKED FLUORESCENT IMMUNOASSAY), NEXT GENERATION, ULTRASENSITIVE

INTERPRETATION:

NOTE:
1.False negative / positive results are observed in patients receiving mouse monoclonal antibodies for diagnosis or therapy.

COMMENTS

- 1.Troponin is a regulatory complex of 3 proteins that resides at regular intervals in the thin filament of striated muscle.
- 2.Cardiac Troponin is a cardiospecific, highly sensitive marker of myocardial damage and has never shown to be expressed in normal, regenerating or diseased skeletal muscle.
- 3.In cases of acute myocardial damage, Troponin I levels rise in serum about 3-4 hours after appearance of cardiac symptoms and remain elevated upto 10 days.
- 4.It is an independent prognostic marker which can predict near, mid and long term outcome in patients with Acute Coronary Syndrome (ACS).

INCREASED LEVELS

- 1.Congestive Heart Failure
- 2.Cardiomyopathy
- 3.Myocarditis
- 4.Heart contusion
- 5.Interventional therapy like cardiac surgery and drug induced cardiotoxicity

USES

- 1.To differentiate patients with Non ST elevation Myocardial Infarction (NSTMI) from Unstable angina-patients with ACS with elevated Troponin I and / or CK-MB are considered to have NSTMI whereas the diagnosis of Unstable angina is established if Troponin I and CK-MB are within the normal range.
- 2.Ideally Troponin I should be measured at presentation (0 hour) and repeated after 6-9 hours & 12-24 hours if earlier specimens are normal and the clinical suspicion is high.
3. Risk stratification of patients presenting with ACS and for cardiac risk in patients with Chronic Renal Failure. As it offers powerful risk assessment, in ACS, Troponin I monitoring should be included in practice guidelines.
4. For selection of more intensive therapy and intervention in patients with elevated Troponin I.

*** End Of Report ***




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