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Short Communication

The Concept of Cardiac Troponin in the Diagnosis of Myocardial Infraction

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DESCRIPTION

Although numerous molecules are used as therapeutic targets or/and biomarkers for illness laboratory diagnosis, the study of endogenous molecule metabolism is not only of significant fundamental value but also of tremendous practical importance. Since cardiac troponin molecules have been the primary biomarkers for the diagnosis of myocardial infarction for a long time, the development of high-sensitivity test methods has fundamentally altered many of our preconceived notions about how these cardiac indicators are metabolized. The comprehension and justification of these prospective new diagnostic capabilities of cardiac troponins in clinical practice are strongly related to the basic principles governing the metabolism of these molecules [1]. Our current understanding of cardiac troponin metabolism is inadequate and incredibly disjointed from many literary sources.

The potential significance of cardiac troponin metabolism in the laboratory diagnosis of myocardial infarction is thus not properly understood by many studies. This in-depth analysis aims to organize knowledge on cardiac troponin metabolism and concentrate on the potential effects of cTns metabolism on the myocardial infarction laboratory diagnosis. This thorough overview follows a systematic analysis of the metabolic pathway's steps, beginning with potential release mechanisms and concluding with elimination mechanisms [2]. This will make it possible for medical professionals and researchers to comprehend the critical role that cTns metabolism plays in the myocardial infarction laboratory diagnosis.

The proteins known as troponins-troponin I, troponin T, and troponin C-are a component of the troponin complex, which is connected to the protein tropomyosin. Actin and tropomyosin work together to create thin filaments of myocytes, which are the most crucial part of the contractile apparatus of striated muscle cells (of skeletal and cardiac muscles). All three troponins are involved in calcium-dependent control of the contraction-relaxation cycle of striated muscle. Each type of troponin performs particular regulatory tasks during the contraction and relaxation of striated muscles. When actin is bound during relaxation, the troponin I member of the tropomyosin complex inhibits actomyosin's ATPase activity, which prevents muscle contraction in the absence of calcium ions in the cell cytoplasm [3].

The regulatory subunit, troponin T, is responsible for attaching the troponin complex to thin filaments and, as a result, taking part in calcium-regulated contraction. The subunit that binds calcium is called troponin C. The sarcoplasmic reticulum, also known as "the repository of calcium ions," releases calcium ions into the sarcoplasm when the action potential is passed to the muscle cell. When calcium ions attach to troponin C, proteins in the troponin-tropomyosin complex undergo conformational (structural) changes that cause the tropomyosin molecule to shift and release binding sites for the myosin head on the actin filament. It makes it possible for the myosin head to connect with actin, which is essential for the contraction of striated muscles.

Depending on where in the muscle they are located, troponin molecules have varied amino acid structures, which is how troponin isoforms are identified. The three isoforms of troponin I are cardiac troponin I, troponin I from skeletal muscle fibres with fast twitch, and troponin I from skeletal muscle fibres with slow twitch. The three primary isoforms of troponin T are cardiac troponin T, troponin T from fast-twitch skeletal muscle fibres, and troponin T from slow-twitch skeletal muscle fibres. The amino acid sequences of cardiac troponin I and cardiac troponin T differ by about 40%-60% from those of the corresponding isoforms of skeletal troponins localized in skeletal muscle fibres, according to molecular genetic studies [4].

The use of cardiac troponins T and I as specific biomarkers for the diagnosis of myocardial injury in myocardial infarction and other noncardiac clinical situations is made possible by this significant structural uniqueness.

Contrary to troponins I and T, cardiac troponin C shares an exact amino acid structure with skeletal (muscular) troponin C. As a result, elevated blood levels of this protein make it impossible to reliably distinguish between the damage to cardiac muscle tissue and the damage to skeletal muscles, and cardiac troponin C cannot be used as a cardiac marker for myocardial infarction diagnostics [5].

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CONCLUSION

Pulmonary embolism, renal failure, acute aortic dissection, heart failure, acute myocarditis, rhabdomyolysis, administration of cardiotoxic chemotherapy, acute exacerbation of chronic obstructive pulmonary disease, sepsis, and infiltrative cardiac pathologies were the most frequent causes of troponin T increase (for example, amyloidosis). This study's significant finding was that in 30% of cases, elevated troponin T levels had nothing to do with any of the previously mentioned reasons of elevated cardiac troponin.

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