Cardiovascular test
Troponins (cTnI, cTnT)

Description: Troponins are regulatory proteins specific to cardiac muscle. They are the gold standard for diagnosing heart attacks.
Timing: They enter the bloodstream within 2-3 hours after heart injury and remain elevated for several days.
Clinical Use: High sensitivity and specificity make them essential for diagnosing myocardial infarction and acute myocardial ischemia.
The troponin complex found in striated cardiac muscle tissue is composed of three distinct protein molecules: troponin I (cTnI), troponin T (cTnT), and troponin C (cTnC). These proteins, in conjunction with tropomyosin, play a crucial role in regulating the contraction and relaxation of the heart's muscular layer.[1,2] The amino acid composition of these proteins is vital for their functional efficacy. Genetic investigations have identified numerous mutations in the genes responsible for encoding the troponin complex proteins, leading to severe hereditary disorders affecting the contractile function of cardiac muscle, collectively referred to as cardiomyopathies.[3] Notably, two of the three components of the myocardial troponin complex, cTnI and cTnT, exhibit distinct amino acid structures compared to their skeletal muscle counterparts, rendering them unique and suitable as biomarkers for identifying ischemic damage to the myocardium during acute myocardial infarction (AMI). In contrast, the amino acid structure of cTnC remains identical in both cardiac and skeletal muscle tissues, which limits its specificity for laboratory diagnosis of AMI.[4,5]
While it is generally accepted that cTnI and cTnT are exclusive to the myocardium, some studies have indicated their presence outside the heart, specifically in the muscular walls of the vena cava and pulmonary veins in humans and other mammals.[6,7] Given this information, cTnI and cTnT cannot be regarded as entirely specific cardiac markers, and further research into the causes and mechanisms behind the extramyocardial expression of cardiac troponins is warranted.[8,9,10]
Cardiac muscle tissue comprises approximately 4.0-6.0 mg of troponin I and 10.0-11.0 mg of troponin T. Notably, around 95% of these troponin levels are integrated into the troponin complex, which serves a structural role and is essential for the contractile function of the myocardium.[11] Conversely, the remaining 5% of the total troponin I and troponin T molecules are found in the cytosol of myocardial cells, constituting the cytoplasmic troponin fraction, and do not play a role in regulating the contractile activity of cardiac muscle tissue.[12,13]

References

1. Boussouf SE, Geeves MA. Tropomyosin and troponin cooperativity on the thin filament. Adv Exp Med Biol 2007; 592: 99-109. http://dx.doi.org/10.1007/978-4-431-38453-3_10 PMID: 17278359.
2. Maeda Y, Nitanai Y, Oda T. From the crystal structure of troponin to the mechanism of calcium regulation of muscle contraction. Adv Exp Med Biol 2007; 592: 37-46.
3. Chaulin AM. Biology of cardiac troponins: Emphasis on metabolism. Biology (Basel) 2022; 11(3): 429.
4. Vikhorev PG, Vikhoreva NN. Cardiomyopathies and related changes in contractility of human heart muscle. Int J Mol Sci 2018; 19(8): 2234.
5. Cheng Y, Regnier M. Cardiac troponin structure-function and the influence of hypertrophic cardiomyopathy associated mutations on modulation of contractility. Arch Biochem Biophys 2016; 601: 11- 21.
6. Pasquale F, Syrris P, Kaski JP, Mogensen J, McKenna WJ, Elliott P. Long-term outcomes in hypertrophic cardiomyopathy caused by mutations in the cardiac troponin T gene. Circ Cardiovasc Genet 2012; 5(1): 10-7.
7. Messner B, Baum H, Fischer P, Quasthoff S, Neumeier D. Expression of messenger RNA of the cardiac isoforms of troponin T and I in myopathic skeletal muscle. Am J Clin Pathol 2000; 114(4): 544- 9.
8. Ricchiuti V, Apple FS. RNA expression of cardiac troponin T isoforms in diseased human skeletal muscle. Clin Chem 1999; 45(12): 2129-35. http://dx.doi.org/10.1093/clinchem/45.12.2129 PMID: 10585344 [9] Wens SCA, Schaaf GJ, Michels M, et al. Elevated plasma cardiac troponin T levels caused by skeletal muscle damage in Pompe disease. Circ Cardiovasc Genet 2016; 9(1): 6-13.
9. Schmid J, Liesinger L, Birner-Gruenberger R, et al. Elevated cardiac troponin T in patients with skeletal myopathies. J Am Coll Cardiol 2018; 71(14): 1540-9.
10. Rusakov DY, Vologdina NN, Tulayeva ON. The development of striated cardiac muscle tissue in the walls of the caval and pulmonary veins. Journal of Anatomy and Histopathology 2015; 4(3): 105-5.
11. Chaulin AM, Duplyakov DV. Analytical review of modern information on the physiological and pathochemical mechanisms of the release of cardiospecific proteins from muscle tissue, methodology and technologies of their research, interpretation of the results. Laboratory Diagnostics. Eastern Europe 2022; 11(1): 78-97.
12. Dhoot GK, Gell PG, Perry SV. The localization of the different forms of troponin I in skeletal and cardiac muscle cells. Exp Cell Res 1978; 117(2): 357-70.
    Filatov VL, Katruha AG, Bulargina TV. Gusev NB. Troponin: Structure, properties and mechanism of functioning. Biochemistry. 1999.