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Impacts of troponin in heart health and disease

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Abstract

Troponin is a complex protein that has crucial roles in muscles contraction particularly in heart. This protein encompasses of three subunits including (C, I and T). As tissue injury, troponin is released to bloodstream making it a principal marker to detect tissues/cardiac related complications. Especially with introducing of high-sensitivity assays, cardiac troponins had emerged as mainstay of the diagnosis, risks stratification, and prognostication of myocardial injury. Cardiac troponins are important biomarkers in acute coronary syndrome and myocardial infarction, but recent studies have shown them to be of diagnostic and prognostic importance also in a greater variety of non-ischemic conditions including heart failure, myocarditis, pulmonary embolism, and sepsis. This review discusses the changing clinical role of troponin, particularly with respect to its kinetic properties, relevance in diverse pathophysiological scenarios, and incorporation into multi-marker approaches. We also describe future developments, highlighting the integration of troponin into machine learning modelling and precision medicine initiatives. Such innovations can improve diagnostic yield, facilitate early intervention, and guide personalized treatment strategies in both ischemic and non-ischemic heart disease.

Keywords: Troponin; Cardiac Biomarkers; Myocardial Injury; Myocardial Infarction; High-Sensitivity Troponin Assays; Heart Failure; CVD

1. Introduction

Heart diseases are the leading cause of globally morbidity and mortality, according to 2022 statistics, these diseases responsible for about 19.8 million death and accounting for one-third of all globally deaths (1). This burden is exceptionally high in both lower and middle-income countries, where more than 75% of cardiovascular diseases (CVD) linked deaths happen, mostly triggered by the escalating frequency of hypertension, diabetes, obesity, smoking, unhealthy diet, physical inactivity, and excessive consumption of alcohol (2). As people expanding with age and urbanization, the expansion and economic effects of CVD are predictable to rise, posing rising pressure on healthcare settings globally (3).

Biomarkers have become one such tool in the diagnosis, management, and prognostication of CVD in this light. As such, they are threshold markers of biological and pathological processes and are indispensable to the diagnosis of myocardial injury, its clinical application and outcomes prediction (4). High-sensitivity assays that are more advanced in terms of technology help clinicians identify smaller levels of myocardial injury compared to those previously attainable with traditional assays and earlier in the progression of CVD, allowing for timely intervention and better risk stratification in the acute and chronic setting (5).

Among these cardiac biomarkers, is troponin, which considered as a gold standard to detect the myocardial injury owing to its high sensitivity and specificity (6). Troponin or troponin complex protein is a complex from three regulatory

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proteins involving troponin C (TNC), troponin I (TNI), and troponin T (TNT), which represent the chief causes of muscles contractions in skeletal and cardiac muscles; while these types of proteins do not find in smooth muscles (7,8).

Troponins regarded as integral constituents for contractile system in cardiac muscle, and their releasing into circulation indicates damage to cardiomyocytes. The introducing of high-sensitivity cardiac troponin assays had revolutionized acute coronary syndrome (ACS) diagnostics, offering more precise early rule-in and rule-out protocols in patients suffering from chest pains (6). Moreover, increased troponin concentrations are not only linked with ischemic injury, but also high levels use as indicative for other disorders like heart failure, myocarditis, pulmonary embolism, along with chronic kidney diseases, makes the interpretation context-based (5).

The aim of present review article is to epitomize function and impact of troponin on homeostasis and pathology of the heart. It will specifically [1] describe cardiac troponin biology and kinetics in health and disease, [2] provide an overview of the diagnostic and prognostic significance of troponin elevation in a range of cardiovascular and non-cardiovascular diseases, [3] review the clinical utility and limitations of contemporary and high-sensitivity troponin assays, [4] discuss Advances on the horizon, including the potential role of troponin in multi-marker strategies and artificial intelligence-based prediction models. This review addresses the main aspects of this polydiagnostic condition chiefly based on data derived from peer-reviewed clinical studies and international guidelines published in the past years.

2. The Structure and Biological Functions of Troponin

Troponin is an inhibitory protein complex made up of three regulatory subunits: Troponin T (TnT), Troponin I (TnI), and Troponin C (TnC). The third protein that is required for contraction in skeletal and cardiac muscle is called troponin, which is absent in smooth muscles. Troponin attaches itself to a specific protein known as tropomyosin (9). At rested cardiac muscle cell, it prevents myosin from being binds with filaments of actin, while in contraction, otherwise, occurs when (Ca) enter cardiac sarcoplasm that bind to the troponin complex causes conformational changes that permits myosin to allove bind to actin filaments (10, 11). Troponin is attached to other myofilaments about 90% of the time and protein-free in the cytosol about 10% of the time. In cases of muscle injury, this cytosolic troponin increases as a consequence of infiltration from disrupted muscles membranes. Consequently, muscles injuries or death lead to freer cytosolic troponin and increased its serum concentrations [9]. The TnC isoforms being the same in cardiac and skeletal muscles, while Troponin I and T have cardiac specific isoforms, cTnI and cTnT, respectively (Figure 1). Giving to this cardiac specificity, cTnI as well as cTnT are actually suggested for employed as biomarkers for heart damages and diagnostic markers for acute myocardial infarction (12).

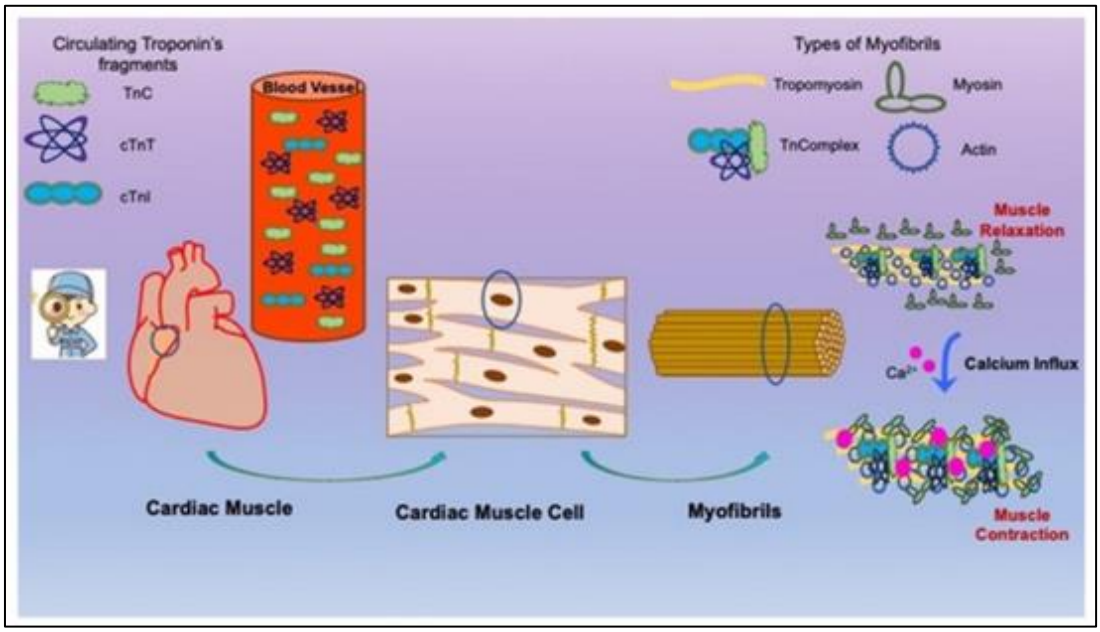

Figure 1 Cardiac muscles working rely on cardiac muscles cells. Tropomyosin, troponin complex, myosin, and actin — the myofibrils that counteract upon calcium entering myoblast to trigger myoblast contraction. When necrosis of cardiomyocytes happens (especially encompasses a wide range of cardiomyocytes), it causes entering in the circulation of troponins, which justifies their very high levels in blood. Such a fixed increase of cTnI and cTnT in plasma level might somehow be indicative of cTnI and cTnT efficaciousness to denote myocardial damage

The isoforms of Troponin reveal differential expression amongst tissues, and both TnI and TnT in cardiac muscle possess isoforms that are structurally and functionally different from those in skeletal muscle. These isoforms are tissue-specific, each encoded by a different gene, possessing unique amino acid sequences that provide insight into myocardial damage detection using immunoassay approaches (13). This specificity has helped to make cardiac troponins key clinical diagnostics, especially in the setting of distinguishing cardiac damage from pathology of skeletal muscle, in which there is shared reactivity that can cloud the diagnosis (14). This molecular specificity highlights the need for isoform characterization in preclinical and clinical studies.

In the context of roles of Troponin in calcium regulation and Myocardial Contractility, it's a key modulator of calcium-dependent myocardial contractility. Calcium ions released from the sarcoplasmic reticulum following excitation-contraction coupling bind to TnC initiating a signaling cascade that allows for actin-myosin cross-bridge formation and contraction (15). Calcium sensitivity of the troponin complex is regulated by several physiological mechanisms such as heart rate, adrenergic stimulation, and post-translational modifications and modulates output and performance of the heart (16). It is well known that dysfunction in this calcium-troponin interaction is associated with multiple cardiac disease states, including heart failure and cardiomyopathies, indicating its central importance to myocardial performance.

3. Troponins in Regular Heart Physiology

3.1. What are The Normal Baseline Troponin Concentrations?

Usually, cardiac troponin levels are undetectable or extremely low in the blood of healthy individuals. According to assayed used normal reference range for cardiac troponin I (cTnI) and troponin T (cTnT) show slight variation, but with most high sensitivity tests reporting normal values at < 0.04ng/mL (17). Since troponin is normally sequestered within the cytosol of the cardiomyocyte, and released into intracellular circulation only following cellular injury or stress, these baseline levels represent an approximation of voluetric integrity of myocardial cells. However, small increases in this parameter in the normal range may even have prognostic value in some populations, like the elderly or those with subclinical cardiovascular disease (18).

3.2. Troponin Significance in Cardiac Adequacy and Rhythm

In addition to serving as a diagnostic tool, troponin also is involved in the central function of enabling calcium-mediated contraction, maintaining cardiac efficiency, and regulating rhythm. Troponin complex (troponin C, I, and T) serves to transmit the calcium-sensitive signal to actin filaments to modulate their interaction with the myosin filaments during systole. Calcium ions are released from the sarcoplasmic reticulum, binding to troponin C and causing conformational changes as a result, relieving the perpendicular placement and inhibitory effect of troponin I and allowing for cross-bridge cycling (19, 20). Many contractile tissues such as those within the myocardium exist in a state of what is called electromechanical coupling.

In addition, troponin functional or expression changes can disturb excitation–contraction coupling that can underlie arrhythmias and loss of cardiac output. Troponin has long been recognized as a fulcrum of cardiac function in that even modest changes in troponin isoform phosphorylation or calcium sensitivity alter myocardial relaxation and contractile force (21). This is a fundamental aspect of normal heart physiology which renders troponin not just a cardiac injury biomarker.

4. The Role of Troponin in Diagnostic Assessment of Cardiac Diseases

4.1. Troponin rising: the signature of damage to the heart muscle

Cardiac troponins (cTnI and cTnT) are highly sensitive and specific markers of myocardial damage. Troponin are proteins found in cardiomyocytes that are released into the blood when these cells are damaged by necrosis or extreme strain, and thus, troponin elevation is a classic marker of acute and chronic injury to the heart. Troponin elevation correlates with the amount of necrosed myocardium and is linked to poor clinical outcomes, including risk of death and the development of heart failure (22). Troponin elevation is not synonymous with myocardial infarction and must be correlated with clinical symptoms and electrocardiographic changes (23). Troponin in the circulation, especially >99th percentile of healthy reference population, identifies myocardial injury [ischemic and non-ischemic etiology (24).

Although earlier biomarkers like creatine kinase-MB (CK-MB) or lactate dehydrogenase were used, troponins have higher myocardial specificity and remain detectable in circulation longer following injury allowing for a wider

diagnostic window. In addition, recent improvements in the assay sensitivity have increased the diagnostic and prognostic value of troponins beyond traditional limits in many heart diseases (25).

4.2. High-Sensitivity Troponin Test: Revolutionizing Early Diagnosis

The advent of hs-cTn tests had revolutionized the early identification of myocardial infarction (MI). These tests could detect troponin levels as low as 1–5 ng/L, enabling for diagnosising the myocardial injury within 1- 2 hours of symptoms onset (26). This has importantly boosted the timeliness and validity of given clinical decision in emergency setting, lessening time to diagnosis and aiding earlier starting of suitable therapy (25).

High-sensitivity tests as well expose low-level chronic troponin rising, which were formerly undetectable employing traditional examinations. While this has improved sensitivity, it has also introduced diagnostic complexity, requiring clinicians to interpret results in the context of dynamic changes (rise or fall) and clinical presentation (5, 27). Moreover, gender-specific and age-specific thresholds had been suggested to boost the diagnostic accuracy of hs-cTn tests (28).

Besides, these tests have also aided the evolution of rapid rule-in and rule-out algorithms for M, many of which were validated internationally; for instance, European Society of Cardiology (ESC) 0/1-hour algorithm had revealed high sensitivity and negative predictive value in rule out acute MI in emergency settings (29).

4.3. Troponin in Acute Coronary Syndrome and Myocardial Infarction

The cardiac troponins (especially cTnI and cTnT) are regard as cornerstone biomarkers to diagnosis MI and assess patients with ACS. These proteins had a great myocardial specificity and sensitivity, making them essential tools to differentiate the MI cases from other causes of chest pains, along with using in prognostic stratification (30). Troponins are releasing into bloodstream if myocardial cells are damaged, thus they being critical components for diagnostic criteria of MI, as described in Fourth Universal Definition of MI, based on this definition, a rising and/or falling in cardiac troponin levels with at least one value above 99th percentile upper reference limit, in coincidence with clinical evidence of myocardial ischemia, asserts the diagnosis of MI (31).

Troponin has a critical role to distinguish among ACS subtypes, where it includes a several conditions inclusive of unstable angina (UA), non-ST-segment elevation MI (NSTEMI), and ST-segment elevation MI (STEMI).

The UA being without myocardial necrosis, therby troponin concentrations stay within normal range. While in NSTEMI, the myocardial necrosis occurs without constant ST-segment elevation, so troponin concentrations are raised. In STEMI, clinical together with ECG criteria are obvious, with notable rising in troponin values next to onset of infarction (32). Given that ECG outcomes could occasionally be obscure, the measure of troponin provides a decisive biochemical standard to recognize UA from NSTEMI, aids to guide suitable therapeutic interventions.

Beyond diagnosis, the troponins are vigorous indicators of ACS prognosis. Several studies had revealed that higher troponin levels, exceptionally with high-sensitivity assays, are linked with escalated risk of adverse cardiovascular consequences, like recurrent MI, HF, as well as all-causes mortality; this prognostic utility is independent of traditional other risk factors and ECG outcomes. For instance, patients with high troponins levels and non-ST-elevation ECG pattern may profit from early invasive strategies, such as angiography and revascularization, as part of risk-guided therapy algorithms (5, 33-34).

The incorporation of troponins tests into clinical algorithms like 0/1-hour and 0/3-hour rule-in/rule-out protocols, had regulated the management of individuals suffered from chest pain. These protocols, validated by European Society of Cardiology, depend on hs-cTn quantifications at presentation and after a short period to rapidly pick out individuals at high or low risk for MI. This approach had been demonstrated to safely minimize time to diagnosis and boost resource utilization in emergency departments (35).

4.4. Troponin in Non-Ischemic Diseases: Heart Failure, Myocarditis, and others

While cardiac troponins are often linked with MI and ACS, but also they are now well accepted that raised troponin levels could happen in non-ischemic cardiac and systemic diseases. The hs-cTn examinations have remarkably augmented the ability to reveal subclinical or chronic myocardial injury in several pathological contexts, many of which aren't occurring by primary coronary artery occlusion. This expands the advantage of troponin beyond diagnostic framework of ACS and highlights the significance of clinical contexts to interpret upraised levels (28,30).

4.4.1. In heart failure

Acute and chronic heart failure (HF) is one of the largest clinical entities in cardiovascular and, more broadly, internal medicine. While cardiac biomarkers are being investigated, significant progress has been made in the diagnosis, evolution, prognosis and treatment of HF. When we speak of HF, the most important of these is cardiac troponins (cTnT and cTnI) (36).

Upon disruption of the myocyte membrane as a consequence of ischemic (most commonly) or non-ischemic factors, the cytosolic troponins gain access to the extracellular space and blood followed by the structural troponins such as troponin fragments. Higher troponin levels are proportional to the amount of cellular injury and peak 4 to 6 hours after ischemic necrosis of myocardium. In this sense, troponin I or T serum rises are a major diagnostic component in acute MI (STEMI and NSTEMI) (37, 38). Over the past 10–15 years, data demonstrated that the sera troponins concentrations could be elevated in ACS, but they also could be increased in non-ischemic myocardial injury (e.g., myocarditis and cardiotoxicity) or in other conditions with multifactorial injury (e.g., HF and pulmonary embolism). Healthy subjects also present high hs-cTn above the detection limit (39). Thus, elevated levels of cTn are shown in many clinical scenarios; but in the present context, where chronic and acute HF coexist, they represent a serious diagnostic (infarction or other myocardial injury), assessment (hs-cTn or conventional troponin) and clinical pathophysiological diagnostic dilemma. Higher cTn values predominate among patients with HF (40).

Troponin levels increased in advanced HF and is often associated with poor prognosis. The elevated cTn concentrations are associated with lower left ventricular ejection fraction, reflecting the severity of HF, and they predict prognosis. Progressive loss of myocytes through necrosis and apoptosis leads to development of worsening HF, either in an ischemic heart or non-ischemic heart. Activation of the renin-angiotensin-aldosterone and sympathetic nervous system and inflammatory mediators are potential mechanisms that may mediate myocardial injury (34).

4.4.2. in Myocarditis

Myocarditis is an inflammation of the myocardium with a heterogenous range of etiologies but may be due to generalized infections, toxins, drugs, and autoimmune processes (41). Standard clinical presentation patterns involve chest pains, arrhythmias, and HF. Its clinical presentation is extremely heterogeneous from asymptomatic or mild flu-like illness to cardiogenic shock or sudden death (42). Integrated clinical assessment (such as history, physical test, electrocardiography) and noninvasive imaging (such as echocardiography, cardiac magnetic resonance [CMR] imaging) is essential to diagnosis of suspected myocarditis cases. The gambling must generally be appropriately excluded by angiogram with chest ache in individuals with myocarditis (43).

An endomyocardial biopsy (EMB) is regarded as gold caliber for an ultimate identification and handling of myocarditis. But it is an expensive invasive test with a complication risk and is subject to sampling error, because myocarditis can be a focal process. In turn, EMB and some specific treatments (such as immunosuppression) must only be considered in individuals who present with dominant clinical syndromes (eg, high likelihood of giant cells myocarditis with severe HF and/or life-alarming arrhythmias) that are unresponsive to traditional treatments. Today, however, there are more innovative, less invasive imaging modalities CMR that may facilitate the detection and direct treatment in acute myocarditis. T2-W leads to the presence of free water in tissue, which is elevated in the inflammatory process that accompanies acute myocarditis; CMR diagnosis of AM can be made accurately and non-invasively and T2-W edema imaging can form a basis for such a diagnosis. T2-W has been shown to have high sensitivity and specificity compared to EMB (44).

Cardiac troponins have long been validated as sensitive and specific markers for myocardial injury. Myocarditis is associated with increased cTnI in only slightly more than one-third of patients (43). Troponin elevation is due to immune-mediated myocyte injury rather than ischemia. Troponin elevation, possibly related to the inflammatory process severity, may not correlate with ECG or imaging findings, particularly in mild or subclinical cases (45) and can be exacerbated in RA patients by high inflammatory background. Despite this, a diagnosis of myocarditis cannot be ruled out by a normal EKG, or negative troponin and/or creatine kinase (46).

Troponin elevation is indeed not specifically indicative of myocarditis, however, could potentially assist in diagnosis when combined with CMR and clinical presentation. In acute myocarditis, CMR shows late gadolinium enhancement and myocardial edema correlating with elevated troponin levels. In addition, elevated troponin portends prognostic implication, with a high level of troponin being correlated with greater risk for cardiovascular events such as arrhythmia and sudden cardiac death (47).

4.4.3. In Pulmonary Embolism and Right Ventricular Strain

Patients with acute pulmonary embolism (PE) may demonstrate elevated troponin levels due to right ventricular (RV) pressure overload and ischemia. In this context, troponin elevation has been associated with RV dysfunction, increased pulmonary artery pressures, and higher short-term mortality (48) Importantly, troponin elevation in PE does not indicate MI but signifies myocardial strain, thereby serving as a valuable prognostic marker.

In cases with PE, high cTn levels were documented in up to 50% of these cases. Depends on cut-off value (0.1 ng/ml vs. 0.01 ng/ml), cTnT rates 32% as well as 50% were reported, respectively. Lately, these troponins had regarded as vital diagnostic mean for risks stratification in cases with PE (49).

4.4.4. Systemic Inflammatory Responses Syndrome and Sepsis/Septic Shock

In ICU patients with sepsis or systemic inflammatory response syndrome, high cTn had reported in a broader range of 36% (cTnT> 0.1 ng/ml) (6) to 85% (cTnI > 0.1 ng/ml). Differences in prevalence is likely due to differences in underlying causes of sepsis, the use of different troponin assays, and different established applied cut-off values. Typically, these troponin elevations do not stem from significant coronary artery disease, suggesting other mechanisms (34). The pathophysiology is complex and probably related to cytokine-mediated myocardial depression, microvascular dysfunction and direct cardiomyocyte injury. Septic patients with high troponin levels have an increased requirement for vasopressors, prolonged length of stay in the intensive care unit, and increased mortality (50).

4.4.5. Chronic Renal Diseases and End Stage Renal Diseases

The results show that the peaks in cTn levels of great height and persistence in advanced renal failure. End-stage renal disease (ESRD) patients have already high troponin values before an acute cardiac event, and also require repeated early measurements to exclude a significant increase indicative for acute ischemia. Many believe that decreased renal. The higher level of cTn in ESRD patients due to clearance explained by the accompanying diseases leading to release of cTn like severe heart failure and left ventricular hypertrophy which induces sub endocardial ischemia (51).

4.4.6. In Tachycardia

Tachycardia alone had been pointed out as a possible cause of troponins increasing levels, as confirmed in small case series. In one series including 21 cases with increase cTnI concentrations and normal coronary angiograms, tachycardia had specified to demonstrate troponin elevation in (6) cases (52).

4.4.7. Extreme Exercise

Exercise, on the other hand, is the best way to improve heart health and longevity. When comparing those limits with upper reference limit (or traditional upper limit) for someone like CTN, it may appreciate it as exercise will elicit an short term increase in CTN release that may exceed the upper reference limit for a high proportion of population.

For decades, the question of whether exercise-induced CTN elevations are a physiologic or pathophysiologic response, and their clinical relevance has been hotly debated. Thus far, CTN elevations caused by exercise have been regarded as the only benign type of CTN elevation. However, more recent studies have uncovered some intriguing results that may provide us with new insight into the mechanisms behind and clinical significance of CTN elevations with exercise. The appearance of CTNT or CTNI has been described after intense ultra-endurance exercise (49). The reasons for these elevations and their prognostic implications are not clearly understood. In the young, exercise can increase membrane permeability via a number of mechanisms (dehydration, hemoconcentration, acidosis, etc.) leading to an elevation of the non-pathological troponin (8).

5. Advancing on Horizon: The prospective Role of Troponin in Multi-Marker Strategies and Artificial Intelligence-depended Prediction Models

Cardiac troponins, especially HS-CTN tests, had converted the landscape of cardiovascular diagnostic by providing earlier and valid diagnosis for myocardial injury. However, the growing complexity of CVD pathophysiology and overlapping of biochemical markers in ischemic and non-ischemic diseases underline the limitations of depending on troponin as a stand-alone marker. By advancing the precision medicine, there is escalating interests in multi-marker protocols and artificial intelligence (AI)-bases models that integrating troponins levels with other clinical, biochemical, and imaging signatures to reinforce diagnostic, prognostic, and therapeutic decisions (53,54).

5.1. Troponins in Multi-Markers Strategies

The integration of troponin with other cardiac and systemic parameters is being progressively investigated to present complemental pathophysiological insights. While troponins levels reflect myocytes injuries, other biomarkers like natriuretic peptides reveal myocardial wall stresses, C-reactive protein indicates systemic inflammations, and growth differentiation factor-15 indicates cellular stressed and aging (55). When employed with each other, they can reinforce diagnostic recognition, boost risk stratification, along with guide therapy more precisely.

Research had revealed that combined HS-CTN with natriuretic peptides boosted the prognostic estimations in individuals with HF, ACS, and stable coronary artery disease (56). (Omland et al., 2009). In steady out cases, even modest rising in these markers pick out cases at substantially higher risk of CVD events versus to those with normal levels (57).

Additionally, in emergency settings, combining troponin with copeptin, a stress-linked marker released early in MI, had been studied to speed up rule-out protocols, particularly within first hour of presenting (58). These multi-marker protocols may lessen on serial troponins measurements and short the decision times.

However, still there are challenges like standardization of cut-offs, interpretation complexity, and cost-effectiveness examination. Besides, these protocols need advanced algorithms and clinical decision bolsters up systems to ease integration into routine examination.

5.2. Artificial Intelligence and Machine learn with Troponins

Artificial intelligence, especially machine learning and deep learning, provides transformative prospective in combining troponin data with broad-ranging clinical, demographic, genomic, along with imaging databases. These models may identify complex, non-linear patterns that aren't easily visible to doctors and can present real-time, individualized risks prediction, diagnosis, and therapy optimization (59).

5.2.1. Predictive Models in Emergency Departments

These models that combine HS-CTN readings alongside ECG, age, comorbidity, and other laboratory measurements were revealed excellent accuracy in predicting acute MI, as compared with conventional algorithms. Then et al. (60) revealed that machine learning-based prediction approaches boosted the early rule-in and rule-out of MI as compared to classical threshold-based assays.

5.2.2. Artificial Intelligence in Prognostication

The algorithms of AI were also sophisticated to prophesy long-term CVD consequences in patients with raised troponin, even in lack of apparent ACS. These methods utilize troponin as a key characteristic among several of variables to approximate risks of future HF, sudden cardiac death, or recurrent ischemic event (61).

5.2.3. Natural Language Processing and electronic health records Integration

With advancing natural language processing, AI systems may essence and synthesize information from electronic health records, consolidating structured troponins outcomes with unstructured clinical observations, imaging results, and doctor’s impressions. This approach helps real-time clinical decision bolster up, enabling identify individuals at risk of adverse consequences or improper discharge (62).

6. Conclusion

Troponin was highlighted as a key marker in cardiovascular field; it bridges molecular physiology with clinical diagnostic. Its impacts in regulation cardiac muscles contraction emphasize its biological importance, while its rising in bloodstream serves as a sensitive measure of myocardial injury across various conditions. The advent of hs-cTn assays had revolutionized early diagnosis and risks stratification. Where, troponin was rapidly changed from a single diagnostic biomarker into a node within complex diagnostic and prognostic networks. Together, multi-marker strategies add incremental value by accessing different facets of cardiovascular pathology, and AI and machine learning models have the computational capability to utilize these high-dimensional datasets. As a whole, they constitute an advance at the San’s frontier of cardiovascular precision medicine, it's a setting in which troponin is both marker of injury and central part of a real-time, data-driven decision-making process.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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