



Clinical Assessment and Management of Blunt Cardiac Injury for Nursing and Frontline Healthcare Providers

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Abstract

Background: Blunt cardiac injury (BCI) encompasses a spectrum of conditions resulting from chest trauma. While most patients, particularly those with minor injuries, recover fully, a subset with severe trauma faces significant acute and delayed complications. The diagnosis and management of BCI are challenging due to its variable presentation and the lack of universal diagnostic criteria.

Aim: This article aims to review the potential complications, long-term management strategies, and the critical role of interprofessional collaboration in optimizing outcomes for patients with BCI.

Methods: The approach is based on a review of clinical practices and consensus. It advocates for a systematic management protocol involving initial screening with electrocardiography (ECG) and troponin levels, inpatient monitoring for high-risk patients, and structured medium-term follow-up.

Results: Most patients with minor BCI recover without lasting consequences. However, severe injuries can lead to acute complications (e.g., chamber rupture) or delayed sequelae such as heart failure, arrhythmias, pericardial effusion, and constrictive pericarditis. Effective management hinges on a coordinated, interprofessional team approach, clear patient education on warning symptoms, and a recommended follow-up evaluation at 3 to 6 months post-injury.

Conclusion: A proactive and collaborative approach, balancing the generally favorable prognosis with vigilance for rare but serious complications, is essential for the effective long-term care of patients with BCI.

Keywords: Blunt cardiac injury, cardiac contusion, trauma, complications, follow-up, interprofessional care, patient education.

Introduction

Blunt cardiac injury (BCI) represents a broad and heterogeneous spectrum of cardiac damage resulting from non-penetrating trauma to the thorax. The range of pathology extends from subtle myocardial bruising, which may be clinically silent, to catastrophic structural disruption such as chamber rupture that is rapidly fatal if not promptly recognized and treated. Although only a minority of affected individuals manifest overt abnormalities on electrocardiogram (ECG) or present with

hemodynamic compromise in the form of shock at the time of initial assessment, a substantial proportion are asymptomatic or minimally symptomatic in the early phase. This deceptive clinical quiescence is clinically significant because the myocardium may still be vulnerable to the development of sudden malignant arrhythmias or acute impairment of cardiac pump function. These potential complications underscore the imperative that BCI be routinely considered in the evaluation of patients with significant blunt chest trauma, so that appropriate monitoring, diagnostic

evaluation, and timely intervention can be instituted to mitigate the risk of preventable morbidity and mortality.[1] Historically, the designation “cardiac contusion” has been widely employed in the literature and clinical practice to describe cardiac injury sustained following blunt impact to the chest wall.[2] From a histopathological standpoint, cardiac contusion is characterized by a contused myocardium demonstrating hemorrhagic infiltration, focal or patchy myocyte necrosis, and interstitial edema. These structural alterations are most reliably and definitively documented at the time of surgical exploration or on post-mortem examination, when direct visualization and histologic sampling are feasible.[3] Nevertheless, reliance on this term alone has become increasingly recognized as inadequate in contemporary practice, as it implies a relatively narrow subset of myocardial injury and does not fully capture the breadth of possible anatomic and functional derangements. As a result, the broader clinical term “blunt cardiac trauma” has gained preference, since it more accurately encompasses the diverse range of potential injuries that may arise from blunt chest trauma, including myocardial contusion, valvular disruption, coronary artery injury, septal defects, pericardial lesions, and conduction system disturbances.[4][5]

Epidemiological data further illuminate the clinical relevance of this entity. Between 2017 and 2021, analysis of the National Trauma Data Bank identified 14,219 cases of BCI among approximately 4.8 million patients who sustained blunt trauma, corresponding to an overall incidence of roughly 0.3% within this population.[6] Although this proportion appears numerically small, the absolute number of affected individuals is considerable given the high global burden of blunt trauma, particularly in regions with elevated rates of road traffic injuries. Within this group, BCI may be further delineated according to specific structural injuries, functional disturbances, or patterns of observed cardiac dysfunction, thereby emphasizing that it is not a single uniform pathology but rather a constellation of related conditions.[7] Significant BCI is most commonly associated with high-energy mechanisms of injury. Motor vehicle collisions constitute the predominant cause, accounting for approximately 50% of reported cases. Pedestrians struck by motor vehicles represent an additional 35%, reflecting the severe kinetic forces transmitted to the thorax in such scenarios. Motorcycle crashes contribute about 9% of cases, and falls from substantial heights comprise roughly 6% of the burden.[8][9][10] These distributions highlight that BCI should be particularly suspected in victims of high-impact mechanisms, where rapid deceleration, direct chest compression against the steering wheel or seatbelt, or crush injuries can impart substantial force to the heart within the confines of the thoracic cavity.

The diagnosis of BCI remains intrinsically challenging and is frequently the subject of clinical uncertainty. One major obstacle is the absence of

universally accepted, standardized diagnostic criteria or a definitive “gold standard” diagnostic test. This difficulty is exacerbated in the context of polytrauma, where concurrent injuries—such as pulmonary contusions, rib fractures, hemothorax, or intra-abdominal hemorrhage—may overshadow or mimic manifestations of cardiac involvement. As a result, the reported incidence of cardiac injury following blunt chest trauma varies markedly in the literature, ranging from 8% to 76%. Much of this variability can be attributed to differences in definitions, the diagnostic modalities employed, and the lack of consistent guideline-based criteria used across studies.[10] In patients who do not exhibit severe arrhythmias or hemodynamic instability, the clinical significance of BCI is sometimes questioned or de-emphasized, leading to debate regarding the need for intensive monitoring or extensive cardiac workup in all cases.[8][11] Nonetheless, in the evaluation of any patient with significant blunt trauma, maintaining a high index of clinical suspicion for BCI is essential. Certain patients—particularly those with concerning mechanisms of injury, abnormal ECG findings, elevated cardiac biomarkers, or unexplained hypotension—should be carefully observed and monitored for delayed or evolving cardiac sequelae. Importantly, no single clinical sign or symptom has been reliably identified as pathognomonic for BCI, nor has any specific constellation of findings been consistently validated as a robust predictor of subsequent cardiac complications.[8][12][13] This absence of a definitive clinical hallmark means that clinicians must synthesize information from the mechanism of injury, physical examination, ECG, imaging studies, and laboratory testing to arrive at a reasoned assessment of risk.

Ultimately, in the context of blunt cardiac injury, the downstream consequences—or sequelae—are of greater clinical importance than the mere application of a diagnostic label. The potential for arrhythmias, cardiogenic shock, heart failure, or structural complications such as ventricular septal defects or valvular insufficiency carries direct implications for patient outcomes, resource utilization, and long-term cardiac function. Accordingly, the focus of clinical decision-making should not be confined to whether a patient “does” or “does not” meet a somewhat arbitrary definition of BCI, but rather to the identification, prevention, and management of adverse cardiac events that may ensue in the aftermath of blunt chest trauma.[8][12][13] In this way, the recognition of BCI serves as a framework to guide vigilant monitoring, timely intervention, and appropriate disposition, with the overarching goal of minimizing the risk of preventable cardiac morbidity and mortality in trauma patients.

Etiology

Blunt cardiac injury (BCI) most frequently arises as a consequence of high-energy blunt chest trauma, with motor vehicle collisions representing the

predominant causative mechanism, accounting for approximately 50% of cases. Notably, it is estimated that around 20% of all fatalities resulting from motor vehicle collisions involve significant blunt chest trauma, underscoring the critical contribution of thoracic impact to trauma-related mortality.[4] In addition to road traffic incidents, a variety of other traumatic mechanisms may precipitate BCI, including falls from height, blast-related injuries, interpersonal assault, and diverse forms of blunt force impact to the chest wall.[2] The specific pattern and severity of cardiac injury largely depend on both the mechanism and magnitude of the applied force, as well as the direction and point of impact. When the kinetic energy transmitted to the thorax is sufficiently great, the heart may be violently compressed between the rigid structures of the sternum anteriorly and the vertebral column posteriorly. This mechanism can result in myocardial contusion, chamber rupture, valvular disruption, or injury to the coronary vasculature. In high-velocity deceleration events, such as head-on collisions or falls from substantial heights, shearing forces may act on the heart and great vessels, leading to tearing of the myocardium or disruption of its attachments, including the venae cavae, pulmonary veins, or great arterial roots. These severe lesions are frequently incompatible with life, and many affected individuals succumb at the scene before reaching medical care. Although BCI may coexist with injuries to adjacent intrathoracic structures—including the thoracic aorta, lungs, ribs, sternum, and spine—such associated injuries, while suggestive of significant chest trauma, are not themselves definitive indicators of cardiac involvement. Their presence does, however, signal a high-energy mechanism and should prompt heightened clinical vigilance. Consequently, in patients presenting with substantial thoracic trauma, clinicians should maintain a high index of suspicion for BCI and undertake appropriate evaluation, even in the absence of immediately obvious cardiac manifestations.[9]

Epidemiology

In the United States, trauma remains the fourth leading cause of death, reflecting its substantial public health burden and its capacity to produce complex, multisystem injuries that challenge both prehospital and in-hospital care pathways. Within this broader context, the epidemiology of blunt cardiac injury (BCI) is notably difficult to define with precision. Reported incidence rates vary widely across studies, largely because of the absence of universally accepted diagnostic criteria and a standardized definition that consistently distinguishes BCI from other traumatic or medical conditions with overlapping presentations. As a result, the true prevalence of BCI is likely underestimated or inconsistently captured in trauma databases and clinical reports, complicating attempts to assess its clinical impact on injured populations. The potential

for misdiagnosis also contributes to the variability in reported incidence. Clinical features that may suggest BCI—such as arrhythmias, chest pain, or nonspecific ECG abnormalities—can also arise from unrelated conditions, including preexisting cardiac disease, electrolyte imbalances, substance use, or stress responses associated with trauma. Similarly, laboratory findings such as elevated cardiac troponins may be falsely attributed to myocardial injury when, in fact, the elevation results from non-cardiac mechanisms, such as global hypoperfusion, direct skeletal muscle damage, or stress-induced cardiomyocyte strain occurring in the context of multisystem trauma.[14] These overlapping features complicate the diagnostic process and highlight the importance of clinical judgment, mechanism-of-injury assessment, and integration of multimodal diagnostic tools when evaluating suspected BCI [14].

To address the need for a more structured and reproducible classification system, the American Association for the Surgery of Trauma (AAST) developed the AAST Cardiac Injury Scale, an instrument that grades the severity of both blunt and penetrating cardiac injuries. This standardized scale provides a graduated framework ranging from minor myocardial contusions to catastrophic structural disruptions, thereby offering clinicians a more consistent method for categorizing injury patterns and facilitating communication across trauma teams and research settings. Despite its utility, clinicians must recognize that the scale includes specific rules regarding injury severity: for example, the presence of multiple penetrating injuries confined to a single cardiac chamber, or involvement of more than one chamber, increases the assigned injury grade by one level. Such considerations are essential for accurately determining injury severity, estimating prognosis, and guiding appropriate management decisions. Ultimately, the epidemiological understanding of BCI continues to evolve as diagnostic modalities improve and as efforts toward consensus definitions gain traction. Continued refinement of diagnostic criteria, documentation practices, and trauma registry reporting will be essential to more accurately characterize the incidence, clinical consequences, and resource implications of this complex and often underrecognized injury pattern [14].

Pathophysiology

Blunt cardiac injury (BCI) arises through several distinct but sometimes overlapping mechanical forces applied to the thorax. A variety of mechanisms have been proposed, including direct and indirect impact, bidirectional compressive forces, rapid deceleration, blast-related overpressure, crush injuries, concussive forces, and complex combined patterns of trauma.[2][4][15] Among these, direct impact to the anterior chest wall is regarded as the most frequent and clinically important mechanism. This is particularly hazardous when the blow occurs at a point in the

cardiac cycle when the ventricles are maximally filled, namely at the end of diastole, as the increased chamber volume and wall tension render the myocardium more vulnerable to structural disruption.[8] Indirect mechanisms can also contribute significantly to myocardial damage. A sudden increase in venous return, such as from a force transmitted through the abdomen or extremities that acutely elevates preload, can produce an abrupt rise in intracardiac pressure. This sharp pressure surge may exceed the tensile strength of the myocardial or valvular structures, predisposing the heart to rupture or other forms of structural failure.[8] In addition, bidirectional forces, such as those generated when the chest is compressed between an external object and the rigid vertebral column, can squeeze the heart between the sternum and spine. This mechanism can create intense localized stress on the cardiac chambers and great vessels, leading to contusion, laceration, or full-thickness rupture of cardiac tissue.[8]

Deceleration forces represent another important pathophysiologic pathway in BCI. Rapid deceleration, as occurs in high-speed motor vehicle collisions or falls from height, may cause shearing stresses at the points where mobile cardiac structures attach to relatively fixed anatomical anchors. This can result in tears of the valvular apparatus, disruption of the myocardium, or injury to the coronary arteries via intimal disruption or dissection.[2][8] Collectively, these mechanisms highlight that BCI is not a single lesion but rather the end result of complex biomechanical forces acting on a dynamic organ within the confined space of the thoracic cavity. The distribution of cardiac chamber involvement in BCI closely reflects the anatomical orientation of the heart within the chest. The right ventricle and right atrium are positioned more anteriorly than the left-sided chambers and therefore are more directly exposed to external forces applied to the sternum and anterior thoracic wall.[8][16] As a result, these right-sided chambers are particularly susceptible to contusions, lacerations, and rupture. Among patients who sustain fatal blunt cardiac trauma, the most commonly reported lethal injuries include rupture of one or more cardiac chambers, accounting for approximately 64% of cases. Venous-atrial confluence tears, involving the junctions of the venae cavae or pulmonary veins with the atria, constitute an additional 33% and are often rapidly exsanguinating. Coronary artery tears or dissections, either isolated or in combination with chamber rupture and venous-atrial disruption, also contribute substantially to mortality in this cohort.[9]

A distinctive and dramatic manifestation of BCI is commotio cordis, a phenomenon most frequently observed in young male athletes who experience sudden cardiac death following a seemingly minor blow to the chest, often from a projectile such as a baseball or hockey puck. In these cases, the affected individuals have no known preexisting cardiac disease, and post-mortem

examination reveals no structural or morphological abnormality of the heart. The prevailing hypothesis is that the impact occurs during a vulnerable phase of the cardiac cycle, particularly during ventricular repolarization, precipitating ventricular fibrillation without leaving any macroscopic or microscopic evidence of damage.[4][9] This underscores the fact that lethal electrical disturbances can be triggered purely by mechanical stimuli, even in the absence of persistent structural injury. Most patients who survive the initial traumatic event have less severe forms of BCI, which may still span a broad range of structural and electrophysiologic abnormalities.[9] One such structural entity is the intramural hematoma, in which hemorrhage occurs within the myocardial wall. Clinically, intramural hematomas are often self-limited, with a generally benign natural history; they tend to resolve over a period of approximately 4 to 12 weeks. During this time, however, the altered myocardial substrate can act as an arrhythmogenic focus, leading to premature ventricular contractions or transient conduction disturbances such as bundle branch block.[9] Another important structural complication is papillary muscle rupture, which compromises the integrity of the atrioventricular valve apparatus. In the acute setting, this can lead to severe valvular regurgitation with resultant volume overload of the affected ventricle, acute pulmonary edema, and cardiogenic shock. Such injuries frequently require early recognition and surgical repair to restore valvular competence and prevent progressive hemodynamic deterioration.[9] Septal injuries, including ventricular septal defects, may also occur following blunt trauma. Early in their evolution, these lesions may present with subtle signs such as a new murmur or arrhythmia identified on physical examination or echocardiography. The pathophysiologic sequence often begins with myocardial contusion, followed by necrosis and subsequent delayed rupture of the septal tissue.[8][9] Because of their potential to progress from a relatively contained lesion to a full-thickness defect, timely diagnosis is essential; in selected cases, early intervention can be lifesaving and may improve long-term functional outcomes [8][9].

Electrical and conduction disturbances are central to the clinical expression of BCI. The most frequently observed dysrhythmias include sinus tachycardia, premature atrial contractions, premature ventricular contractions, and atrial fibrillation.[4][9] These rhythm abnormalities may result from direct injury to the conduction system, ischemia of the myocardium, autonomic imbalance, or the effects of intramural hemorrhage and edema on impulse propagation. It is important, however, to interpret these findings within the broader context of trauma physiology. In the injured patient, sinus tachycardia is more commonly a manifestation of hypovolemia, ongoing hemorrhage, pain, anxiety, or systemic inflammatory response than of isolated BCI. Consequently, the presence of tachycardia in a trauma

patient should initially prompt an aggressive search for occult bleeding and other life-threatening causes. Only after such causes have been excluded should BCI assume greater prominence in the differential diagnosis.[4][9] In summary, the pathophysiology of blunt cardiac injury encompasses a spectrum of mechanical forces, anatomical vulnerabilities, and structural and electrical sequelae. Direct and indirect impact, bidirectional compression, deceleration, and concussive phenomena all play roles in determining the pattern and severity of cardiac damage.[2][4][15] The right-sided chambers are particularly exposed due to their anterior position, and fatal injuries frequently involve chamber rupture, venous-atrial confluence disruption, and coronary artery tears.[8][9][16] Survivors may exhibit a range of structural lesions and dysrhythmias that require careful evaluation and monitoring.[9] Understanding these mechanisms is fundamental to recognizing BCI, anticipating its complications, and guiding appropriate diagnostic and therapeutic strategies in the trauma setting.

History and Physical

A meticulous approach to history-taking and physical examination is essential in evaluating patients with suspected blunt cardiac injury (BCI), particularly because early manifestations may be subtle or overshadowed by other traumatic injuries. The initial step in assessment involves prompt identification of the mechanism of trauma. Understanding how the injury occurred often provides the first and most important clue to possible cardiac involvement. For example, in motor vehicle collisions, determining whether the patient struck the steering wheel or dashboard can help estimate the degree of chest wall impact. Significant vertical deceleration injuries also carry substantial risk; one study demonstrated that more than half—specifically 54%—of patients who sustained falls greater than 20 feet exhibited evidence of BCI.[9] These findings reinforce the need to correlate mechanism with clinical suspicion, as the energy transferred to the thorax often dictates the likelihood and severity of cardiac damage. Patients may present with a variety of symptoms, although the most frequently reported complaints include chest pain and shortness of breath.[2][4] These symptoms may reflect underlying myocardial contusion, pericardial irritation, or associated thoracic injuries. In other cases, patients may report palpitations, which could signify arrhythmias stemming from myocardial irritation. Less commonly, BCI may mimic ischemic heart disease, and individuals may present symptoms characteristic of angina, complicating differentiation from acute coronary syndromes in the trauma setting.[4][17] Because symptomatology may overlap significantly with that of non-traumatic cardiac conditions, careful probing into the nature, timing, and evolution of symptoms is crucial [17].

A comprehensive assessment of cardiac risk factors further enhances diagnostic accuracy.

Evaluating a history of myocardial infarction, structural heart disease, arrhythmias, and comorbidities such as hypertension or diabetes is essential. These conditions may predispose patients to complications or alter the clinical presentation. Equally important is obtaining an accurate and detailed medication history. Cardiac medications may mask or accentuate symptoms: for instance, beta-blockers and calcium channel blockers can blunt tachycardic responses, potentially obscuring early physiological compensation and leading clinicians to underestimate the severity of injury. The physical examination must be thorough, systematic, and attentive to both subtle and overt signs. One of the most critical diagnoses to identify on physical exam is cardiac tamponade, a potentially life-threatening condition that may occur in the setting of blunt cardiac trauma. Clinicians should maintain a high index of suspicion for tamponade in patients manifesting jugular venous distention, hypotension, or muffled heart sounds. Although Beck's triad is classic, it is often absent in trauma patients, making adjunctive imaging essential. The focused assessment with sonography for trauma (FAST) is a valuable tool in this regard; it allows rapid bedside identification of pericardial fluid, which may indicate tamponade or significant cardiac injury. Beyond tamponade, several nonspecific but important examination findings may heighten suspicion for BCI. These include tachypnea, irregular or diminished breath sounds, anterior chest wall tenderness, visible chest abrasions or ecchymosis, rib or sternal fractures, and seatbelt marks across the thorax.[2][4][12][17] Such findings not only signify substantial force applied to the chest but also commonly coexist with BCI, emphasizing the relationship between high-energy impact and cardiac vulnerability. Importantly, severe BCI often occurs in conjunction with multisystem trauma. In such cases, distracting injuries—such as long bone fractures, abdominal hemorrhage, or traumatic brain injury—may divert clinical attention away from subtle cardiovascular signs.[12][17] This interplay highlights why vigilance is crucial: even minimal symptoms may mask significant underlying cardiac pathology [12][17]. In summary, the evaluation of patients with suspected blunt cardiac injury relies on a nuanced understanding of trauma mechanisms, a detailed history that incorporates both symptomatology and comorbid conditions, and a comprehensive physical examination that integrates bedside diagnostic tools. Appreciating the potential interplay between visible thoracic injuries, nonspecific clinical indicators, and coexisting traumatic insults enables clinicians to identify BCI more reliably and initiate timely management.

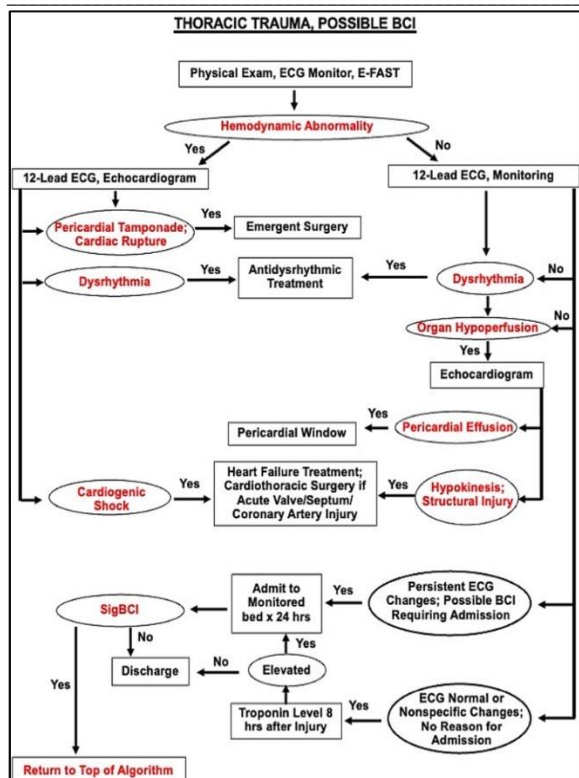


Fig. 1: Cardiac blunt Algorithm.

Evaluation

The evaluation of blunt cardiac injury (BCI) remains one of the more challenging aspects of trauma assessment because no universally accepted diagnostic criteria or consensus guidelines exist to clearly define the condition. Despite this lack of standardization, important clinical frameworks have been developed to guide practice. In 2012, the Eastern Association for the Surgery of Trauma published practice guidelines recommending that all patients with suspected BCI undergo an electrocardiogram (ECG) as an initial screening tool.[8][9] The rationale behind this recommendation lies in the capacity of ECG abnormalities—such as arrhythmias, conduction delays, or signs of ischemia—to signal underlying myocardial insult. Patients whose ECG results are abnormal should be admitted for continuous cardiac monitoring due to the potential for evolving arrhythmias or hemodynamic instability. However, it is equally important to acknowledge the limitations of the ECG. A normal tracing does not entirely exclude BCI. Studies have shown that a subset of patients with initially normal ECG findings may later demonstrate biochemical evidence of myocardial injury, particularly through elevated cardiac troponin I (cTnI) levels measured approximately 24 hours after trauma.[4][8] Conversely, the combination of a normal ECG and normal cardiac troponin levels has been shown to reliably identify patients at low risk, allowing them to be safely discharged in the absence of other concerning clinical features.[4][8][9] Nonetheless, even a normal ECG and cTnI value do

not fully eliminate the possibility of BCI, as certain injuries—such as septal defects—may evolve over time and present in a delayed fashion. For this reason, clinical judgment must complement laboratory and imaging findings when evaluating trauma patients who may be at risk.

The principal diagnostic modalities employed in assessing BCI include ECG, echocardiography, cardiac biomarkers, and specialized imaging such as radioisotope scans. When the initial 12-lead ECG is normal, guidelines suggest repeating the ECG after 4 to 6 hours while simultaneously obtaining cardiac biomarker levels, including troponin and creatine kinase. Serial trends in these measurements are important because certain patterns of BCI may manifest progressively rather than immediately. Decisions regarding additional testing, including repeat biomarker measurements, follow-up echocardiography, or radioisotope imaging, depend on evolving clinical status and the appearance of new or changing abnormalities in ECG or laboratory data.[18] The choice of diagnostic pathway is highly institution-dependent, as hospitals vary in their protocols, resource availability, and thresholds for advanced imaging. Thus, the diagnosis of BCI often requires individualized assessment and remains a complex task for the trauma team. Advanced imaging modalities such as computerized tomography (CT) and magnetic resonance imaging (MRI) are not recommended as part of the initial diagnostic evaluation for BCI. While CT and MRI provide detailed anatomical visualization, they are not sufficiently sensitive or specific for diagnosing acute myocardial injury in the trauma setting and are typically reserved for cases in which symptoms persist without a clear explanation or when structural abnormalities are suspected but not confirmed through standard testing.[4][8] These modalities serve primarily as adjuncts rather than primary diagnostic tools [4][8].

Bedside ultrasonography, specifically the focused assessment with sonography for trauma (FAST), plays an essential role in trauma evaluation and can contribute valuable information in the assessment of potential cardiac injury. FAST is particularly useful for detecting pericardial fluid, which may indicate tamponade, and is strongly recommended for hemodynamically unstable patients with blunt abdominal trauma, those with penetrating injuries near the thoracoabdominal junction where peritoneal violation is uncertain, and any patient exhibiting unexplained clinical instability. The traditional FAST examination includes assessment of the pericardial space to identify effusion, the right upper quadrant, left upper quadrant, and pelvis for the detection of hemoperitoneum.[6] The extended FAST (e-FAST) examination expands this protocol by including evaluation of the pleural spaces to identify hemothorax or pneumothorax, conditions that can coexist with or complicate blunt cardiac injury.[6] This expanded assessment enhances the clinician's

ability to rapidly diagnose multiple life-threatening thoracic and abdominal conditions at the bedside. For additional detail on the FAST and e-FAST protocols, reference may be made to StatPearls' companion resource, "Focused Assessment With Sonography for Trauma," which provides an in-depth discussion of technique and interpretation. Overall, the evaluation of BCI depends on a strategic integration of clinical history, mechanism of injury, hemodynamic assessment, ECG interpretation, biomarker analysis, and targeted imaging. Given its diagnostic challenges and potential for delayed manifestations, careful observation and judicious use of diagnostic tools are essential for accurate identification and optimal management of patients with suspected blunt cardiac injury [4][6][8].

Treatment / Management

The management of blunt cardiac injury (BCI) must be grounded in the systematic principles of trauma care, beginning with the primary survey as outlined by the American College of Surgeons' Advanced Trauma Life Support (ATLS) guidelines. This structured approach prioritizes airway, breathing, and circulation, ensuring that life-threatening conditions are rapidly identified and addressed. In any injured patient, especially those with significant blunt thoracic trauma, the presence of shock mandates a thorough evaluation of all potential etiologies. Particular attention should be given to cardiogenic and compressive causes of shock, as these are more prevalent when the chest has been subjected to high-energy impact. Physical examination, electrocardiography (ECG), and extended focused assessment with sonography for trauma (e-FAST) are fundamental tools in this setting, enabling the early detection of critical conditions such as pericardial tamponade, major wall motion abnormalities, and significant hemodynamically relevant dysrhythmias.[1] These early investigations lay the foundation for subsequent diagnostic refinement and therapeutic decision-making, as summarized in the Blunt Cardiac Injury Algorithm. Once initial stabilization has been achieved, further management is guided by a combination of ECG findings and cardiac biomarker levels, particularly cardiac troponin I (cTnI). Patients who exhibit abnormalities in both ECG and cTnI should be admitted for continuous cardiac monitoring for a period of 24 to 48 hours, as available evidence suggests that life-threatening arrhythmias or the onset of cardiac failure most commonly occur within this time window.[4][17] The choice between admission to an intensive care unit and placement on a telemetry ward is influenced by several factors, including the presence and severity of concomitant injuries, the type and degree of ECG derangement, and the extent of hemodynamic instability. Importantly, routine admission of all patients with isolated sternal fractures and normal ECG findings is not warranted solely to exclude BCI,

as the presence of a sternal fracture in isolation does not reliably predict clinically significant cardiac injury. The most frequently encountered subset of BCI comprises patients with either isolated ECG abnormalities or isolated elevations in cTnI. In the majority of these cases, the clinical course is benign, and long-term functional impairment is uncommon.[2]

Management of dysrhythmias in the context of BCI generally parallels that in non-trauma cardiac patients, adhering to standard advanced cardiac life support principles. Care must be taken to optimize the physiological milieu by correcting electrolyte imbalances, particularly abnormalities in potassium and magnesium, as well as avoiding or correcting hypoxia and acidosis, both of which can exacerbate arrhythmic tendencies. When indicated, antiarrhythmic medications and advanced life support algorithms should be employed in accordance with established guidelines. Although uncommon in isolated BCI, high-grade atrioventricular block or complete heart block may occur, and in such instances, temporary or permanent pacemaker insertion may be required to maintain adequate cardiac output. ST-segment elevations on ECG in a trauma patient present a diagnostic challenge: they may reflect myocardial contusion or a traumatic myocardial infarction due to coronary artery injury. In these circumstances, coronary angiography is often necessary to differentiate between these entities and to guide revascularization strategies when appropriate.[4] Patients who are found to have significant structural cardiac injury on clinical, echocardiographic, or other imaging grounds require urgent evaluation by cardiology and, frequently, cardiothoracic surgery. These injuries may include chamber rupture, valvular disruption, septal defects, or major coronary or great vessel lesions. While definitive repair often necessitates surgical intervention, temporizing medical measures play a crucial role in stabilizing patients before and during transfer to the operating room. Such measures may include careful fluid resuscitation to optimize preload, judicious use of inotropes to augment myocardial contractility, and vasopressors to maintain systemic perfusion pressure in the face of cardiogenic shock or vasodilation. The therapeutic strategy must be individualized based on the pattern of injury and the overall trauma burden.[2][4]

Cardiac tamponade, most frequently associated with cardiac chamber rupture in the setting of BCI, constitutes a true surgical emergency. Rapid recognition and immediate intervention are vital, as ongoing accumulation of pericardial blood can swiftly compromise cardiac filling and output. In most cases, definitive management requires emergent cardiothoracic surgical exploration and repair, with pericardiocentesis serving only as a temporary or bridging measure in select situations.[2][4] In patients with refractory cardiogenic shock, despite optimal

medical therapy and correction of reversible factors, mechanical circulatory support may be beneficial. An intra-aortic balloon pump, by augmenting diastolic coronary perfusion and reducing afterload, can support the “stunned” myocardium and provide hemodynamic stabilization, allowing time for myocardial recovery over days to weeks.[2] Overall, the treatment and management of blunt cardiac injury demand a careful balance between standardized trauma protocols and individualized, physiology-driven cardiac care, with close interdisciplinary collaboration to optimize outcomes.

Differential Diagnosis

The evaluation of suspected blunt cardiac injury (BCI) requires careful consideration of a broad differential diagnosis, as many cardiac and traumatic conditions share overlapping clinical features. Distinguishing among these possibilities is essential because each condition carries unique management implications and varying degrees of urgency. One of the most common diagnostic challenges involves differentiating BCI-related arrhythmias from those arising due to preexisting cardiac disease, metabolic disturbances, or physiologic stressors associated with trauma. Trauma patients frequently exhibit tachyarrhythmias caused by pain, anxiety, hypovolemia, or electrolyte derangements, making it necessary to contextualize rhythm abnormalities within the overall clinical picture rather than attributing them automatically to myocardial contusion. Cardiac tamponade also figures prominently in the differential diagnosis, as it may present with hypotension, muffled heart sounds, and jugular venous distention—features that can mimic or coexist with manifestations of BCI. In trauma settings, tamponade is most often the result of penetrating injury, but blunt mechanisms can also cause hemopericardium secondary to cardiac rupture or laceration. Rapid assessment through bedside ultrasound is therefore indispensable. Similarly, abnormalities in cardiac wall motion may arise either from contusion-related myocardial dysfunction or from ischemic injury, creating diagnostic ambiguity, especially when ECG changes or biomarker elevations are nonspecific. Cardiogenic pulmonary edema and cardiogenic shock may occur in severe BCI due to impaired myocardial contractility or mechanical complications such as acute valvular regurgitation. However, these conditions must be differentiated from hemorrhagic shock, which is far more common in trauma and may initially present similar signs, including hypotension and tachycardia. Prompt recognition of hemorrhagic shock is crucial, as rapid control of bleeding and volume resuscitation take precedence in such cases. Distinguishing between cardiogenic and hypovolemic mechanisms often requires a combination of hemodynamic assessment, imaging, and laboratory interpretation [2][4][6][8].

Myocardial infarction is another important consideration, as ST-segment elevations or troponin

elevations may result from coronary artery injury, coronary dissection, or preexisting ischemic heart disease rather than blunt cardiac trauma. Careful assessment of mechanism of injury, risk factors, and diagnostic studies including coronary angiography may be needed to clarify the cause. Finally, acute valvular regurgitation—particularly involving the mitral or tricuspid valves—may mimic symptoms and signs of BCI and can result from traumatic papillary muscle rupture or chordae tendineae injury. Because valvular dysfunction can lead to rapid hemodynamic deterioration, its early recognition is essential. Altogether, a comprehensive differential diagnosis ensures that clinicians evaluating blunt cardiac injury maintain a broad perspective, integrating clinical findings with targeted diagnostic testing to accurately identify the underlying cause of cardiovascular instability in trauma patients [2][4][6][8].

Prognosis

The prognosis of blunt cardiac injury (BCI) is shaped by multiple interrelated factors, including the specific type and severity of cardiac damage, the presence or absence of associated traumatic injuries, and the patient’s underlying cardiovascular health. These variables contribute significantly to the clinical trajectory and long-term outcomes of affected individuals. In general, patients presenting with isolated BCI—manifesting solely as an abnormal electrocardiogram (ECG) or an elevated cardiac troponin I (cTnI) level—tend to experience more favorable outcomes compared with those who arrive hemodynamically unstable or with clear structural cardiac compromise. The latter group, often exhibiting high trauma scores and substantial multisystem injuries, is at greater risk for complications, prolonged recovery, and higher mortality.[9][17] Nonetheless, because the majority of patients diagnosed with BCI fall into the former category, the overall prognosis for the condition remains positive when appropriate diagnostic vigilance and monitoring are applied. Long-term outcomes after myocardial contusion, one of the more common forms of BCI, have been explored in prospective studies. In a notable investigation comparing 12 patients with documented myocardial contusion to 12 matched controls who sustained blunt chest trauma without myocardial contusion, no significant differences were identified at a 12-month follow-up regarding ECG findings or right and left ventricular function. These results suggest that uncomplicated myocardial contusion, in the absence of structural injury or persistent arrhythmia, generally does not contribute to long-term ventricular dysfunction or chronic cardiac pathology [9].

More recent prospective research continues to refine understanding of the long-term cardiac consequences of BCI. In a cohort of patients evaluated at 3 and 12 months following blunt thoracic trauma, those with documented myocardial contusion and associated wall motion abnormalities exhibited a variable recovery pattern. At the 3-month assessment,

10 of 17 patients continued to demonstrate persistent wall motion abnormalities, indicating that structural or functional myocardial changes may take several months to improve. By the 12-month follow-up, this number decreased to 4 of 17 patients, suggesting partial or substantial recovery in the majority of individuals over time. Importantly, despite these imaging findings, none of the patients demonstrated ECG abnormalities during exercise testing, and no cardiac-related symptoms or functional limitations were reported throughout the follow-up period.[1] These findings collectively underscore that while some patients with BCI may exhibit lingering radiologic abnormalities, these do not necessarily correlate with clinical symptoms or impaired cardiac performance. Most cases of mild to moderate BCI, including myocardial contusion without major structural injury, resolve without long-term sequelae. Conversely, patients with severe mechanical complications—such as papillary muscle rupture, septal defects, or coronary artery injury—have a more guarded prognosis and may require surgical intervention, intensive monitoring, and prolonged rehabilitation. In summary, the prognosis of BCI is highly dependent on the interplay between injury severity, associated trauma, and baseline cardiac health. While severe presentations carry significant risks, the majority of patients with isolated biochemical or electrical abnormalities experience favorable outcomes, often with full recovery and minimal long-term impairment [1][9][17].

Complications

Complications following blunt cardiac injury (BCI) are relatively uncommon when viewed across the entire spectrum of affected patients, particularly in those with minor or isolated myocardial involvement.[9][17] In cases of severe cardiac trauma, however, acute complications can be profound and demand immediate recognition and intervention. Patients who survive the initial impact and emergent phase may later experience sequelae that are closely related to the specific type and extent of their original cardiac injury. For example, those with significant structural damage, such as chamber rupture or valvular disruption that has been surgically repaired, may remain at risk for long-term alterations in ventricular function, valvular insufficiency, or arrhythmogenic substrates. Nevertheless, the majority of individuals with BCI—especially those whose presentation is limited to modest biomarker elevation or minor ECG abnormalities—do not develop lasting clinical consequences and recover fully without chronic impairment. Despite this generally favorable outlook, a number of late complications have been documented and warrant careful attention. Among these are delayed cardiac rupture, which can occur after an initially contained myocardial injury undergoes progressive weakening and eventual tearing; complete atrioventricular block, which may arise from damage

to the conduction system; and chronic heart failure, which can develop when myocardial contusion or structural injury results in persistent systolic or diastolic dysfunction.[9] Additional reported sequelae include pericardial effusion, which may be asymptomatic or evolve into hemodynamically significant tamponade, and constrictive pericarditis, a condition in which fibrotic thickening of the pericardium impairs diastolic filling and leads to symptoms of right-sided heart failure.[9] Although these complications occur in only a minority of cases, their potential severity justifies continued clinical vigilance [9][17]. In light of these considerations, best practice involves not only appropriate acute management and short-term monitoring but also medium-term follow-up. Reevaluation at approximately 3 to 6 months after the initial trauma is widely regarded as prudent.[9] During this interval visit, clinicians can reassess symptoms, repeat ECGs or echocardiography when indicated, and ensure that no delayed structural or functional abnormalities have emerged. This approach balances recognition of the overall low complication rate with the need to detect and manage the small subset of patients who may develop clinically significant late effects of blunt cardiac injury.

Patient Education

Deterrence and patient education play a crucial role in the comprehensive management of individuals who have experienced blunt cardiac injury. Even though long-term outcomes for most patients after hospital discharge are excellent, structured follow-up and informed self-monitoring are essential components of high-quality care. Patients should be clearly informed that, while the majority of BCIs resolve without lasting cardiac dysfunction, a small risk of delayed complications persists. Accordingly, they should receive explicit instructions to attend a follow-up evaluation, typically scheduled 3 to 6 months after the initial traumatic event, to reassess for any signs of residual or emerging cardiac injury and dysrhythmia. During hospitalization and at discharge, clinicians should provide education regarding the nature of BCI, the rationale for initial monitoring, and the importance of follow-up. Patients should be made aware of symptoms that warrant earlier medical attention, such as new or worsening chest pain, palpitations, syncope, unexplained dyspnea, or decreased exercise tolerance. Clear, jargon-free explanations help patients understand that such symptoms may reflect delayed arrhythmias, evolving structural cardiac issues, or other cardiovascular complications that can often be managed effectively if identified promptly. Empowering patients with this knowledge facilitates early recognition of clinically important changes and encourages timely re-presentation to healthcare services when necessary. In addition to symptom-focused education, reinforcing general cardiovascular health principles is beneficial,

particularly in individuals with preexisting cardiac risk factors. Guidance on smoking cessation, blood pressure and diabetes control, adherence to cardiac medications, and gradual return to physical activity can support overall recovery and reduce the impact of any underlying heart disease that might interact with the effects of BCI. For athletes or individuals involved in contact sports or high-risk occupations, specific counseling on safe return-to-play or return-to-work timelines, ideally in coordination with cardiology and occupational or sports medicine experts, may be appropriate. Ultimately, effective deterrence and patient education strategies rely on clear communication, written and verbal discharge instructions, and a well-defined plan for follow-up. By emphasizing both the generally favorable prognosis and the importance of surveillance for rare but serious delayed complications, clinicians help ensure that patients with blunt cardiac injury are not only treated appropriately in the acute phase but also supported in their long-term recovery and wellbeing [8][9][17].

Enhancing Healthcare Team Outcomes

Enhancing outcomes in patients with blunt cardiac injury depends heavily on coordinated, interprofessional care and an organized systems-based approach. The diagnostic challenge of BCI arises from its variable clinical presentation, the wide spectrum of injury severity, and the absence of universally accepted diagnostic criteria. In this context, maintaining a high index of suspicion in trauma patients is fundamental. Early use of electrocardiography and measurement of cardiac enzyme levels, such as troponin, provides a pragmatic initial screening strategy. Abnormal results may warrant hospital admission, continuous cardiac monitoring, and further evaluation with echocardiography, recognizing that some manifestations of BCI may not become apparent until up to 24 to 48 hours after injury.[1] Establishing local protocols and decision pathways based on available evidence can assist clinicians in applying these principles consistently. Achieving optimal patient outcomes requires effective collaboration among physicians, advanced practice providers, nurses, pharmacists, imaging specialists, and other allied health professionals. Emergency physicians and trauma surgeons are often the first to suspect BCI based on mechanism of injury and initial findings. Cardiologists and cardiothoracic surgeons contribute critical expertise in interpreting complex diagnostic results, planning advanced imaging, and determining the need for invasive interventions or surgery. Nurses play a central role in continuous monitoring, early recognition of hemodynamic or rhythm changes, and patient education at the bedside. Pharmacists help optimize medication regimens, particularly for antiarrhythmics, anticoagulants, and cardiovascular agents, and ensure that drug interactions and dosing are carefully managed. Interprofessional communication and care coordination are essential at

every stage, from prehospital care through emergency department resuscitation, inpatient monitoring, and discharge planning. Structured handoffs, shared care plans, and multidisciplinary rounds help align team members around patient-specific goals and ensure that important diagnostic clues are not overlooked. Even though many cases of BCI may ultimately prove to be clinically inconsequential, the true significance of the diagnosis remains debated, partly due to limited long-term outcome data. This uncertainty reinforces the need for systematic observation of patients with suspected BCI who are hemodynamically stable but present with risk factors or subtle abnormalities. A short period of monitored observation allows time for arrhythmias or evolving injuries to declare themselves while avoiding unnecessary prolonged hospitalization for very low-risk patients. Ongoing research and consensus-building efforts among clinicians are crucial to refine diagnostic criteria, standardize management strategies, and better characterize the long-term implications of blunt cardiac injury. By integrating emerging evidence into trauma protocols and fostering a culture of interprofessional collaboration, healthcare teams can enhance patient-centered care, reduce preventable complications, and continuously improve performance and safety in the management of this complex and often underrecognized condition [1].

Conclusion:

In summary, the management of blunt cardiac injury requires a balanced and vigilant approach that extends beyond the initial hospitalization. While the majority of patients experience an excellent prognosis and full recovery, the potential for severe, delayed complications in a minority justifies a structured long-term strategy. This strategy is twofold. First, it necessitates a systematic, interprofessional model of care where emergency physicians, trauma surgeons, cardiologists, nurses, and pharmacists collaborate seamlessly from diagnosis through discharge. Establishing clear protocols for initial screening with ECG and troponins, followed by monitored observation for at-risk patients, is fundamental to preventing acute adverse events. Second, effective patient education and a defined follow-up plan are crucial components of high-quality care. Empowering patients with knowledge of warning symptoms and the importance of a 3 to 6-month reevaluation ensures that any delayed complications are identified and managed promptly. Ultimately, by integrating coordinated, team-based acute management with diligent long-term surveillance and patient communication, healthcare providers can optimize outcomes, support full recovery, and mitigate the risks associated with this complex traumatic condition.

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