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Translational Pathophysiology

Review

Therapeutic translation across myocardial infarction and myocardial contusion: opportunities, boundaries, and bidirectional lessons

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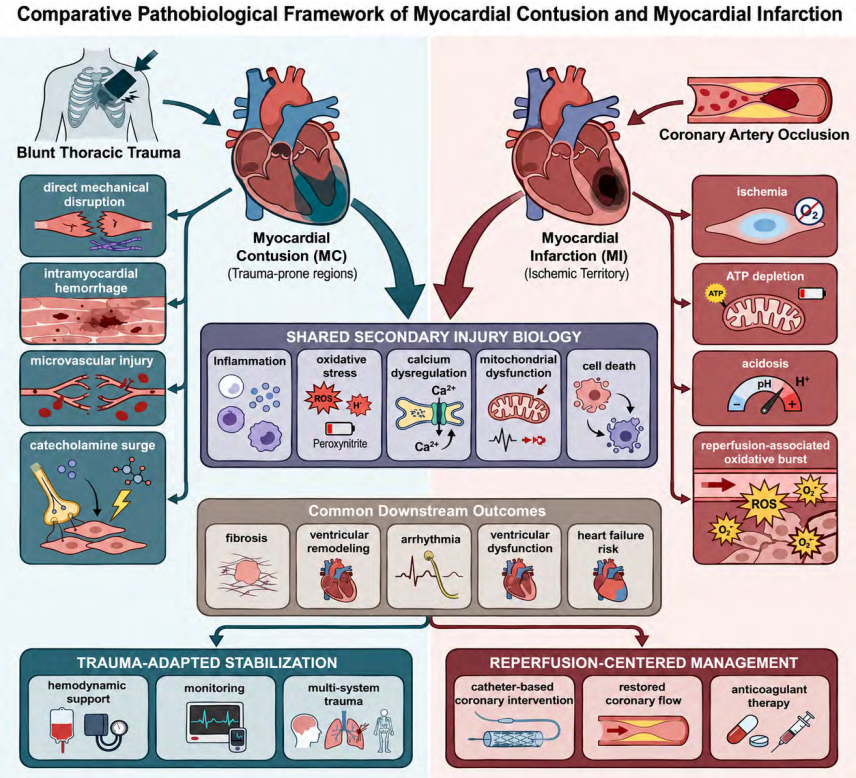
ABSTRACT

This review examines the asymmetric therapeutic relationship between myocardial contusion (MC) and myocardial infarction (MI). Although these two forms of myocardial injury arise from different initiating events, they converge across key secondary injury pathways, including inflammatory activation, oxidative stress, calcium dysregulation, cell death, fibrosis, and ventricular remodeling. Its aim is to clarify which elements of MI generate biologically portable hypotheses for MC, which remain clinically non-transferable, and how trauma reveals the contextual limits of canonical MI care. Evidence was retrieved from PubMed and CNKI primarily covering publications between January 2000 and January 2026 including experimental studies, clinical investigations, imaging studies, and guideline or consensus documents. The comparative literature indicates that the major divergence between MC and MI lies not in the mere presence of myocardial injury, but in the primary insult, the spatial organization of tissue damage, the logic of diagnostic interpretation, and the sequence of therapeutic decision-making. In this context, MI should be regarded not as a directly transferable treatment template for MC, but as a more mature source of mechanism-based hypotheses, particularly for the modulation of inflammatory amplification, oxidative injury, maladaptive remodeling, rhythm-risk surveillance, and biomarker-imaging integration. Conversely, MC is clinically informative less as a therapeutic analogue of MI than as a boundary condition that clarifies the dependence of myocardial injury management on etiology, bleeding risk, structural injury, and competing clinical priorities. Future research should prioritize trauma-specific phenotyping, multimodal diagnostic stratification, biomarker-imaging integration, and prospective evaluation of adjunctive targeted compatible with trauma care without delaying stabilization.

Graphical abstract



Key Words: blunt cardiac injury; ischemic myocardial injury; cardioprotection; biomarker-imaging integration; ventricular remodeling; rhythm-risk stratification; trauma-adapted management; translational pathobiology

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REVIEW
DIAGNOSIS, DIFFERENTIAL

Introduction

Myocardial contusion (MC) and myocardial infarction (MI) are usually discussed within separate clinical frameworks, yet comparison between them is increasingly valuable because the key question is no longer whether they look superficially similar, but how far therapeutic reasoning can travel across them. MC remains a diagnostically and clinically heterogeneous entity within blunt cardiac injury [1, 2], whereas MI is a more mature ischemic syndrome with clearer diagnostic and therapeutic architecture [3]. Their initiating insults are fundamentally different: mechanical trauma in MC and ischemia in MI, but both converge across several downstream pathways of myocardial injury. The relevance of this comparison is therefore therapeutic rather than merely descriptive: MI offers a disease model from which mechanism-based hypotheses may be drawn [4, 5], whereas MC clarifies why diagnostic and treatment algorithms cannot be transferred uncritically across etiologies [6].

Current work can be read through two complementary lenses. One emphasizes shared secondary injury biology, especially inflammation, oxidative stress, calcium dysregulation, mitochondrial dysfunction, and remodeling [7, 8]. The other emphasizes clinical non-equivalence, including differences in trigger, injury geometry, tempo, monitoring logic, and treatment priority [9, 10]. The mainstream view is not that one perspective displaces the other, but that biological overlap and clinical divergence must be interpreted together.

From a translational standpoint, this asymmetry is central. MI is managed through guideline-defined pathways organized around ischemic recognition, invasive evaluation, reperfusion or revascularization when indicated, antithrombotic therapy, and secondary prevention [9, 11]. Contemporary classification work and the universal definition further emphasize that MI is not a generic troponin-positive state but an ischemic syndrome requiring etiologic interpretation [12–14]. MC, by contrast, commonly arises in polytrauma, hemorrhagic shock, thoracic injury, and uncertain structural contexts. Its clinical pathway is therefore governed by stabilization, phenotyping, rhythm surveillance, and selective structural intervention rather than automatic entry into an acute coronary syndrome (ACS) algorithm [15–17].

Accordingly, this review examines MC and MI across four interrelated domains: initiating and secondary injury mechanisms, biomarkers and imaging, divergent clinical pathways, and therapeutic significance. Its aim is to clarify which elements of MI provide biologically portable hypotheses for MC,

which remain clinically non-transferable, and how the trauma setting exposes the contextual limits of canonical MI care. Literature searches were performed in PubMed and CNKI predominantly between January 2000 and January 2026 using key terms including “blunt cardiac injury”, “myocardial contusion”, “myocardial infarction”, “pathophysiological mechanisms”, “biomarkers”, “electrocardiographic and imaging findings”, “acute management”, “remodeling”, and “long-term outcomes”. Where necessary to clarify specific aspects of the publications under review, earlier studies were also considered. Only experimental models, clinical investigations, guidelines, and major reviews in English were included, while case reports were used selectively when relevant to rare complications or unusual clinical scenarios.

Shared pathobiological mechanisms

Primary insult and early secondary injury

The most fundamental difference between MC and MI lies in the initiating lesion. MC is trauma-driven and typically begins with direct mechanical disruption of cardiomyocytes, the microvasculature, and myocardial architecture. Depending on the force vector and site of impact, the resulting injury may include focal hemorrhage, edema, myofibrillar damage, and regional contractile dysfunction [3, 18], with the right ventricle often being particularly vulnerable because of its anterior position, with anteriorly located cardiac structures exposed to direct precordial force in some mechanisms [17, 18]. MI, by contrast, is ischemia-driven. Its primary lesion reflects abrupt and sustained impairment of coronary perfusion, most commonly due to acute atherothrombosis in type 1 MI, whereas acute infection and other systemic stressors can contribute to type 2 MI through oxygen supply-demand imbalance [19–21]. The current definitional literature further underlines that this ischemic setting is central to the meaning of infarction itself [13].

An additional temporal distinction is essential for therapeutic interpretation. In MI, particularly when the ischemic interval is limited, contractile dysfunction may precede irreversible cardiomyocyte death. This creates a clinically important window in which viable myocardium, postischemic stunning, and chronically dysfunctional hibernating myocardium may recover after timely reperfusion or revascularization [7, 8, 22]. Reperfusion remains indispensable for infarct-size limitation, but it may also intensify oxidative stress, calcium overload, inflammation, endothelial dysfunction, and microvascular impairment [7, 8]. MC follows a different sequence. Mechanical impact can produce immediate sarcolemmal disruption, myofibrillar damage, microvascular rupture, intramyocardial hemorrhage, and spatially irregular structural destruction before secondary inflammatory or metabolic amplification becomes clinically dominant [3, 18, 23]. Therefore, the reversible ischemic phenotypes of stunning and hibernation in MI should not be treated as direct analogues of primary traumatic tissue disruption in MC.

Despite this difference at onset, the early secondary injury response in the two conditions shows substantial convergence. Across both entities, the literature mainly centers on a common cluster of early disturbances: bioenergetic failure, impaired oxidative phosphorylation, intracellular acidosis, membrane instability, and calcium overload. In MC, these alterations are usually interpreted as consequences of tissue disruption, local hypoperfusion, trauma-related metabolic stress, and, in severe cases, post-traumatic cardiac dysfunction within shock physiology [23–25]. Severe blunt chest trauma studies also support an early metabolic and electrolyte component to this

injury environment [25]. In MI, the same downstream cascade is driven by ischemia and is often intensified during reperfusion [7, 8, 26].

A key conceptual point is that the overlap emerges downstream rather than upstream. The two diseases do not share a common trigger, but they do converge in the expansion phase of myocardial injury. This is precisely why comparison is biologically informative: it identifies a shared substrate of secondary damage without obscuring the fundamentally different nature of the primary insult.

Inflammation, oxidative stress, and calcium dysregulation

Among the shared mechanisms, inflammatory activation is one of the most consistently emphasized in literature. In MI, the dominant model describes a sequence in which necrotic cardiomyocytes release danger-associated signals, activate resident immune cells, recruit circulating leukocytes, and initiate a transition from injury amplification to repair [26–28]. In MC, the mechanistic literature is smaller and less standardized, but the prevailing view is that trauma likewise induces an acute inflammatory response characterized by oxidative imbalance, contractile impairment, and tissue-level repair signaling [24, 29, 30]. Experimental work further suggests trauma-associated shifts in structural and intercellular signaling that are compatible with this inflammatory phenotype [23, 30, 31].

Oxidative stress represents a second major area of convergence. Research in MI has moved from general recognition of reactive oxygen species injury toward a more detailed focus on mitochondrial dysfunction, reperfusion-associated oxidative burst, and redox-sensitive injury pathways [7, 32]. Lipid-lowering and plaque-oriented interventional studies remain important to MI care but should not be confused with generic transferability to traumatic injury [33]. In MC, the literature remains more limited, but current work broadly supports the view that oxidative injury arises from cellular disruption, inflammatory activation, mitochondrial damage, and depletion of endogenous antioxidant defenses [34, 35]. In this respect, the research trajectory has shifted from descriptive acknowledgment of post-traumatic injury toward more mechanism-based discussion of redox imbalance and metabolic vulnerability.

Calcium dysregulation can be regarded as a mechanistic bridge linking energetic failure, inflammation, and electrical instability. In both MC and MI, impaired production of adenosine triphosphate and membrane transport dysfunction favor intracellular calcium accumulation, with downstream effects on excitation-contraction coupling, protease activation, mitochondrial injury, and arrhythmogenic susceptibility [24, 25]. The immediate upstream drivers differ: trauma-related catecholamine excess may intensify calcium-mediated myocardial dysfunction after blunt chest trauma [25, 36, 37], whereas ischemia-reperfusion injury and sarcoplasmic reticulum-mitochondrial calcium signaling are more central in MI [7, 8]. The clinical relevance of this electrical vulnerability is underscored by the close relationship between ischemic injury and rhythm disturbance after MI [38].

Cell death, fibrosis, and remodeling

The major downstream consequence of these intersecting pathways is cardiomyocyte loss followed by structurally significant repair. In MC, experimental work has mainly focused on apoptosis-related signaling, caspase activation, and alterations in structural proteins involved in cytoskeletal integrity and intercellular coupling [30, 31]. In MI, literature is broader and more differentiated. This broader mechanistic literature is also supported by well-

established experimental models of MI and ischemia-reperfusion injury [39]. Beyond necrosis, current work increasingly emphasizes apoptosis, necroptosis, ferroptosis, and fibroblast-driven repair programs that shape infarct expansion and scar formation [26, 40, 41]. The inflammatory-remodeling literature also continues to support this broader reparative framework [27, 28].

Fibrosis and ventricular remodeling are also shared endpoints, but they follow different spatial and pathological logic. MI typically produces organized scar formation within a vascular territory and may drive remote ventricular remodeling, chamber dilation, and progressive systolic dysfunction [4, 42]. MC may show acute and late abnormalities visible on cardiac magnetic resonance imaging [43]. Emerging antifibrotic work underscores how central remodeling has become to post-MI therapeutic thinking [44]. MC more often produces focal, heterogeneous, and trauma-shaped repair that reflects injury geometry rather than coronary anatomy [45, 46]. Contemporary state-of-the-art reviews likewise emphasize the irregular, mechanically determined distribution of traumatic cardiac injury [47]. Thus, while both diseases can culminate in wall-motion abnormalities, arrhythmogenic substrate formation, and impaired ventricular reserve, the architecture of repair differs substantially.

Taken together, the literature supports a general framework in which the shared biology of MC and MI is concentrated in secondary injury and repair, whereas the most important differences lie in the origin, distribution, and clinical expression of that injury. This distinction is central to any meaningful comparison between the two conditions.

Divergent clinicopathological pathways

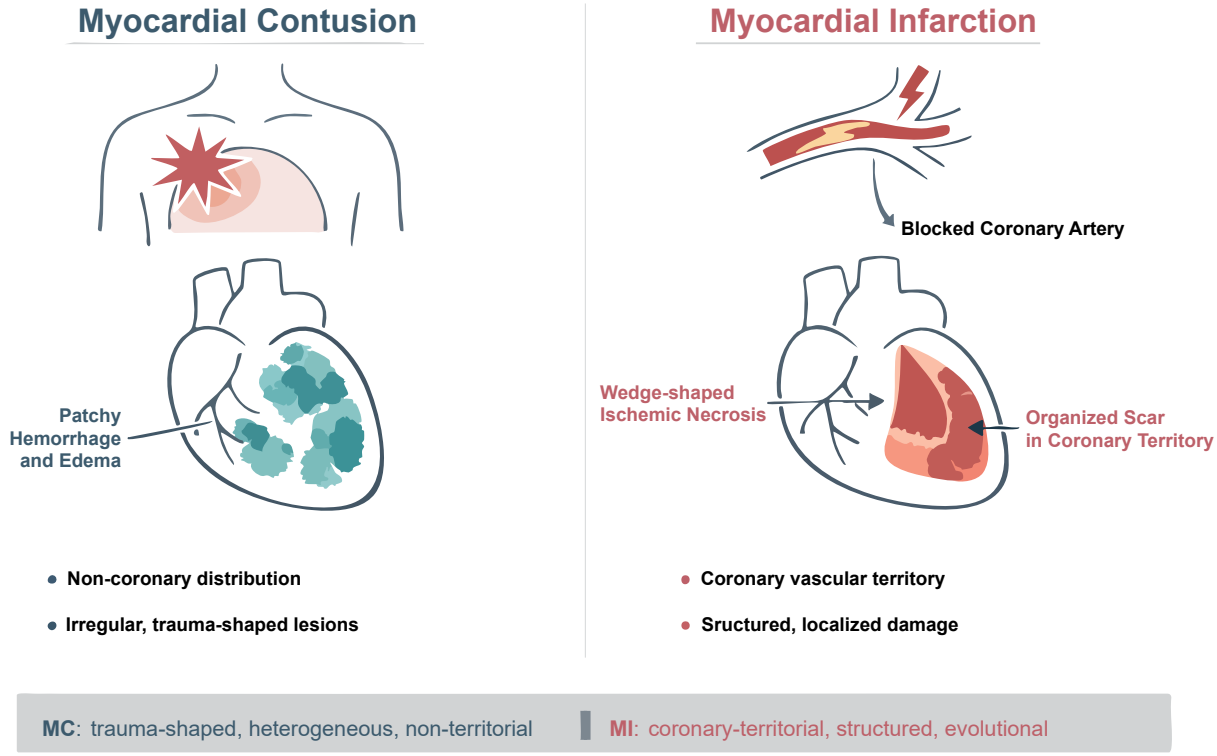
Morphology, injury distribution, and pathology

The most clinically relevant divergence between MC and MI lies in the morphology and spatial logic of injury. MI is typically organized by coronary anatomy. Its pathology evolves along a relatively recognizable trajectory from ischemia to coagulative necrosis and finally to scar formation within a defined vascular territory [4, 14]. MC, in contrast, is structurally heterogeneous. It is often focal, sometimes epicardial or transmural, and may include intramyocardial hemorrhage, patchy tissue disruption, and sharply demarcated lesions that do not correspond to coronary distribution [45, 48]. Case-based and forensic observations likewise illustrate how blunt impact can produce irregular injury patterns that are anatomically and mechanically determined rather than territory-based [45, 46, 48].

This difference in injury geometry has important interpretive consequences. In MI, coronary territory remains central to the interpretation of symptoms, biomarker kinetics, electrocardiographic changes, and imaging findings. In MC, however, lesion distribution is often patchy, non-specific, or clinically obscured by associated thoracic trauma. As a result, literature increasingly favors multimodal assessment over reliance on any single diagnostic modality [47, 49]. Review data and imaging studies reinforce this preference for combined testing strategies in blunt cardiac injury [50, 51]. Multimodality imaging reports of cardiac contusion complications further support phenotype-based interpretation in ambiguous cases [52, 53]. Figure 1 summarizes this contrast by visually comparing the spatial and morphological logic of injury in MC and MI.

A broader trend in the field is therefore apparent. Work on MI has long emphasized territory-based pathological reasoning, whereas research on MC is gradually moving toward phenotype-based characterization, in which

FIG. 1. Comparative morphology and injury geometry of myocardial contusion and myocardial infarction



Note: MI — myocardial infarction; MC — myocardial contusion.

structural heterogeneity and clinical context are treated as defining features rather than diagnostic obstacles.

Biomarkers, electrocardiography, and imaging

Diagnostic evaluation represents another major area of divergence. In MI, high-sensitivity troponin is central but must be interpreted with symptoms, serial change, and electrocardiographic evidence rather than in isolation [54, 55]. Assay thresholds and assay transitions can further influence downstream clinical decisions [5, 56]. In MC, troponin is better understood as one component of risk stratification and exclusion. The dominant clinical approach supports combining admission electrocardiography with troponin testing, because concurrent normal findings substantially reduce the likelihood of clinically significant blunt cardiac injury [1, 57]. Experimental and clinical work also supports the biological relevance of troponin release after blunt cardiac trauma [58, 59]. Additional trauma studies underscore that risk depends on the broader injury pattern rather than biomarker elevation alone [60, 61]. This distinction is especially important when traumatic myocardial injury is evaluated against type 2 MI. A posttraumatic troponin rise should not be assigned reflexively to MC, type 1 MI, or type 2 MI. The universal definition requires evidence of acute ischemia before myocardial injury can be classified as infarction, whereas type 2 MI denotes ischemic injury caused by oxygen supply-demand imbalance without acute atherothrombosis [14, 20, 21]. In blunt chest trauma, hypoxemia, anemia, hypotension or hemorrhagic shock, and tachycardia or tachyarrhythmia may produce an oxygen supply–demand mismatch compatible with type 2 MI when accompanied by evidence of acute ischemia [14, 62, 63]; conversely, MC is favored by a compatible impact mechanism, focal traumatic wall-motion abnormality, pericardial or valvular injury, noncoronary injury geometry, and computed tomography (CT),

echocardiographic, or cardiac magnetic resonance evidence of structural trauma [52, 64, 65]. Selective CT and trauma-specific guidance further support imaging-based phenotyping when the mechanism and biomarker pattern are ambiguous [66, 67]. Therefore, diagnostic classification should integrate the mechanism of injury, hemodynamic trajectory, serial electrocardiogram (ECG) changes, troponin kinetics, imaging phenotype, and likelihood of acute coronary thrombosis before selecting an ACS pathway, anticoagulation, coronary angiography, or trauma-specific monitoring [9, 11, 68]. This distinction also aligns with practical recommendations for separating type 2 MI from acute nonischemic myocardial injury [62, 63, 69]. Table 1 summarizes the practical distinction between myocardial contusion and type 2 MI in trauma settings.

Electrocardiography follows a similar pattern of divergence. In MI, ST-segment elevation, dynamic ischemic change, and rhythm disturbance are interpreted within a structured and clinically actionable ischemic framework [9, 13]. Contemporary ACS guidance further reinforces this interpretive hierarchy [11]. In MC, electrocardiographic abnormalities may include sinus tachycardia, conduction delay, bundle branch block, ectopy, and nonspecific repolarization change, but these findings are neither sufficiently sensitive nor sufficiently specific to define diagnosis on their own [47, 70]. Delayed conduction disturbance after blunt cardiac injury also highlights the need for continued surveillance in selected patients [71, 72]. Trauma cohorts and experimental ECG studies show that arrhythmia burden reflects injury heterogeneity and procedural context with these observations summarized in a review that presents the full framework [73]. Experimental and review-based evidence supports the use of ECG primarily as a screening and monitoring tool in MC [1, 67, 74].

Imaging plays a broader phenotyping role in MC than in uncomplicated MI. Echocardiography remains valuable in both disorders, but in trauma it is especially useful for identifying wall-motion abnormalities, pericardial effusion, valvular injury, septal defects, and occult structural complications [47, 49, 50]. Cardiac magnetic resonance and selected computed tomography approaches further extend this role by helping distinguish blunt cardiac injury from acute coronary syndromes in diagnostically uncertain settings [18, 52]. Dual-energy CT feasibility data support this selective diagnostic expansion [66, 75]. Natriuretic peptide studies are also suggestive: NT-proBNP has been explored as a marker of blunt cardiac contusion [76], whereas after MI it retains prognostic value for subsequent ventricular risk [77, 78]. Overall, diagnostic literature suggests a clear trend: whereas MI relies on a comparatively standardized triad of symptoms, electrocardiography, and troponin dynamics, MC increasingly requires multimodal phenotyping adapted to the trauma context.

Table 1. Practical differentiation between myocardial contusion and type 2 MI in trauma settings

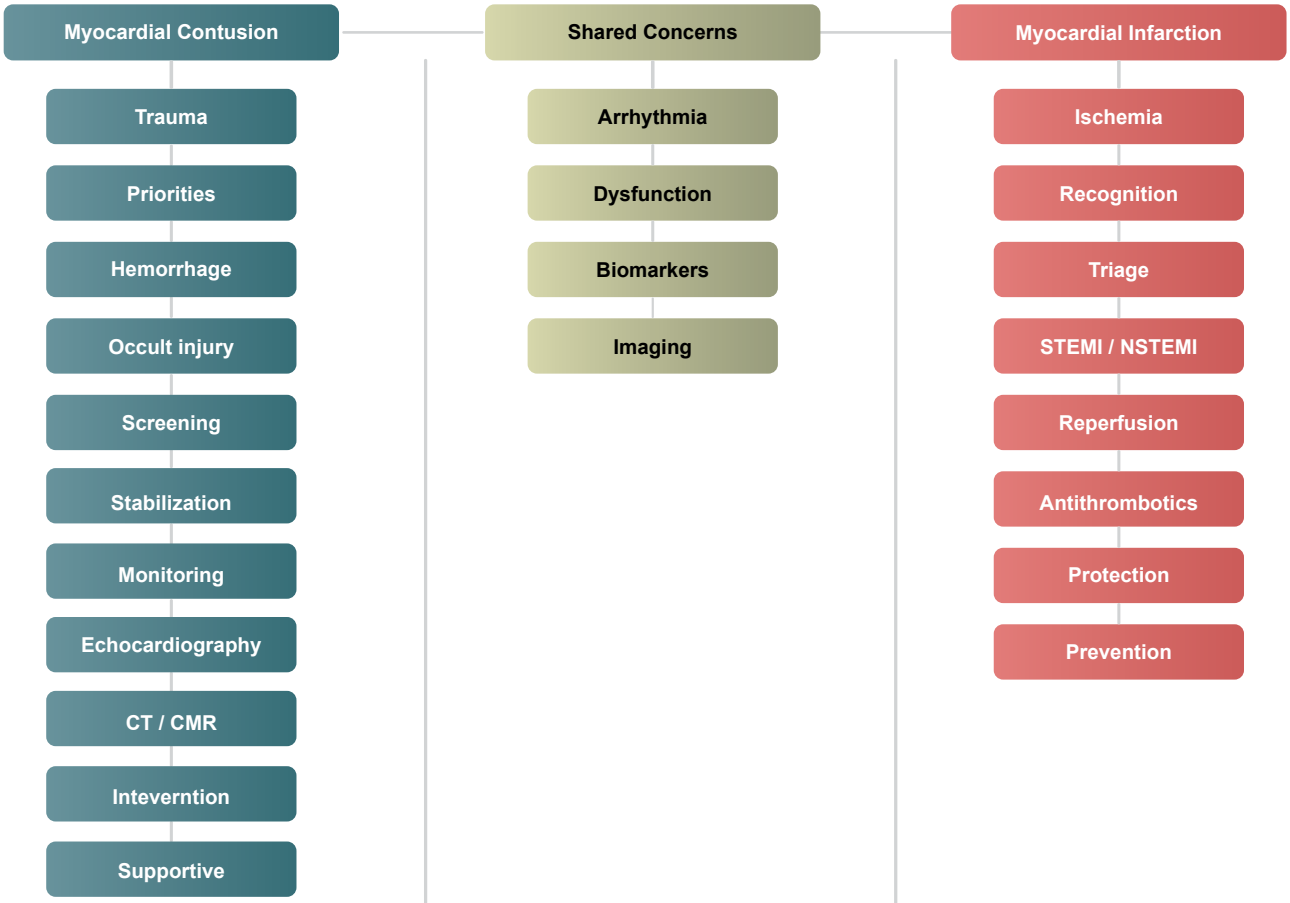
Dimension	Myocardial contusion	Type 2 myocardial infarction
Mechanism	Direct mechanical injury [3]	Supply-demand ischemia [14]
Trigger	Blunt chest trauma [3]	Anemia, hypoxemia, shock, tachyarrhythmia [14]
Electrocardiogram	Arrhythmia or conduction change [1]	Dynamic ischemic ST-T change [4]
Troponin kinetics	Variable trauma-related elevation [4]	Rise/fall with ischemic context [14]
Imaging	Focal noncoronary injury [5]	Ischemic or territory-based pattern [63]
Management	Stabilize, monitor, treat trauma [1]	Correct trigger; selective coronary work-up [63]

Therapeutic priorities and clinical pathways

The clearest divergence between MC and MI emerges in treatment strategy. MI is fundamentally an ischemia-centered and reperfusion-oriented disease, with care organized around rapid ischemic recognition, reperfusion or invasive evaluation when indicated, antithrombotic therapy, lipid-lowering therapy, and secondary prevention within contemporary ACS guideline frameworks [9, 11]. In parallel, cardiogenic shock, mechanical complications, post-infarction ventricular dysfunction, medication adherence, and long-term remodeling prevention are managed within an increasingly standardized continuum of care [79–81]. Mechanical complications after acute MI represent a distinct post-infarction management domain [82, 83].

MC follows a different clinical logic. The priority is not reperfusion but stabilization. Management is centered on hemodynamic support, rhythm monitoring, treatment of associated thoracic or extracardiac injuries, and selective evaluation of structural complications [1, 84]. Broader reviews of blunt thoracic and blunt cardiac trauma likewise frame MC within stabilization-first care pathways [15, 85]. In selected severe cases, suspected blunt cardiac injury may require urgent procedural or surgical evaluation for hemodynamic instability, tamponade, rupture, septal or valvular injury, traumatic arrest, or other mechanically significant cardiac lesions [67, 84, 86]. Trauma-focused resuscitation literature also supports emergency preservation or thoracotomy in selected catastrophic scenarios [87, 88]. Across age groups and procedural

FIG. 2. Divergent clinical pathways in traumatic and ischemic myocardial injury



Note: shared downstream injury does not imply identical management; myocardial contusion is stabilization-centered; myocardial infarction is reperfusion-centered; CT — computed tomography; CMR — cardiovascular magnetic resonance; STEMI — ST-elevation myocardial infarction; NSTEMI — non-ST-elevation myocardial infarction.

settings, arrhythmia surveillance and scenario-specific resuscitation remain central in suspected cardiac contusion [16, 73, 89]. Figure 2 summarizes this divergence in clinical logic by contrasting the stabilization-centered pathway of MC with the reperfusion-centered pathway of MI, while also highlighting the shared importance of rhythm surveillance and ventricular function assessment.

The major therapeutic conclusion of comparative literature is therefore twofold. First, direct transfer of MI algorithms to MC is generally inappropriate. Second, MC should not remain therapeutically under-theorized. The relevant opportunity is to test trauma-compatible adjunctive strategies aimed at inflammatory amplification, oxidative stress, receptor-signaling pathways, and maladaptive remodeling [34, 35]. Existing translational reviews of cardiac contusion provide further experimental rationale for such an approach [90, 91]. Experimental anti-inflammatory and biologically targeted interventions remain promising but investigational, and any future use in MC must be integrated without delaying life-saving trauma care [87, 92].

MI therefore provides mechanism-based hypotheses for MC, whereas MC does not offer a symmetrical treatment template for MI. Its reverse contribution is different: it reminds clinicians that myocardial injury management is inseparable from etiology, bleeding risk, structural injury, and treatment sequence.

Clinical significance of the comparison

The clinical value of comparing MC with MI lies in defining an asymmetric translational relationship. MI provides the more mature therapeutic framework, so its principal contribution to MC is conceptual: it supplies candidate mechanisms for adjunctive cardioprotection, remodeling control, and risk-oriented monitoring that may later be tested in trauma-specific settings [9, 11]. Reperfusion and ischemia-reperfusion literature provides the most relevant mechanistic basis for such hypothesis generation [7, 8]. Antifibrotic and remodeling-oriented work in MI further sharpens this hypothesis-generating role [42, 44]. MC, by contrast, does not offer a symmetrical treatment template for MI.

Conversely, MC's contribution to MI is qualitatively different. Because blunt cardiac injury is managed within a spectrum of bleeding risk, structural uncertainty, competing injuries, and staged stabilization, MC highlights the conditions under which canonical MI care remains appropriate and the point at which algorithmic transfer becomes misleading [4, 16]. Recent trauma-focused guidance makes this boundary problem even more explicit [67, 86]. This is why the key distinction is not between "similar" and "different" diseases, but between mechanistic portability and protocol portability.

A further contribution of the comparison is that it points toward a research agenda. The most urgent needs in MC include standardized phenotyping, better integration of biomarkers with imaging and monitoring, trauma-compatible diagnostic algorithms, and more systematic evaluation of ventricular and arrhythmic outcomes [17, 50]. Recent narrative and biomarker-focused reviews reinforce this agenda by emphasizing molecular characterization, biomarker validation, multimodal assessment, and the need for more systematic clinical data collection in blunt cardiac injury [17, 93]. More broadly, these priorities echo the developmental trajectory of MI research, in which descriptive pathological understanding was gradually linked to risk stratification and mechanism-based care. Based on this comparative synthesis, Table 2 summarizes the key domains that distinguish MC and MI in pathogenesis, diagnosis, and treatment, while also highlighting areas of partial overlap.

Table 2. Comparative characteristics of myocardial contusion and myocardial infarction across key domains of pathogenesis, diagnosis, and treatment

Domain	Myocardial contusion	Shared focus	Myocardial infarction
Injury mechanisms	Blunt trauma; patchy injury	Secondary injury biology	Ischemia-reperfusion; territorial loss
Biomarkers and imaging	Screening and phenotyping	Same modalities; different meaning	Risk stratification; coronary angiography
Clinical pathways	Stabilization in polytrauma	Rhythm and function assessment	Rapid ischemic triage
Therapeutic significance	Supportive and investigational care	Selective translational relevance	Reperfusion-centered management

The significance of this review is not that it proposes a unified model of myocardial injury. Its significance is that it refines the terms of comparison. It shows that true biological overlap should stimulate hypothesis-driven research, whereas superficial similarity should not justify therapeutic conflation. That distinction is precisely what makes the comparison clinically useful.

Limitations

Several limitations should be acknowledged. First, this is a narrative review rather than a formal systematic review or meta-analysis; accordingly, the present synthesis is interpretive and thematic rather than quantitatively pooled. Second, the evidence base for MC remains smaller and more heterogeneous than that for MI, with greater dependence on retrospective cohorts, mixed trauma populations, case series, and experimental models. Third, direct head-to-head comparative studies are scarce, which means that some of the conclusions necessarily derive from structured comparison across adjacent literatures rather than from unified datasets. Fourth, diagnostic definitions, biomarker thresholds, and imaging protocols for MC remain insufficiently standardized, limiting cross-study comparability.

Finally, this review primarily contrasts MC with the canonical ischemic architecture of MI, especially type 1 MI and reperfusion-centered ACS care. Although type 2 MI is addressed as a supply-demand mismatch syndrome, this article does not provide a complete diagnostic algorithm for separating MC from type 2 MI in trauma populations. This limitation is clinically relevant because polytrauma, hypoxemia, anemia, shock, tachyarrhythmia, systemic inflammation, and adrenergic stress may all produce dynamic troponin elevation and ischemic myocardial injury without primary coronary thrombosis; at the same time, MC may coexist with or mimic these processes through direct structural damage. Future comparative studies should therefore evaluate MC, type 2 MI, and acute nonischemic myocardial injury within a shared but etiologically stratified diagnostic framework (Table 3) [63, 66, 69].

Table 3. Research agenda for advancing comparative understanding of myocardial contusion and myocardial infarction

Stage 1 Current limitations	Stage 2 Immediate priorities	Stage 3 Mechanistic opportunities	Stage 4 Clinical translation
• Small evidence base • Heterogeneous definitions • Limited standardization • Scarce comparative data	• Trauma-specific phenotyping • Biomarker-imaging integration • Rhythm-risk stratification • Standardized follow-up	• Inflammation • Oxidative injury • Calcium dysregulation • Remodeling targets	• Selective intervention • Trauma-compatible algorithms • Risk-adapted surveillance • Precision supportive care

These limitations are not merely methodological constraints; they also reflect the current developmental stage of the field. In this sense, the uneven maturity of the two evidence bases is itself one of the central findings of the comparison.

Conclusions

MC and MI are linked by convergent secondary injury biology, yet they remain clinically non-equivalent conditions defined by distinct initiating insults, spatial patterns of damage, diagnostic frameworks, and therapeutic priorities. Their comparison is therefore informative only when mechanistic overlap is interpreted alongside irreducible differences in clinical context.

The central conclusion of this review is that the relationship between MC and MI is translationally asymmetric. MI provides a more mature mechanistic framework from which biologically plausible hypotheses may be derived for MC, particularly in relation to inflammatory amplification, oxidative injury, maladaptive remodeling, rhythm-risk surveillance, and integrated biomarker–imaging assessment. However, this translational relevance is selective rather than algorithmic. Reperfusion-centered management, routine ACS antithrombotic strategies, and coronary-occlusion-based decision pathways remain specific to ischemic disease and should not be extrapolated uncritically to traumatic myocardial injury.

Conversely, the value of MC lies not in serving as a therapeutic analogue of MI, but in exposing the contextual limits of canonical MI care. By embedding myocardial injury within a trauma setting shaped by bleeding risk, structural uncertainty, competing injuries, and staged stabilization, MC makes clear that myocardial injury management cannot be separated from etiology and clinical sequence. Future research should prioritize trauma-specific phenotyping, multimodal stratification, and prospective evaluation of adjunctive targeted therapies that can be integrated into trauma-first care without delaying stabilization.

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