Myocardial Injury Secondary to Blunt Thoracic Trauma in Dogs: Incidence and Pathophysiology*

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ABSTRACT: Blunt thoracic trauma to humans and dogs may result in myocardial injury. Myocardial injuries commonly manifest as arrhythmias within the first 48 hours following a traumatic event. These injuries may be occult or may result in delayed onset of clinical signs and, therefore, are frequently overlooked. This article aims to increase the awareness of myocardial injuries, review the current literature, and discuss the mechanisms and physiology of myocardial injury secondary to blunt thoracic trauma in dogs.

The first reports in the human literature of myocardial injury secondary to blunt thoracic trauma appeared in the 1920s.1,2 Since then, numerous papers and editorials have been published that discuss the classification, prevalence, diagnosis, clinical significance, and treatment of cardiac injuries. In humans and dogs, the prevalence and clinical significance of myocardial injuries caused by blunt thoracic trauma remain controversial because of the difficulty in confirming this diagnosis.3 Because of prehospital death and a lack of recognition of clinical signs, it is theorized that the true prevalence of clinically significant myocardial injury in humans may actually be higher than reported.1,2

In the veterinary literature, there are few reports that discuss the diagnosis and treatment of myocardial injuries in dogs. However, the term traumatic myocarditis has been used frequently in the literature to describe presumed myocardial injury and arrhythmias associated with thoracic trauma in dogs.4 In dogs and humans, intermittent or continuous electrocardiography has been traditionally used to identify arrhythmias following blunt thoracic trauma.5–12 Recent evi-

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Intrathoracic injuries are often overlooked on initial physical examination in the face of severe trauma.

In dogs and humans, the prevalence of myocardial injuries resulting from blunt thoracic trauma remains controversial.

Injury to the myocardium can cause decreased membrane potentials, spontaneous myocyte depolarization, and arrhythmias.
Incidence suggests that physical examination findings and intermittent electrocardiograms (ECGs) may be unreliable in identifying intrathoracic injuries, cardiac arrhythmias, and myocardial injuries. The controversy surrounding myocardial injuries in traumatized patients is due to the difficulty in confirming the diagnosis without gross or histologic evaluation of the heart. This article discusses the prevalence, etiology, and cellular and structural cardiac damage that may occur secondary to thoracic trauma as well as the likely consequences of these injuries.

**INCIDENCE**

The reported prevalence of human myocardial injuries secondary to blunt thoracic trauma ranges from 8% to 95%. Reported variations in the frequency of myocardial injuries in dogs are similar to those described in humans. Five studies (three prospective, two retrospective) have examined the prevalence of presumed myocardial injury in dogs and report a range from 10% to 96%. Selzer et al prospectively evaluated 100 dogs with radiographic evidence of skeletal trauma resulting from automobile injury for concurrent thoracic trauma. Within 12 hours of admission and before anesthesia, thoracic radiography, ECGs (lead II), and arterial blood gas samples were obtained from each patient. A 17% prevalence of cardiac arrhythmias in all dogs sustaining skeletal injury was found. The prevalence of cardiac arrhythmias increased to 30% in animals with both skeletal injury and evidence of thoracic injury. The study also found that thoracic injury was not recognized on routine physical examination in 45 of 57 (79%) dogs, suggesting myocardial injuries may be overlooked in many patients.

A recent prospective study that used continuous ambulatory electrocardiography (i.e., Holter monitoring) to evaluate electrocardiographic features in dogs following trauma discovered abnormalities in 96% (n = 30) of patients. Although the rhythm disturbances found in this study population were considered infrequent (i.e., <100 ventricular ectopic complexes per day) in 62% of dogs, frequent arrhythmias (i.e., >4,000 ventricular ectopic complexes per day) were discovered in 16% of the study population. At least one episode of ventricular tachycardia (four or more successive ventricular ectopic complexes) was identified in 43% of dogs examined. Arrhythmias were identified during initial ECG (before Holter monitoring) in only four dogs, with the remaining abnormalities detected during continuous monitoring. These findings indicate that many arrhythmias in traumatized dogs are likely to be overlooked when ECGs are performed at presentation only; however, continuous cardiac monitoring can improve the likelihood of detecting abnormal ECG findings in severely injured dogs.

Another recent prospective study of traumatized dogs compared ECG findings with serum analysis for biochemical markers of myocardial cell integrity. The biochemical markers of myocardial cell integrity (i.e., cardiac troponin I and T, creatine kinase–myocardial band) analyzed in this study were suggestive of myocardial injury in 58% of the dogs examined. Relevant ECG abnormalities (e.g., supraventricular tachycardia, atrial or ventricular premature contraction, ventricular tachycardia, atrioventricular conduction blocks, bundle branch blocks, ST segment elevation greater than +.15 mV, electrical R wave alternans, intermittent change in P wave polarity) were observed in 30% of dogs.

This study found no significant differences in serum concentrations of the biochemical markers of myocardial injury between dogs with and without relevant ECG abnormalities. These results suggest that ECG abnormalities are not specific indicators of myocardial cell injury in dogs with blunt chest trauma. It should be noted that intermittent—and not continuous—ECG recordings were used to identify conduction disturbances in this study.

Two recent retrospective studies examining pulmonary injuries in dogs sustaining blunt thoracic trauma also noted cardiac arrhythmias. The first study evaluated the records of 143 traumatized dogs with radiographic evidence of pulmonary contusion and found cardiac arrhythmias in 10% of this population. The second retrospective study examined 10 traumatized dogs requiring ventilatory support due to the severity of their pulmonary injuries. Forty percent of the dogs (n = 4) in this study were noted to have ventricular arrhythmias.

When comparing studies, it is evident that the prevalence of arrhythmias and suspected myocardial injuries remains a controversial subject in both humans and dogs. Variations in study design, terminology, diagnostic modalities, and criteria used to identify myocardial injuries contribute to the wide range in the reported frequency of this type of injury in both the human and veterinary literature. However, the results of these studies agree that myocardial injuries are often overlooked.

**ETIOLOGY AND MECHANISM OF INJURY**

Thoracic trauma is common in dogs injured by automobiles. Currently, the exact mechanism of myocardial injury following trauma is unknown and most likely remains multifactorial in origin. Research in humans has shown that a direct thoracic blow may result in an alteration in thoracic diameter of up to
The elastic nature of the thoracic cage in humans and animals subjects its contents to compressive, concussive, and penetrating injuries. The most common mechanism of myocardial injury in dogs is secondary to lateral chest compression. It has been proposed that distortion of the thoracic cage results in a rise in intrathoracic and intracardiac pressures, causing shearing stresses within the myocardium powerful enough to result in contusions.

Pandian et al measured intracardiac pressures before and after iatrogenic thoracic trauma to dogs. The study found significant increases in left and right end-diastolic pressures and decreases in left ventricular systolic and aortic pressures following an injury. Although these intracardiac pressure changes were considerable, they did not result in significant changes in peripheral pressures. Rapid elevations in systemic blood pressure during chest or abdominal compression have been suggested to result in injury to the coronary vessels or cardiac valves or rupture of the ventricular wall or septum. In addition, the suspended position of the heart within the thoracic cavity subjects it to potential concussive injury from forceful contact with the ribs, sternum, and vertebrae when rapid acceleration or deceleration occurs. The severity of injury caused by trauma is affected by the rate and magnitude of the impacting force as well as the volume of soft tissue absorbing that force. Additional factors that may influence the severity of injury include the position of the animal relative to the direction of the traumatic force and whether the animal is aware of the impending event and is able to initiate a protective response.

Conditions other than direct injury to the heart may also cause arrhythmias in traumatized dogs. Physiologic alterations associated with trauma and shock, such as metabolic acidosis, hypoxia, electrolyte imbalance, intracranial injuries, and catecholamine release, can predispose the heart to the development of arrhythmias.

**PATHOPHYSIOLOGY**

In a study of 25 dogs in which blunt chest trauma was delivered using a captive bolt pistol, Pandian et al correlated histopathologic areas of myocardial injury with areas of injury found during echocardiographic examination. In this model, the impact was delivered randomly to either the right or left thorax, resulting in different areas of myocardial injury. When the trauma was delivered to the left side of the chest, abnormalities were located primarily in the craniolateral wall of the left ventricle. Right-sided chest trauma produced septal and right ventricular wall damage. Injury and subsequent malfunction of the right ventricle may indirectly play a significant role in the development of functional abnormalities of the left ventricle. Right ventricular malfunction can cause a decrease in ejection fraction and an increase in end-diastolic volume, resulting in a leftward shift of the interventricular septum into the lumen of the left ventricle. The septal shift decreases the volume of the left ventricle, which is available to fill with blood, resulting in depression of left ventricular ejection fraction and potential pump failure.

Pathologic findings in the traumatized heart were characterized by localized edema, ecchymosis, and intramyocardial hematoma formation. In this study, no evidence of injuries to the valves or coronary arteries was identified on gross pathologic evaluation. The area of myocardial injury was often transmural with the epicardial surface being more severely affected. Multiple clinical reports have described similar gross and microscopic pathologic findings following traumatic injury.
Radioactive labeled microsphere perfusion studies have shown that regional myocardial blood flow remains normal in the injured region. Thus hypoperfusion or ischemia does not appear to be responsible for impairment of function in these areas. \textsuperscript{14}

Arrhythmias and conduction defects are the most common physiologic consequences to myocardial injuries in humans and dogs. \textsuperscript{5–8,15,18,36,37} Trauma to the myocardium lowers the resting membrane potential (it becomes less negative and moves closer to the threshold potential) as well as the ratio of effective refractory period to action potential duration in damaged myocardial cells\textsuperscript{39} (Figure 1). In addition, myocardial trauma results in alterations of sodium and calcium currents across cell membranes and increases the availability of intracellular calcium. This combination of intracellular derangements secondary to trauma can potentiate arrhythmogenesis.\textsuperscript{39} The arrhythmogenic effects of myocardial injuries are enhanced by acidosis, hypoxia, and increased catecholamine release that often occurs concurrently in trauma patients.\textsuperscript{6,7,39} Acidosis, hypoxia, and catecholamines all promote alterations in membrane transport and permeability of cations (e.g., sodium, potassium, calcium) that lead to a decrease in resting membrane potential, which facilitates depolarization.\textsuperscript{39,40} These changes result in the loss of organized myocardial cell depolarization, which is manifested as cardiac arrhythmias.\textsuperscript{7,39} ECG abnormalities are detected when the injured myocardium becomes the site of the most rapid impulse formation, overcoming the sinus node as the dominant (overdrive) pacemaker.\textsuperscript{39} The new overdrive pacemaker, originating in the injured myocardium, propagates the arrhythmia by depolarizing the sinus node before it has a chance to fire and recapture the cardiac rhythm.\textsuperscript{39}

The most common arrhythmias encountered secondary to canine myocardial injuries include premature ventricular contractions, ventricular tachycardia, and nonspecific ST segment elevation or depression\textsuperscript{5–8,14,36,41} (Figure 2). Although ST segment abnormalities may be indicative of myocardial infarction/ischemia, they also have been reported to occur secondary to electrolyte imbalance, digitalis administration, cardiac trauma, and pericarditis.\textsuperscript{39,42} Secondary ST segment changes may follow abnormalities of the QRS complex, such as ventricular premature contractions.\textsuperscript{39} Although less frequent than ventricular arrhythmias, other arrhythmias reported in dogs with chest trauma include atrial fibrillation, sinus arrest with ventricular or junctional escape complexes, and second- and third-degree atrioventricular block.\textsuperscript{5,7,9,11,36,43,44}

**SUMMARY**

Although myocardial injuries may cause significant alterations in cardiac function in traumatized dogs, they are often overlooked in the face of severe trauma. Ventricular arrhythmia is the most common abnormality caused by direct myocardial injury.\textsuperscript{5–8,15,18,36,37}

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**Figure 2**—Lead II ECG tracings demonstrating the following arrhythmias: \textit{A} = premature ventricular contractions (arrows, 25 mm/sec, 0.5 cm/mV); \textit{B} = ventricular tachycardia (25 mm/sec, 0.25 cm/mV); and \textit{C} = nonspecific ST segment depression (arrows, 50 mm/sec, 1 cm/mV).
Although ECG monitoring has been traditionally used to diagnose myocardial injuries, the onset of arrhythmias associated with these injuries is often delayed, making recognition difficult. 5-7,10,18,43 Arrhythmias are likely secondary to alterations in transport of cations (e.g., calcium, potassium, sodium) across the membranes of injured myocytes, resulting in a decrease of resting membrane potential, aberrant firing of injured cells, and loss of organized myocyte depolarization. 19,40 The controversies that plague myocardial injuries secondary to blunt thoracic trauma are due to the lack of a noninvasive diagnostic gold standard, differing opinions on the significance of these injuries and the arrhythmias that result, and inconsistency in terminology and definitions used by clinicians and researchers.

REFERENCES


1. The most common mechanism of myocardial injury in dogs is secondary to
a. penetrating injuries.  c. concussive forces.
b. compressive forces.  d. shearing stresses.

2. Traditionally, which diagnostic modality has been used to identify suspected myocardial injuries in traumatized dogs?
   a. intermittent ECG
   b. Holter monitoring
   c. blood gas evaluation
   d. biochemical markers

3. Which of the following conditions contributes to the arrhythmogenic effects of myocardial injuries?
   a. hypoxia
   b. intracranial injuries
   c. catecholamine release
   d. all of the above

4. Significant increases in ____________ pressure were found after iatrogenic trauma to the heart.
   a. left-end diastolic
   b. right-end diastolic
   c. left ventricular systolic
   d. a and b

5. What role do intracardiac pressure changes after thoracic trauma play in altering peripheral blood pressure?
   a. Although intracardiac pressure changes are significant, they do not result in significant changes in peripheral pressures.
   b. Significant intracardiac pressure changes result in changes in peripheral blood pressure.
   c. Myocardial injuries do not cause intracardiac pressure changes.
   d. Although intracardiac pressure changes were not significant, they did result in significant changes in peripheral pressures.

6. Trauma to the myocardium initiates arrhythmias by
   a. causing alterations in resting membrane potential.
   b. shortening the ratio of effective refractory period to action potential duration.
   c. changing currents of sodium and calcium across cell membranes.
   d. all of the above

7. Experimental injury to the canine heart resulted in which of the following injuries?
   a. injury to tricuspid and mitral valves
   b. localized edema, ecchymosis, and intramyocardial hematoma
   c. coronary artery rupture
   d. aortic rupture

8. The elastic nature of the thoracic cage in humans and animals subjects its contents to which of the following injuries?
   a. compressive, concussive, and penetrating
   b. compressive, hydrostatic, and shearing
   c. concussive, shearing, and penetrating
   d. penetrating, distressing, and compressive

9. Research in humans has shown that a direct thoracic blow may result in up to a ___% alteration in thoracic diameter.
   a. 10
   b. 20
   c. 50
   d. 75

10. Which of the following is not a common arrhythmia caused by myocardial injury?
    a. premature ventricular contractions
    b. atrial fibrillation
    c. ventricular tachycardia
    d. ST segment alterations