MYOCARDIAL CONTUSION

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KEY POINTS

- Myocardial injuries often are overlooked in the trauma patient.
- The most common physiologic consequence of myocardial injury in dogs and humans is arrhythmias.
- Arrhythmias associated with myocardial injury may be delayed in onset up to 48 hours.
- Holter monitoring or continuous electrocardiographic (ECG) monitoring should be considered in high-risk patients.
- Troponins, cardiac-specific proteins, are an effective biomarker of myocardial injury in dogs.
- Normal ECG findings and cardiac troponin I levels on admission in traumatized human patients are an efficient way to rule out myocardial injuries and arrhythmias associated with trauma.
- Management of myocardial injuries is aimed toward maintaining optimal cardiac output and suppressing life-threatening arrhythmias.
- The class I antiarrhythmic agents, including lidocaine and procainamide, are used commonly to manage ventricular ectopy associated with myocardial injury.

Traumatic myocarditis is a controversial subject. Much of the controversy in human studies revolves around a lack of consistent evidence that this injury has any effect on patient outcome and the expense associated with diagnostic testing, cardiac monitoring, and prolonged hospital stays.[1] Additional controversies associated with this injury revolve around its name, incidence, and how it is diagnosed. What appears to be agreed on consistently in the literature is the basic definition of this injury and that there is lack of an antemortem diagnostic gold standard. Direct visualization of the heart or histologic examination of damaged myocardium are considered the current diagnostic gold standard.[2]

The term *traumatic myocarditis* has been used often in veterinary literature to describe an assumed myocardial injury associated with arrhythmias in patients suffering from blunt thoracic trauma.[3] This term is used interchangeably with *myocardial injury* in this chapter.

INCIDENCE

Blunt thoracic trauma has been reported to result in myocardial injuries in 8% to 95% of human patients.[3-10] Reported variations in the frequency of myocardial injuries of dogs are similar to those described in humans. Several studies (three prospective, two retrospective) have examined the prevalence of traumatic myocarditis in the dog and report a range from 10% to 96%.[4,11-14] Variations in study design as well as disagreements regarding terminology, diagnostic modalities, and criteria used to identify myocardial injuries in humans and dogs contribute to the wide range in the reported frequency of this type of injury in both the human and veterinary literature.[2,3,7,13-21] The authors of these studies do agree, however, that myocardial injuries are easily overlooked.[18]

ETIOLOGY, MECHANISM OF INJURY, AND PATHOPHYSIOLOGY

Thoracic trauma is common in dogs injured by automobiles, animal attacks (bites, kicks), and falls from a height.* Because of the elastic nature of the thoracic cage, blunt trauma may subject the myocardium to compressive and concussive forces.[13,14,22-24] The most common mechanism of myocardial injury in the dog is that secondary to lateral chest compression.[22,24] In addition to potential concussive injury from forceful contact with the ribs, sternum, and vertebrae when rapid acceleration or deceleration occurs, 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.[6]

In vivo studies performed in dogs to mimic blunt chest trauma have correlated histopathologic areas of myocardial injury with areas of injury found during echocardiographic examination. Experimental trauma delivered to the left side of the chest resulted in abnormalities that were located primarily in the craniolateral wall of the left ventricle, and right-sided chest trauma produced septal and right ventricular wall damage.[6]

Gross pathologic findings in the traumatized heart have been characterized by localized edema, ecchymosis, and intramyocardial hematoma formation. Myocardial injuries were often transmural, with the epicardial surface being more severely affected.[6]

Arrhythmias and conduction defects are the most commonly reported consequences of myocardial injuries in humans and dogs.[7,11,22-27] One proposed proarrhythmic mechanism of myocyte trauma is the lowering of the ratio of effective refractory period to action potential duration and an increase in the resting membrane potential (less negative) in damaged myocardial cells. Additionally it is proposed that myocyte injury results in alterations of sodium and calcium currents across cell membranes, increasing the availability of intracellular calcium, resulting in increased sensitivity to depolarization.[3] These proposed intracellular derangements secondary to trauma can potentiate arrhythmogenesis.[3] Arrhythmias become apparent when the injured myocardium becomes the site of the most rapid impulse formation, overcoming the sinus node as the dominant (overdrive) pacemaker. The injured myocardium becomes the new overdrive pacemaker, propagating the arrhythmia by depolarizing the sinus node before it has a chance to fire and recapture the cardiac rhythm.[3]

Isolated rabbit hearts have been subjected to injury during high-resolution mapping of epicardial excitation to identify the origin of arrhythmias in injured myocardium. The results of this study identified reentry as the mechanism of arrhythmia caused by myocardial contusion. The authors found that the site of impact became electrically silent (temporarily), resulting in a fixed and functional conduction block that caused reentry initiation.[28]

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*References 2, 3, 11-13, 19-21.

Traumatized patients may also develop arrhythmias associated with metabolic acidosis, hypoxia, electrolyte imbalance, intracranial injuries, and catecholamine release.[23,25-27,29] These physiologic aberrations all promote alterations in membrane transport and permeability of cations (sodium, potassium, and calcium), which lead to a decrease in resting membrane potential, as described earlier, contributing to aberrant depolarization and arrhythmias.[3,23,25]

The most commonly reported arrhythmias secondary to canine myocardial injuries include premature ventricular contractions, ventricular tachycardia, and nonspecific ST segment elevation or depression.[6,22-27,29] Less commonly reported arrhythmias reported in dogs with chest trauma include atrial fibrillation, sinus arrest with ventricular or junctional escape complexes, and second-degree and third-degree atrioventricular block.[7,12,22,27]

DIAGNOSIS

Although uncommonly performed in the live patient, gross or histologic examination of the heart remains the diagnostic gold standard for myocardial contusions.[2,17,30] Because of the impracticality of visualizing the heart or performing myocardial biopsy, an understanding of the mechanism of injury, an awareness of associated injuries, and a high index of suspicion for myocardial injury are essential in making a diagnosis.[10] Emergency clinicians should consider myocardial injury in all traumatized dogs that have the following injuries: (1) fractures of extremities, spine, or pelvis, (2) external evidence of thoracic trauma, (3) radiographic evidence of chest trauma such as pulmonary contusions, pneumothorax, hemothorax, diaphragmatic rupture, and rib or scapular fractures, and (4) neurologic injury.*

Dogs with any of these injuries should have a lead II electrocardiograph (ECG) performed and, depending on the patient's condition and the clinician's index of suspicion, the ECG should be repeated intermittently (i.e., every 2 to 24 hours). ECG abnormalities commonly are delayed in onset for up to 48 hours after blunt chest trauma, so in cases in which there is a high index of suspicion for myocardial injury ECG monitoring should be considered for that time frame.[22,23,25] Holter monitoring is the most sensitive and least invasive indicator of arrhythmias in dogs with suspected myocardial injuries. However, the lack of immediate Holter interpretation (rapid turnaround time) may limit the practical application of this modality for veterinarians.[12] Other forms of continuous ECG monitoring, such as single patient monitors and telemetry, would likely provide a similar advantage over intermittent ECGs without the delays in interpretation encountered with Holter monitoring.[18]

An echocardiogram should be considered in severely traumatized dogs with a poor response to resuscitative efforts and evidence of thoracic injuries even if no ECG abnormalities are present. Transthoracic echocardiography in the dog can be used to identify and localize both structural and functional abnormalities of injured myocardium caused by blunt chest trauma. The echocardiographic features of myocardial injuries in the dog include (1) increased end-diastolic wall thickness; (2) impaired contractility, indicated by wall motion abnormalities and decreased fractional shortening; (3) increased echogenicity; and (4) localized areas of echolucency consistent with intramural hematomas.[6]

Serum myocardial isoenzyme analysis (cardiac troponins T and I [cTnT and cTnI]) has been used to diagnose myocardial injury in dogs and humans. The skeletal isoforms of the troponin proteins expressed are different from those in cardiac muscle.[19,31] The troponin structure is highly conserved across many differing species,

allowing for veterinary application of tests currently in use at human care facilities.[32]

Troponin testing is based on immunologic detection of the cardiac-specific isoforms of troponin T and troponin I.[31] In both human and dogs, detectable levels appear in the circulation within 4 to 6 hours of cardiac myocyte injury and serum elevations may be present for up to 7 days.[7,19,32] In a comparison of multiple myocardial enzyme and protein markers and ECG to detect myocardial injury in traumatized dogs, cTnI was the most sensitive indicator of this type of injury.[2] One of the most important findings of the many human studies investigating the clinical use of cardiac troponins appears to be the negative predictive values for cardiac complications in trauma patients. In human trauma patients a normal cTnI level in combination with a normal ECG tracing on arrival has a negative predictive value of 100% for myocardial injuries, allowing these patients to avoid intensive cardiac monitoring and even be discharged safely in the absence of other significant injuries.[33] Because of the controversies and difficulty diagnosing myocardial injuries in dogs, veterinarians should consider using these two tests to rule out this disease in a quick and practical manner. Although there are no studies confirming this hypothesis in dogs, clinicians could consider performing a baseline ECG and cTnI measurement within 4 hours of injury. Extrapolating from human findings, dogs with a combination of normal ECG findings and cTnI levels (normal < 0.03 to 0.07 ng/ml[34]) would be less likely to develop arrhythmias and therefore would not require intensive cardiac monitoring. A positive finding on either test would suggest the possibility of myocardial injury and would indicate continuous ECG monitoring in those dogs.

TREATMENT

Treatment of myocardial injuries typically is aimed at suppressing potentially life-threatening arrhythmias and maintaining adequate tissue perfusion.[30] Antiarrhythmic therapy is not recommended if arterial pulse quality is good and synchronous on auscultation, mean arterial pressure is higher than 75 mm Hg, mucous membranes are pink, capillary refill time is 2 seconds or less, and the patient has no clinical signs of weakness or cardiopulmonary distress.[30] Antiarrhythmic therapy should be considered when properly stabilized patients (i.e., received adequate fluids, electrolytes, oxygen, pain control) develop arrhythmias such as multiform premature ventricular complexes, ventricular tachycardia, and the R-on-T phenomenon.[23,26,27,30] Treatment is imperative when arrhythmias are accompanied by clinical evidence of decreased cardiac output such as hypotension, weakness, pale mucous membranes, delayed capillary refill time, collapse, or syncope.[23,26,30] Additionally, treatment is indicated when an arrhythmia has a sustained (>15 to 30 seconds) ventricular rate that exceeds 140 to 180 beats/min in the dog.[12,23,26]

Lidocaine (2 mg/kg IV bolus) is the agent of choice for traumatized dogs suffering from ventricular ectopy fulfilling the criteria described in the previous paragraph.[30] Intravenous boluses of lidocaine may be repeated every 10 to 20 minutes until a cumulative dose of 8 mg/kg is given. A constant rate infusion (CRI) of 40 to 80 mcg/kg/min may be initiated to maintain a cardiac rate and rhythm that provides appropriate tissue perfusion.[23,30] Additional boluses of lidocaine are often required to suppress arrhythmias while steady-state blood levels are achieved by the CRI. The upper end of the recommended dosages of lidocaine may cause vomiting or seizures, so administration should be slowed or temporarily discontinued if these signs develop (see Chapter 48 for more information).[23,30,35]

If lidocaine does not resolve ventricular ectopy, procainamide may be administered intravenously or intramuscularly (6 to 15 mg/kg q4-6h).[23,30] If repeated boluses of procainamide are required to suppress arrhythmias, a CRI (10 to 40 mcg/kg/min) may be

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*References 2, 12, 22, 25, 29, 30.

started. Oral procainamide (sustained release formulation 20 mg/kg q8h) may be initiated if continued management is required and oral medications can be tolerated. Potential side effects of procainamide administration include hypotension and atrioventricular conduction block.[23,35] Additional oral arrhythmia management options include tocainide (10 to 20 mg/kg PO q8-12h) and mexiletine (4 to 8 mg/kg PO q8h).[23] The reported side effects of tocainide include nausea, vomiting, and anorexia; although less commonly observed, complications associated with mexiletine include excitement or depression.[35]

β-Blockers (propranolol, metoprolol, atenolol, sotalol) should be considered cautiously when traumatized dogs with ventricular ectopy are unresponsive to class I antiarrhythmic agents, have been treated appropriately for shock and pain, and are not receiving positive inotropic medications.[30,35] An ultrashort-acting intravenous β-blocker, such as esmolol, may be used to test the efficacy of β-blockers in managing ventricular arrhythmias that have not responded to other medications.[26] The potential for serious side effects such as atrioventricular block, hypotension, bronchoconstriction, and decreased cardiac contractility must be considered when using β-blockers.[23,35]

Arrhythmias secondary to myocardial trauma that do not fulfill the stated guidelines for management are likely to be self-limiting and resolve within 3 to 10 days.[30] The end point of therapy is not necessarily complete resolution of the arrhythmia; appropriate therapeutic response includes reduction of the heart rate (<140 beats/min) and the return of adequate tissue perfusion.[30] In most cases antiarrhythmic therapy can be discontinued within 48 to 72 hours; however, it is recommended that intermittent ECG monitoring continue up to 1 week after discharge.[30] Medications being used to suppress arrhythmias should be discontinued a minimum of 24 hours before reexamination.[30] The most sensitive way to detect complete resolution of arrhythmias after discontinuing antiarrhythmic medications is continuous ECG (Holter) monitoring.[12] If some form of continuous monitoring is not available, intermittent lead II ECG monitoring can be performed to ensure that the arrhythmia has resolved and it is safe to discontinue therapy. If the arrhythmia persists, long-term oral therapy may be initiated.[30]

If a dog with a suspected myocardial injury must undergo anesthesia, agents that are least likely to induce arrhythmias, such as acepromazine, butorphanol, isoflurane, and glycopyrrolate, should be used.[3,23] Halothane, atropine sulfate, and the thiobarbiturates should be avoided because they are reported to exacerbate arrhythmias and to sensitize the heart to catecholamine-induced arrhythmias.[23,25]

SUMMARY

Although myocardial injuries may cause significant alterations in cardiac function in the traumatized dog, they are often overlooked in the face of severe trauma. Ventricular arrhythmias are the most common abnormalities caused by blunt myocardial injury. Although ECG monitoring traditionally has been used to diagnose myocardial injuries, the onset of arrhythmias associated with these injuries is often delayed, making recognition difficult. Arrhythmias may be secondary to alterations in transport of cations, such as calcium, potassium, and 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 or reentry mechanisms.

The medical community has yet to provide a prospective study that investigates the need for therapeutic intervention of traumatic myocardial injuries in humans or dogs. Although newer noninvasive tests for myocardial injuries such as troponin levels may assist in diagnosing these injuries, they have not yet proven to be the single noninvasive diagnostic modality to detect myocardial injury.[18] In the real world of veterinary practice, continuous ECG monitoring has been shown to be a sensitive, noninvasive indicator of arrhythmias in traumatized dogs and should be considered in dogs with suspected myocardial injuries.[12]

A human study may have inadvertently stumbled upon an approach that makes more sense than those previously discussed: ruling out this disease rather than ruling it in. This study showed that in traumatized patients a combination of normal ECG and cTnI findings on admission had a 100% negative predictive value for cardiac complications, meaning that none of the patients with these test results developed arrhythmias requiring intervention.[33] These findings may be helpful in determining the need for continuous ECG monitoring in canine patients with suspected myocardial trauma and should be considered for further investigation in dogs.

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