Trauma Associated With Cardiac Dysrhythmias: Results From a Large Matched Case–Control Study

Rovshan M. Ismailov, MD, MPH, PhD, Roberta B. Ness, MD, MPH, Carol K. Redmond, ScD, Evelyn O. Talbott, PhD, and Hank B. Weiss, MPH, PhD

Background: Various cardiac dysrhythmias such as supraventricular and ventricular premature beats, supraventricular and ventricular paroxysmal tachycardia, atrial and ventricular fibrillation and atrial flutter have been reported in case series, as complications of blunt cardiac and thoracic trauma. The objective of this research was to determine whether thoracic or blunt cardiac injury is associated with cardiac dysrhythmia in a large multistate hospitalized population.

Methods: Cases and matched (by age) controls were identified based on hospital discharge information that was collected from 986 acute general hospitals across 33 states in 2001. Both the exposure (thoracic trauma and blunt cardiac injury) and the outcome (cardiac dysrhythmias) were identified based on ICD-9-CM discharge diagnoses. Unadjusted and conditional adjusted (for gender, race, length of stay, and primary source of payment) multivariate logistic regression analyses were performed.

Results: After adjusting for potential confounders, patients 50 years and younger diagnosed with blunt cardiac injury had a fourfold (95% confidence interval, 1.40–11.60) increase in the risk of cardiac dysrhythmia. Independent of potential confounding factors, discharge for blunt cardiac injury among patients 51 to 70 years old was associated with a twofold (95% confidence interval, 1.36–3.82) increased risk for cardiac dysrhythmia.

Conclusion: Blunt cardiac injury was found to be a significant risk factor for cardiac dysrhythmia. Longitudinal studies are needed to better establish the association between trauma and cardiac dysrhythmias.

Key Words: Trauma, Cardiac dysrhythmias.


Cardiac dysrhythmias have been frequently reported in case reports as a complication of trauma.1–17 Thoracic trauma and blunt cardiac injury have been shown to produce various types of dysrhythmias such as ventricular and supraventricular extrasystoles,18–21 atrial fibrillation and flutter,18–21 supraventricular and ventricular paroxysmal tachycardia,6,8,12–15,20,24 and ventricular fibrillation.16,23,25 Traumatic cardiac dysrhythmias have been observed after being hit by a brick,4 by a steering column,24 and by a soccer ball.12 Fabian et al.9 described 92 patients who experienced various cardiac dysrhythmias after anterior chest impact (i.e. sternal or rib fractures) and Leor et al.12 observed multiple ventricular premature contractions in patient with blunt trauma to the left precordium. In some studies, patients who developed cardiac dysrhythmia had no history of cardiovascular diseases.1,15 Traumatic cardiac dysrhythmias usually developed within the first several hours26,27 or within 24 to 48 hours after injury;19,28–30 however, some patients with trauma experienced life-threatening dysrhythmias several days after an episode of injury.7

To our knowledge, no population-based studies have examined the relationship between thoracic trauma and cardiac dysrhythmias. Our previous study showed that trauma may be independently associated with such serious cardiac events as acute myocardial infarction31 and cardiac valve insufficiency.32 Although potentially treatable, cardiac complications of thoracic or cardiac trauma such as heart failure or cardiac dysrhythmias are difficult to predict.33,34 The objective of this research, therefore, was to determine whether thoracic and blunt cardiac injuries are significantly associated with certain cardiac dysrhythmias. A matched case–control study of the association between thoracic and blunt cardiac injuries and cardiac dysrhythmias was conducted based on a database of all hospital discharges from 33 states in the United States.

MATERIALS AND METHODS

The Agency for Healthcare Research and Quality developed the 2001 Healthcare Cost and Utilization Project Nationwide Inpatient Sample (NIS; http://www.hcup-us.ahrq.gov/db/nation/nis/Overview_of_NIS_2001_25Jul03.pdf). The 2001 NIS file has 7.45 million uniform hospital discharge abstracts for all inpatient stays. Such discharge information was collected from 986 acute care general hospitals across 33 states (Arizona, California, Colorado, Connecticut, Florida, Georgia, Hawaii, Illinois, Iowa, Kansas, Kentucky, Maryland, Massachusetts, Maine, Minnesota, Missouri, Montana, Nebraska, North Carolina, New Jersey, New York, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Tennessee, Texas, Utah, Vermont, Virginia, Washington, West Virginia, Wisconsin, Wyoming). The NIS file includes data on hospital discharge characteristics, patient demographics, and hospital charges. The NIS database was used to identify cases and controls for this study.

Cardiac dysrhythmias were identified based on ICD-9-CM codes. These included premature supraventricular beats (ICD-9-CM 427.61), paroxysmal supraventricular (427.0), atrial flutter (427.32), atrial fibrillation (427.31), premature ventricular beats (427.69), paroxysmal ventricular tachycardia (427.1), and ventricular fibrillation (427.41).

There were 672,043 cases with the dysrhythmia of interest. All dysrhythmias of interest were combined into a single outcome. Controls were matched for age (±1.5 years) in the ratio 1:1 by random selection of individuals within the database. All controls were free of reported dysrhythmia based on ICD-9-CM codes.

Thoracic trauma was identified based on ICD-9-CM diagnostic codes: 807.0–807.4, 839.61–839.71, 848.3, 848.4, 860–862, 875, 879.0, 879.1, 901, 927.0, 922.1, and 942.x1–942.x2. Body part groupings were based on an early version of the Barell Matrix developed by Barell et al.37 Blunt cardiac injury was identified based on ICD-9-CM diagnostic code (861.01).

Potential confounding factors included patient-level demographic characteristics such as age, race (white vs. non-white), and gender (male vs. female). Length of stay was categorized as a categorical variable (≥3 days vs. >3 days). Primary source of payment included Medicare, Medicaid, private including HMO, self-pay, no charge, and other.

Paired case–control analyses were conducted with results reported for analyses and subset analyses where the number of observations was significant (Tables 2 and 3). In the multivariate conditional logistic regression analyses, patients 71 years and older whereas blunt cardiac injury was significantly associated with increased risk for cardiac dysrhythmia among all age groups; however, this result was not statistically significant (Tables 2 and 3). In the multivariate conditional logistic regression analyses, patients ≥50 years old diagnosed with blunt cardiac injury had a fourfold (95% CI, 1.40–11.60) increase in the risk of cardiac dysrhythmia (Table 2). Independent of potential confounding factors, discharge for blunt cardiac injury among patients 51 to 70 years was associated with a twofold (95% CI, 1.36–3.82) increased risk for cardiac dysrhythmia (Table 2). After adjusting for potential confounders, discharge for thoracic trauma was found to have moderately decreased risk for cardiac dysrhythmia among people 71 years and older (Table 3).

**Table 1 Social and Demographic Characteristics of Cases and Controls**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Cases (672,043)</th>
<th>Controls (672,043)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female (%)</td>
<td>342,565 (50.9)</td>
<td>398,295 (59.3)</td>
</tr>
<tr>
<td>White (%)</td>
<td>436,232 (64.9)</td>
<td>401,572 (59.8)</td>
</tr>
<tr>
<td>Length of stay (median)</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Medicare</td>
<td>522,193 (77.7)</td>
<td>515,303 (76.7)</td>
</tr>
<tr>
<td>Medicaid</td>
<td>21,100 (3.1)</td>
<td>25,881 (3.9)</td>
</tr>
<tr>
<td>HMO</td>
<td>109,719 (16.3)</td>
<td>108,155 (16.1)</td>
</tr>
<tr>
<td>Self-pay</td>
<td>8,842 (1.3)</td>
<td>10,299 (1.5)</td>
</tr>
<tr>
<td>No-charge</td>
<td>641 (0.1)</td>
<td>686 (0.1)</td>
</tr>
<tr>
<td>Thoracic trauma</td>
<td>988 (0.1)</td>
<td>1,193 (0.2)</td>
</tr>
<tr>
<td>Blunt cardiac injury</td>
<td>126 (0.02)</td>
<td>90 (0.01)</td>
</tr>
</tbody>
</table>

Copyright © Lippincott Williams & Wilkins. Unauthorized reproduction of this article is prohibited.
suggesting that the strength of the impact is less important, mias may result from relatively "mild" sports trauma, perhaps the size of the contact area. Evidence that cardiac dysrhythmia was found to be directly proportional to both the impact force and speed of the impact and inversely proportional to cardiac electric cycle. In addition, the risk of cardiac dysrhythmia depend on when the impact occurred during the conduction by damaged myocardial cells. Local hypoxia and ischemia caused by increased intravascular rouleaux formation owing to trauma may also contribute to the mechanism of traumatic cardiac dysrhythmias. The mechanism of traumatic cardiac dysrhythmias was studied in animals. Schlomka conducted a series of experiments where he traumatized the heart by direct blows. Both ventricular tachycardia and fibrillation were observed. Link et al. conducted a series of low-energy impacts to the chest wall in a swine model. It has been demonstrated that the risk and type of dysrhythmia depend on when the impact occurred during the cardiac electric cycle. In addition, the risk of cardiac dysrhythmia was found to be directly proportional to both the force and speed of the impact and inversely proportional to the size of the contact area. Evidence that cardiac dysrhythmias may result from relatively "mild" sports trauma, perhaps suggesting that the strength of the impact is less important, has been also introduced by various researchers who noticed that various types of dysrhythmias may appear from usually innocent-appearing chest blows in various sport activities. On the other hand, by using the swine model it was shown that even low-energy impact can have immediate and significant effect if applied during a short and vulnerable time interval (i.e. upstroke of the T wave), resulting in ventricular fibrillation. Atrial fibrillation, one of the most common cardiac dysrhythmias encountered in clinical practice was found to be the most common form of dysrhythmia that presents after chest injury. In the study conducted by Seguin et al., independent of confounding factors, blunt thoracic trauma was associated with a 17-fold increased risk for atrial fibrillation. The relatively short observation of patients for the presence of some confounding factors such as the presence or absence of shock as well as the relatively low incidence of atrial fibrillation were the main limitations of this study. Another limitation of this study was the lack of assessment for blunt cardiac injury among patients with blunt thoracic injury, which may explain the disagreement in results between this and our study.

| Table 2 Conditional Multivariate Logistic Regression Analysis on Blunt Cardiac Injury and Cardiac Dysrhythmias |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
|                                | ≤50 Years Old   | 51–70 Years Old | ≥71 Years Old   |
|                                | OR   | p   | 95% CI | OR   | p   | 95% CI | OR   | p   | 95% CI |
| Blunt cardiac injury, unadjusted | 1.67 | 0.32 | 0.61–4.59 | 1.39 | 0.19 | 0.84–2.31 | 1.38 | 0.06 | 0.98–1.93 |
| Blunt cardiac injury, adjusted* | 4.03 | 0.01 | 1.40–11.60 | 2.28 | <0.01 | 1.36–3.82 | 0.78 | 0.16 | 0.55–1.10 |

* Adjusted for gender, race, length of stay, and source of payment.
CI, confidence interval; OR, odds ratio.

| Table 3 Results of the Conditional Multivariate Logistic Regression Analysis on Thoracic Injury and Cardiac Dysrhythmias |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
|                                | ≤50 Years Old   | 51–70 Years Old | ≥71 Years Old   |
|                                | OR   | p   | 95% CI | OR   | p   | 95% CI | OR   | p   | 95% CI |
| Thoracic injury, unadjusted     | 0.94 | 0.76 | 0.64–1.41 | 0.90 | 0.24 | 0.76–1.07 | 0.80 | <0.01 | 0.72–0.88 |
| Thoracic injury, adjusted*      | 1.35 | 0.11 | 0.93–1.97 | 1.11 | 0.26 | 0.93–1.34 | 0.72 | <0.01 | 0.65–0.80 |

* Adjusted for gender, race, length of stay, and source of payment.
CI, confidence interval; OR, odds ratio.

DISCUSSION

This article represents the first attempt to look at the association between thoracic and cardiac trauma and cardiac dysrhythmias at a large population-based level. Population-based studies are important in that they reduce the potential for selection bias and confounding, both of which may limit the interpretation of case reports. In addition, population-based studies that include control groups provide quantitative estimates of association as well as better estimates of public health impact.

We found that patients 50 years and younger diagnosed with blunt cardiac injury had a fourfold increase, whereas patients 51 to 70 years old diagnosed with the same type of injury had a twofold increase in the risk of cardiac dysrhythmia. Several mechanisms have been hypothesized to explain cardiac dysrhythmias resulting from trauma, including abnormal perfusion patterns, vagal sympathetic reflex, and abberant conduction by damaged myocardial cells. Local hypoxia and ischemia caused by increased intravascular rouleaux formation owing to trauma may also contribute to the mechanism of traumatic cardiac dysrhythmias. The mechanism of traumatic cardiac dysrhythmias was studied in animals. Schlomka conducted a series of experiments where he traumatized the heart by direct blows. Both ventricular tachycardia and fibrillation were observed. Link et al. conducted a series of low-energy impacts to the chest wall in a swine model. It has been demonstrated that the risk and type of dysrhythmia depend on when the impact occurred during the cardiac electric cycle. In addition, the risk of cardiac dysrhythmia was found to be directly proportional to both the force and speed of the impact and inversely proportional to the size of the contact area. Evidence that cardiac dysrhythmias may result from relatively "mild" sports trauma, perhaps suggesting that the strength of the impact is less important, has been also introduced by various researchers who noticed that various types of dysrhythmias may appear from usually innocent-appearing chest blows in various sport activities. On the other hand, by using the swine model it was shown that even low-energy impact can have immediate and significant effect if applied during a short and vulnerable time interval (i.e. upstroke of the T wave), resulting in ventricular fibrillation. Atrial fibrillation, one of the most common cardiac dysrhythmias encountered in clinical practice was found to be the most common form of dysrhythmia that presents after chest injury. In the study conducted by Seguin et al., independent of confounding factors, blunt thoracic trauma was associated with a 17-fold increased risk for atrial fibrillation. The relatively short observation of patients for the presence of some confounding factors such as the presence or absence of shock as well as the relatively low incidence of atrial fibrillation were the main limitations of this study. Another limitation of this study was the lack of assessment for blunt cardiac injury among patients with blunt thoracic injury, which may explain the disagreement in results between this and our study.

Although chronic cardiovascular conditions, such as ischemic heart disease or rheumatic diseases, are major cause of atrial fibrillation, in about 10% of people with this type of dysrhythmia, the “true” cause is unknown. Blunt cardiac injury was found to be one of the causes of atrial fibrillation but this type of injury is difficult to diagnose. In a previous study, we have found that blunt cardiac injury is much less frequent when identified through ICD-9-CM and when compared with thoracic injury (57,270 vs. 2,709 respectively). In addition, relatively mild mechanical impact to the chest can result in serious cardiac dysrhythmia even without significant blunt cardiac injury.
other hand, cardiac injury may be produced by external traumatic agent without symptoms of significant chest trauma. Thus, this probably can explain the lack of association between chest trauma and cardiac injury in our study.

Both supraventricular and ventricular paroxysmal tachycardia have been reported after thoracic and cardiac trauma. Most traumatic cardiac dysrhythmias and electrocardiogram (ECG) changes are transitory conditions that are usually represented as ST-T changes of extrasystoles. Some traumatic cardiac dysrhythmias such as ventricular fibrillation, however, may lead to immediate death. This phenomenon is poorly understood and in some cases is described as commotion cordis, a life-threatening event occurring mostly in young sportsmen. Although commotion cordis is a relatively rare event, its prevalence is likely underestimated.

We found that thoracic trauma was not a risk factor for cardiac dysrhythmias among patients 70 years and younger. Among elderly patients (≥71 years old) thoracic trauma was related to moderately decreased risk for cardiac dysrhythmias after adjusting for other variables. Readers should bear in mind that cardiac injury (i.e., areas of patchy necrosis, local hypoxia, and ischemia) may be produced by external traumatic agent without significant damage to the chest wall. On the other hand, the existing Barrel matrix provides only limited information about the anatomic site of thoracic trauma (for instance, whether the impact was toward the left precordium). Future population-based studies are warranted to better understand the relationship between thoracic trauma and cardiac dysrhythmias.

There are several limitations related to this study. Because of the nature of the data (i.e., administrative), temporal trends between trauma and cardiac dysrhythmias could not be established. There might be a higher probability of sustaining cardiac dysrhythmia in the presence of pre-existing cardiac disease; however, this database cannot provide any insight into this issue, since administrative data may not provide accurate insight into timing of events. Certain other problems such as coding accuracy and variation as well as a limited insight into temporal relationship between events have been related to administrative data. In addition, such data may not provide full clinical information on certain important confounders such as smoking status or seat belt use. Medical chart review, although more expensive, may provide more detailed information on both the exposure and the disease whereas longitudinal studies would provide in-depth insight into timing of events and mortality. Finally, although we have gained power by combining all dysrhythmias into one group, we have lost the precision of the individual types of dysrhythmia.

It is important to establish the nature and degree of association between certain types of trauma, such as cardiac and thoracic, and dysrhythmias. Ventricular paroxysmal tachycardia can degenerate spontaneously into ventricular fibrillation or may result in congestive heart failure. However, it may be even more important to establish a link between trauma and supraventricular paroxysmal tachycardia, since such patients may be completely asymptomatic. Nevertheless, depending on coexisting cardiac diseases, such dysrhythmia can cause pulmonary edema or myocardial ischemia. Patients with blunt cardiac injury, therefore, might benefit from certain screening procedures for cardiac dysrhythmias such as ECG, although normal ECG at admission and during 24 hours in the intensive care unit does not exclude fatal cardiac dysrhythmias after discharge. Results of this study, however, do not lend themselves to aggressive screening for cardiac dysrhythmias; rather, they suggest more research in this particular direction. Future research should also focus on possible association between other types of trauma such as back or abdominal trauma and cardiac dysrhythmias.

**CONCLUSION**

Blunt cardiac injury was found to be a significant risk factor for cardiac dysrhythmia. Longitudinal studies are needed to better establish the association between trauma and cardiac dysrhythmias.

**REFERENCES**

12. Leor J, Gilkson M, Vered Z, Kaplinsky E, Motro M. Ventricular tachycardia after soccer ball blow to the chest: first manifestation of
arhythmogenic right ventricular dysplasia in two brothers.  


