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Blunt Traumatic Cardiac Rupture: Single-Institution Experiences over 14 Years

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Background: Blunt traumatic cardiac rupture is rare. However, such cardiac ruptures carry a high mortality rate. This study reviews our experience treating blunt traumatic cardiac rupture. **Methods:** This retrospective study included 21 patients who experienced blunt traumatic cardiac rupture from 1999 to 2015. Every patient underwent surgery. Several variables were compared between survivors and fatalities. **Results:** Sixteen of the 21 patients survived, and 5 (24%) died. No instances of intraoperative mortality occurred. The most common cause of injury was a traffic accident (81%). The right atrium was the most common location of injury (43%). Ten of the 21 patients were suspected to have cardiac tamponade. Significant differences were found in preoperative creatine kinase–myocardial band (CK-MB) levels (p=0.042) and platelet counts (p=0.004) between the survivors and fatalities. The patients who died had higher preoperative Glasgow Coma Scale scores (p=0.007), worse Trauma and Injury Severity Scores (p=0.007), and higher Injury Severity Scores (p=0.004) than those who survived. **Conclusion:** We found that elevated CK-MB levels, a low platelet count, and multi-organ traumatic injury were prognostic factors predicting poor outcomes of blunt cardiac rupture. If a patient with blunt traumatic cardiac rupture has these factors, clinicians should be especially attentive and respond promptly in order to save the patient's life.

Key words: 1. Cardiac tamponade
2. Trauma
3. Rupture

Introduction

Blunt traumatic cardiac rupture is rare but highly fatal [1-3]. The incidence of blunt cardiac rupture among hospital admissions for trauma is only approximately 0.16%–2% [4-6]. However, the actual incidence may be higher because many victims may die before arrival at the hospital. With advances in traumatology, several studies have investigated the prognostic factors associated with traumatic cardiac rupture [1,7]. In this study, we attempted to find factors affecting the prognosis of patients with blunt traumatic cardiac rupture.

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Table 1. Patient characteristics

Characteristic	Value
Age (yr)	48 (17–95)
Gender	
Male	13 (62)
Female	8 (38)
Cause of trauma	
Fall	2 (9.7)
Motor vehicle accident	
Inside car	11 (52.0)
Pedestrian	4 (19.0)
Motorcycle	2 (9.7)
Industrial accident	2 (9.7)
Injury location	
Left atrium	2 (9.7)
RA	9 (43.0)
RA/superior vena cava	2 (9.7)
RA/IVC	1 (4.8)
RV/IVC	1 (4.8)
Left ventricle	3 (14.0)
RV	3 (14.0)
Diagnostic method	
Chest CT	10 (48.0)
Echocardiography	7 (33.0)
CT/echocardiography	4 (19.0)
Cardiopulmonary bypass	4 (19.0)
Cardiopulmonary resuscitation	2 (9.7)
Ventilator care time (hr)	111±318.8
Hospital stay duration (day)	36±35.9
Intensive care unit stay duration (day)	17±29.3

Values are presented as median (range), number (%), or mean± standard deviation.
RA, right atrium; IVC, inferior vena cava; RV, right ventricle; CT, computed tomography.

Methods

1) Patients

This retrospective study was reviewed and approved by the institutional ethics committee of our institution (Ethical Committee of Sungkyunkwan University of Samsung Changwon Hospital, approval number 2015-SCMC-037-00). Between 1999 and 2015, a total of 107,657 patients were admitted to our hospital due to trauma. We selected 47 patients who were diagnosed with traumatic cardiac rupture. We excluded patients with penetrating or iatrogenic cardiac rupture. Ultimately, we analyzed 21 patients who were diagnosed with blunt traumatic cardiac rupture and required emergency surgery. In our analysis of these 21 patients, we identified several prognostic factors.

2) Surgery

All operations were performed via median sternotomy. In each case, cardiopulmonary bypass (CPB) or extracorporeal membrane oxygenation was prepared as a precaution. If the patient was hemodynamically unstable, 2 cardiovascular surgeons operated together to perform the median sternotomy and femoral cannulation simultaneously. After the sternotomy, surgeons opened the pericardium for prompt decompression of the heart, rapidly removed the hematoma, and attempted to find the injury site. The injury sites were primarily repaired using double-arm monofilament stitches with pledgeted, interrupted sutures. After the injury sites were primarily repaired, we weaned the patients from CPB as soon as possible.

3) Statistical analysis

All data are expressed as means±standard deviations. Continuous variables were compared using the Mann-Whitney U-test. Categorical variables were compared using the chi-square test or the Fisher exact test. All p-values <0.05 were considered to indicate statistical significance. Data were analyzed using IBM SPSS ver. 21.0 (IBM Co., Armonk, NY, USA).

Results

The patients' clinical characteristics are presented in Table 1. Most blunt traumatic cardiac injuries resulted from traffic accidents (81%), and most patients had combined trauma. The combined trauma included liver laceration, spleen injury, pelvic bone fracture, spinal fracture, and fractures of the extremities (Table 2). The mean Injury Severity Score (ISS) was 28±17.4 (range, 16 to 97).

Among the 21 patients, 10 were suspected to have cardiac tamponade, 3 of whom died after surgery. The mean central venous pressure (CVP) and systolic blood pressure of the 3 patients who died were 28 cm H_2O and 67 mm Hg, respectively. In contrast, the mean CVP and systolic blood pressure of the 7 surviving patients were 26 cm H_2O and 81 mm Hg, respectively.

Two patients received cardiopulmonary resuscitation (CPR) prior to surgery. One of them died during postoperative care due to pneumonia and the

Table 2. Age, sex, injury mechanism, injury sites, combined injury, injury score, injury severity, hospital course, and outcome of patients

No.	Age (yr)	Sex	Mechanism	Injury location	Combined injury	Injury Severity Score	Tamponade	Shock index[a)]	Cardiopulmonary resuscitation	Cardiopulmonary bypass	Hospital day (day)	Intensive care unit stay day (day)	Cause of death	Outcome
1	39	F	MVA	RA-SVC	Pneumothorax, Rt. patella fracture	21	No	0.65	No	No	15	8	-	Alive
2	25	M	Pedestrian	RA	Diffuse axonal injury, Lt. renal contusion, liver contusion, Lt. hip fracture	24	No	2.5	No	No	92	42	-	Alive
3	35	F	MVA	RA-SVC	Rt. radius fracture, intrahepatic hematoma	24	No	0.86	No	No	18	4	-	Alive
4	19	M	MVA	RA	No	16	Yes	2.26	No	No	13	4	-	Alive
5	59	M	Industrial	RV	No	16	Yes	0.9	No	No	37	4	-	Alive
6	67	M	Motorcycle	RA	Hemopneumothorax (both), Rt. patella fracture, Rt. acetabular fracture	21	No	0.9	No	No	99	55	-	Alive
7	17	M	Motorcycle	RA	Liver laceration, Rt. renal injury, Rt. pubic bone fracture, Lt. facial bone fracture, cerebral contusion,	26	No	2.7	Yes	No	28	7	-	Alive
8	30	F	MVA	RA	Hypoxic brain damage, liver contusion, Rt. ankle fracture	21	Yes	1.14	No	No	21	8	-	Alive
9	50	M	Industrial	RV-IVC	Rt. wrist fracture	20	Yes	1.13	No	No	26	5	-	Alive
10	39	F	MVA	RA	Liver laceration, cerebral concussion	26	No	1.51	No	No	15	9	-	Alive
11	52	F	Pedestrian	RA	Spleen laceration	24	Yes	1.56	No	No	28	3	-	Alive
12	47	M	MVA	RV	No	16	Yes	1.61	No	No	10	2	-	Alive
13	75	F	MVA	RA-IVC	Lt. humerus mid shaft fracture, facial laceration	21	No	1.16	No	Yes	82	8	-	Alive
14	48	M	MVA	LV	Cerebral hemorrhage, panperitonitis, liver contusion, Rt. patellar fracture	34	No	0.94	No	No	70	27	-	Alive
15	73	M	MVA	LA	Pelvic bone fracture	20	No	0.78	No	No	16	4	-	Alive
16	48	M	Fall down	LA	Distal jejunum mesenteric tearing	34	Yes	1.33	No	Yes	23	5	-	Alive

(Continued to the next page)

Table 2. Continued

No.	Age (yr)	Sex	Mechanism	Injury location	Combined injury	Injury Severity Score	Tamponade	Shock index[a)]	Cardiopulmonary resuscitation	Cardiopulmonary bypass	Hospital day (day)	Intensive care unit stay day (day)	Cause of death	Outcome
17	27	M	Pedestrian	RA	L-spine dislocation, paraplegia, hemo-pneumothorax, hemoperitoneum, liver contusion, GI bleeding	42	No	1.44	No	No	31	30	GI bleeding due to gastric ulcer, ARF	Dead
18	36	M	MVA	RA	Hypoxic brain damage, Rt. pneumothorax	97	Yes	1.73	Yes	No	128	128	Pneumonia, brain death	Dead
19	48	M	MVA	LV	Rt. femur fracture	20	Yes	2.46	No	No	2	1	Low cardiac output, brain death	Dead
20	31	F	Fall down	LV	Pelvic bone fracture, spleen laceration, both internal iliac artery rupture	41	No	1.65	No	Yes	2	2	Hypovolemic shock, DIC, ARF	Dead
21	95	F	Pedestrian	RV	Multiple rib fracture, Lt. fibular fracture, Lt. pubic bone fracture	24	Yes	0.97	No	Yes	1	1	DIC, hypovo-lemic shock	Dead

F, female; MVA, motor vehicle accident; RA, right atrium; SVC, superior vena cava; Rt., right; M, male; Lt., left; RV, right ventricle; IVC, inferior vena cava; LV, left ventricle; LA, left atrium; GI, gastrointestinal; ARF, acute renal failure; DIC, disseminated intravascular coagulation.
[a)]Heart rate/systolic blood pressure.

Table 3. Mean preoperative measures

Variable	Value
Creatine kinase-myocardial band isoenzyme (ng/mL)	13.4±9.2
Troponin I (ng/dL)	1.69±2.2
Hematocrit (g/dL)	37.1±5.1
Platelet count (10^3/mL)	189.2±7.2
Creatinine (mg/dL)	1.1±0.3
Systolic blood pressure (mm Hg)	80.9±21.9
Central venous pressure (cm H_2O)	19.2±8.8
Elapsed time from arrival to surgery (min)	74.4±71.4

Values are presented as mean±standard deviation.

other survived.

CPB was used in 4 patients. Three of them had left ventricle injuries, and the other had a right atrium (RA)/inferior vena cava (IVC) junction injury. Among these 4 patients, 2 patients had major combined injuries (Table 2). Therefore, they underwent a co-operation (open reduction and vessel repair) to prevent additional bleeding due to systemic heparinization.

The preoperative lab findings, systolic blood pressure, and elapsed time from arrival to surgery are summarized in Table 3. Creatine kinase–myocardial band (CK-MB) levels (13.4±9.2 ng/mL) were elevated in most patients. In contrast, the preoperative crea-

Table 4. Comparison between survivors and fatalities

Variable	Survivors (n=16)	Fatalities (n=5)	p-value
Age (yr)	50.3±15.3	50.2±31	0.842
Lactate (mmol/L)	5.3±3.2	7.8±0.22	0.212
Creatine kinase–myocardial band isoenzyme (ng/mL)	22.7±28.7	78.5±110.4	0.042
Troponin I (ng/dL)	2.2±4.1	2.8±2.1	0.133
Creatinine (mg/dL)	1.1±0.3	1.1±0.2	0.495
Hematocrit (g/dL)	38.3±4.2	34.8±6.0	0.109
Platelet count (10^3/mL)	196.8±53	144.8±46	0.004
Central venous pressure (cm H_2O)	18.3±5.9	17.3±12.5	0.905
Elapsed time to surgery (min)	66.1±62.4	112.3±104.4	0.445
SBP (mm Hg)	87±22.1	75±19.1	0.354
Ventilator care (hr)	65.4±141.3	11.9±12.9	0.842
Hospital days (day)	38.6±30.9	9.0±14.7	0.313
Intensive care unit stay duration (day)	11.4±12.9	8.5±14.3	0.445
Injury Severity Score	23.2±6.5	33.5±9.3	0.004
Glasgow Coma Scale	13.7±2.5	6.8±4.6	0.007
Trauma and Injury Severity Score	99.2±1.8	87.1±13.2	0.007
Blood transfusion (packs)	6.43±2.98	9±8.97	0.900
Shock index (heart rate/SBP)	1.3±0.56	1.65±0.54	0.186
Diagnostic method			
Chest CT	8 (50.0)	2 (40.0)	0.696
Echocardiography	4 (25.0)	3 (60.0)	0.147
CT and echocardiography	4 (25.0)	0	0.214
Injury location			0.437
Left atrium	2 (12.5)	0	
RA	7 (43.8)	2 (40.0)	
RA-superior vena cava	2 (12.5)	0	
RA-IVC	1 (6.3)	0	
RV-IVC	1 (6.3)	0	
Left ventricle	1 (6.3)	2 (40.0)	
RV	2 (12.5)	1 (20.0)	
Cardiopulmonary bypass	2 (12.5)	2 (40.0)	0.182
Cardiopulmonary resuscitation	1 (6.3)	1 (20.0)	0.372

Values are presented as mean±standard deviation or number (%), unless otherwise stated.
SBP, systolic blood pressure; CT, computed tomography; RA, right atrium; IVC, inferior vena cava; RV, right ventricle.

tinine levels (1.1±0.3 mg/dL) were nearly normal in all patients. The time from hospital arrival to surgery ranged from 10 to 260 minutes. In addition, the mean blood transfusion amount in survivors was 6.3 units, as compared to 9 units in fatalities (p=0.900).

A comparison between the survivors and fatalities is presented in Table 4. There were 16 survivors and 5 fatalities. No instances of intraoperative mortality occurred, but the in-hospital mortality rate was 24% (5 of 21). The causes of death included pneumonia (1), inadequate myocardial protection-related low cardiac output (1), and massive bleeding-related disseminated intravascular coagulation (3). All fatalities showed liver and renal failure, and 2 of them showed brain death. The clinical characteristics of each patient are presented in Table 2.

Significant differences were found in preoperative CK-MB levels (p=0.042) and platelet counts (p=0.004) between the survivors and fatalities. In addition, the preoperative Glasgow Coma Scale score (p=0.007), Trauma and Injury Severity Score (p=0.007), and ISS were significantly worse in the fatalities than in the survivors (p=0.004). No significant differences were found between the two groups in preoperative creatinine levels (p=0.495) or elapsed time to surgery (p=0.445). Similarly, no significant differences were found in injury location (p=0.437), CPR history (p=0.372), use of CPB (p=0.182), amount of blood transfusion (p=0.900), or shock index (p=0.186) between the two groups.

Discussion

Most patients who experience blunt traumatic cardiac rupture die before arrival to the hospital. According to one Japanese study, 91% of patients with blunt traumatic cardiac rupture died within 30 minutes of the accident [7]. We analyzed 21 patients who experienced blunt traumatic cardiac rupture and compared our results with those of previous studies.

Most of our results are consistent with those of previous papers. Traffic accidents were the most common cause of blunt traumatic cardiac rupture (80%), and the most common site of injury was the right atrium (43%) [2,5,7-9]. However, some of our results were different from those of previous studies.

When a patient arrived at the hospital, we first focused on the vital signs, peripheral circulation, presence of an open wound, neck vein distension, and facial plethora. If cardiac rupture was suspected, we performed chest computed tomography, focused assessment with sonography for trauma, or transthoracic echocardiography as soon as possible. If patients had unstable vital signs, we performed intubation and transfusion, using intravenous inotropics as needed. If patients had hemopneumothorax, we inserted a chest tube at the emergency department and then rapidly transferred the patients to the operating room.

Ten of the 21 patients were suspected to have cardiac tamponade because they had hypotension, high CVP, and echocardiographic signs of tamponade (the presence of pericardial effusion, diastolic collapse of the right ventricle, IVC dilatation, etc.). When we suspected cardiac tamponade, we rapidly moved the patient to the operating room rather than trying to insert a pericardial catheter in the emergency room, because it takes almost the same time to perform a pericardial catheter insertion as to prepare the operating room in our hospital. The average elapsed door-to-surgery time in patients with cardiac tamponade was 29 minutes. In contrast, when cardiac tamponade was not suspected, further evaluations were performed prior to surgery.

If a patient’s vital signs are unstable, we consider an emergency department thoracotomy to have several advantages, including the prompt decompression of pericardial pressure and the rapid restoration of hemodynamics. However, our emergency room had no facilities available for emergency department thoracotomies.

All cases were performed via median sternotomy. CPB was applied in 4 patients with left ventricular rupture, and in 1 patient with an RA/IVC junction injury.

Sixteen (75%) of the 21 patients survived. The overall survival rate was similar to that of a prior study, which found that 70%–80% of patients who were transferred to trauma centers survived after blunt cardiac injury [8].

Several discrepancies are present between our results and those of Nan et al. [4]. They found significant differences between survivors and fatalities in preoperative creatinine levels, but no differences in preoperative CK-MB or platelet count [4]. In contrast, we found no significant difference in preope-

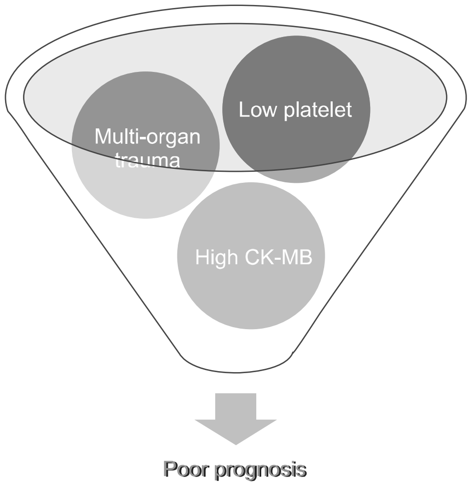

Fig. 1. Elevated CL-MB, a low platelet count, and multi-organ traumatic injuries were associated with a poor prognosis after blunt traumatic cardiac injury. CK-MB, creatine kinase–myocardial band isoenzyme.

rative creatinine levels between survivors and fatalities. In addition, our results revealed significant differences in preoperative CK-MB levels and platelet counts between the two groups.

Several possible reasons exist for these discrepancies. Patients with impaired renal function preoperatively would be expected to have poorer postoperative outcomes than those with normal renal function. In our study, however, all patients had normal preoperative creatinine levels. This may explain why we did not identify a significant difference between survivors and fatalities.

Elevated preoperative CK-MB may be associated with a poor prognosis. This enzyme is not specific for cardiac injury, but can reflect severe muscle injury and potentially predict rhabdomyolysis. According to a 1998 study by Swaanenburg et al. [10], among 38 patients with thoracic injuries, 18–30 had increased CK-MB levels, increased CK-MB activity, an elevated ratio of CK-MB activity to total CK, or an elevated ratio of CK-MB mass to total CK upon admission. Although elevated CK-MB levels are not specific for cardiac injury, they can be indicative of severe combined injuries and be associated with a poor prognosis.

In addition, a low preoperative platelet count may also be associated with a poor prognosis. Nijboer et al. [11] recently reported that the early-stage platelet count was independently related to mortality in blunt trauma patients. A low platelet count reflects increased platelet consumption as well as massive blood loss and hemodilution. Increased platelet consumption can lead to a systemic inflammatory response or disseminated intravascular coagulation [11]. Early platelet consumption can be explained based on the fact that platelet aggregation and blood clot formation begins within 15–20 seconds of a major vessel rupture [12].

We did not find any significant difference in elapsed time from hospital arrival to surgery between fatalities and survivors. Disease severity may have acted as a suppressor variable. For instance, when a patient was suspected to have cardiac tamponade, surgery was promptly performed within 30 minutes. In contrast, surgery was delayed more in hemodynamically stable patients.

1) Study limitations

This was a case-control study, and case-control studies are prone to bias and confounding. The small, heterogeneous study population is a major limitation of this study. Although we were able to identify some characteristics of survivors, we were not able to clearly confirm causality. Given this limitation, we attempted to identify factors associated with a poor prognosis. Larger studies in the future are needed to substantiate our findings.

2) Conclusion

Few prior studies have investigated blunt traumatic cardiac rupture due to the rarity of such cases. This study analyzed factors associated with a poor prognosis of blunt traumatic cardiac rupture. We found that elevated CK-MB levels, a low platelet count, and multi-organ traumatic injury were associated with a poor prognosis (Fig. 1). If a patient with blunt traumatic cardiac rupture exhibits these factors, clinicians should be especially attentive and respond promptly to save the patient's life.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

Acknowledgments

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References

1. Bright EF, Beck CS. *Nonpenetrating wounds of the heart: a clinical and experimental study*. Am Heart J 1935;10: 293-321.
2. Brathwaite CE, Rodriguez A, Turney SZ, Dunham CM, Cowley R. *Blunt traumatic cardiac rupture: a 5-year experience*. Ann Surg 1990;212:701-4.
3. Kang JK, Yoon YS, Kim HT, Park ID, Soh DM, Lee CJ. *Surgical management of traumatic cardiac injury*. Korean J Thorac Cardiovasc Surg 2004;37:335-41.
4. Nan YY, Lu MS, Liu KS, et al. *Blunt traumatic cardiac rupture: therapeutic options and outcomes*. Injury 2009;40: 938-45.
5. Martin TD, Flynn TC, Rowlands BJ, Ward RE, Fischer RP. *Blunt cardiac rupture*. J Trauma 1984;24:287-90.
6. Perchinsky MJ, Long WB, Hill JG. *Blunt cardiac rupture: the Emanuel Trauma Center experience*. Arch Surg 1995; 130:852-6.
7. Namai A, Sakurai M, Fujiwara H. *Five cases of blunt traumatic cardiac rupture: success and failure in surgical management*. Gen Thorac Cardiovasc Surg 2007;55:200-4.
8. Fitzgerald M, Spencer J, Johnson F, Marasco S, Atkin C, Kossmann T. *Definitive management of acute cardiac tamponade secondary to blunt trauma*. Emerg Med Australas 2005;17:494-9.
9. Fulda G, Brathwaite CE, Rodriguez A, Turney SZ, Dunham CM, Cowley RA. *Blunt traumatic rupture of the heart and pericardium: a ten-year experience (1979-1989)*. J Trauma 1991;31:167-72.
10. Swaanenburg JC, Klaase JM, DeJongste MJ, Zimmerman KW, ten Duis HJ. *Troponin I, troponin T, CKMB-activity and CKMB-mass as markers for the detection of myocardial contusion in patients who experienced blunt trauma*. Clin Chim Acta 1998;272:171-81.
11. Nijboer JM. *New insights in outcome after major trauma*. Groningen: University Library Groningen; 2009.
12. Guyton AC, Hall JE. *Textbook of medical physiology*. Philadelphia (PA): Saunders; 2000.