Impact of Controlled Pericardial Drainage on Critical Cardiac Tamponade With Acute Type A Aortic Dissection

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Background—Cardiac tamponade is associated with fatal outcomes for patients with acute type A aortic dissection, and the presence of cardiac tamponade should prompt urgent aortic repair. However, treatment of the patient with critical cardiac tamponade who cannot survive until surgery remains unclear. We analyzed our experience of controlled pericardial drainage (CPD) managing critical cardiac tamponade.

Methods and Results—Between September 2003 and May 2011, 175 patients with acute type A aortic dissection were treated surgically, including 43 (24.6%) who presented with cardiac tamponade onarrival. Eighteen patients, who did not respond to intravenous volume resuscitation, underwent CPD in the emergency department. An 8F pigtail drainage catheter was inserted percutaneously, and drainage volume was controlled by means of several cycles of intermittent drainage to maintain blood pressure at ≈90 mm Hg. After CPD, all of the patients were transferred to the operating room, and immediate aortic repair was performed. Systolic blood pressure before CPD was 64.3±8.2 mm Hg and elevated significantly in all of the cases after CPD. Systolic blood pressure after CPD was 94.8±10.5 mm Hg, and increase in systolic pressure was 30.5±11.7 mm Hg. Total volume of aspirated pericardial effusion was 40.1±30.6 mL, and 10 patients required only ≤30-mL aspiration volume. All of the patients underwent aortic repair successfully. In-hospital mortality was 16.7%; however, there was no complications or mortality related to CPD.

Conclusions—Preoperative pericardial drainage with control of volume is a safe and effective procedure for acute type A aortic dissection complicated by critical cardiac tamponade. In our patient population, timely controlled pericardial drainage is warranted. (Circulation. 2012;126[suppl 1]:S97–S101.)

Key Words: aortic dissection ■ aortic surgery ■ cardiac tamponade ■ pericardiocentesis ■ surgery

Preoperative cardiac tamponade is an ominous clinical predictor of poor outcomes, as well as the leading cause of mortality of acute type A aortic dissection (AADA). The optimal management of cardiac tamponade in the context of AADA remains to be solved. The presence of cardiac tamponade should prompt urgent aortic repair. However, controversy surrounds how to treat patients with hemopericardium and cardiac tamponade who cannot survive until surgery.

Pericardiocentesis has been recognized as the primary treatment of choice for cardiac tamponade caused by various diseases, except for hemopericardium, which complicates AADA. Careless drainage risks aggravating the leak or rupture by restoring arterial pressure, which then exacerbates the tear and the driving pressure of the leak, thereby increasing mortality. In guidelines offered by the European Society of Cardiology, pericardiocentesis was not recommended and was classified as a class III management of choice for patients with AADA. However, according to recent published guidelines for the diagnosis and management of patients with thoracic aortic disease, pericardiocentesis can be performed by withdrawing just enough fluid to restore perfusion in patients with hemopericardium and cardiac tamponade who cannot survive until surgery. In addition, cardiac tamponade is a considerable risk when anesthesia is induced, because preload reduction by anesthetic agents may precipitate electromechanical dissociation.

We focused on a percutaneous drainage procedure using an 8F pigtail drainage catheter, which has been widely used to drain the pericardial space (PS) and the pleural space. The volume of drainage of fluid could be controlled by a milliliter, and an abrupt rise in blood pressure could be avoided during drainage if applied to cardiac tamponade. Therefore, we postulated that blood pressure could be mildly restored through volume controlled pericardial drainage (CPD) and the patient could survive until aortic repair.
Methods

Patient Characteristics
Between September 2003 and May 2011, 175 consecutive patients presented with AADA and underwent aortic repair in double cardiovascular centers (153 cases in Kobe Red Cross Hospital/Hyogo Emergency Medical Center and 22 cases in Seirei-Mikatahara General Hospital). On arrival, all of the patients underwent echocardiography and computed tomography. Pericardial effusion (PE) was recorded in 92 patients, and 43 of those 92 patients arrived with low systolic blood pressure (<90 mm Hg). Cardiac tamponade was clinically diagnosed in cases in which fluid in the PS and collapsed right ventricle were observed through bedside echocardiography, along with an unstable hemodynamic state (blood pressure ≤90 mm Hg). Initially, intravenous volume resuscitation was performed to elevate blood pressure and to maintain a stable hemodynamic state. For volume resuscitation, infusion of 500 mL of crystalloid solution in 5 minutes was performed initially and followed with additional infusion of 500 mL of crystalloid in 5 to 10 minutes, if blood pressure did not respond. In addition, endotracheal intubation was performed if necessary. However, 18 patients did not respond to rapid volume resuscitation and showed hypotension (systolic blood pressure was kept <80 mm Hg) while being prepared for the operating room to undergo aortic repair. We surmised that pericardial drainage was the only treatment that would restore circulation, so the 18 patients underwent CPD in the emergency department.

Procedure
With the patients in a prone position and under local anesthesia, an 8F pigtail drainage catheter with multiple side holes (Aspiration Seldinger kit/Nippon Sherwood Medical Industries Ltd, Tokyo, Japan) was inserted into the PS under ultrasound guidance, as described below. In acute cardiac tamponade caused by AADA, PE was more accumulated around the apex of the heart rather than just above the diaphragm (Figure 1A). Usually PE was thickest just beneath the skin of the fourth or fifth intercostal space and on or just inside of the left midclavicular line. Before CPD, the thickest point was marked up on the skin under echocardiography (Figure 1A). As described in Figure 1, puncture was performed from the marked up point at a 90° angle to the skin using an 18-gauge plastic shielded catheter and carefully advance a needle vertically to the PS. Within 3.0 cm from the skin, a point of needle could be reached into the PS. Then, the plastic sheath was left in place, and a guide wire (0.89 mm in diameter) was introduced, followed with advancing an 8F pigtail catheter into the PS. To avoid complications, such as laceration of coronary artery, vein, or myocardium, thickness of the PE needs to be >10 mm in length, and a needle should be inserted meticulously. If the needle was advanced laterally, injury of the lung and pneumothorax might be induced.

This procedure took 5.3±1.8 minutes (Table). After the catheter had been placed into the PS, we started to aspirate the PE. At the beginning, 5 to 10 mL of hemopericardium were aspirated, and changes in blood pressure were closely observed to prevent the excessive elevation of blood pressure. The drainage volume was controlled through intermittent aspiration, using a 10-mL syringe and 5 to 10 mL of aspiration each time, to maintain systolic blood pressure at ~80 to 90 mm Hg (Figure 2). After CPD was performed and circulation was restored, the patients were transferred to the operating room to undergo immediate aortic repair. The time interval between CPD and surgery was 58.2±25.0 minutes (Table).

Figure 1. A, Schematic illustration demonstrates accumulation of the pericardial effusion of acute type A aortic dissection (AADA) in a prone position, and black arrow shows the point and direction of the controlled pericardial drainage (CPD). Schema (B) and thoracic computed tomography (D) of patient 1 demonstrates AADA with massive pericardial effusion with compression of the right ventricle, and black arrow shows angle of the puncture is 90° to the skin. C, Ultrasound findings from the fifth costal space shows pericardial effusion just beneath the skin. PE indicates pericardial effusion; AO, aorta; LV, left ventricle; RV, right ventricle.

Figure 2. Actual tracing of the radial artery blood pressure of patient 1. After 10 mL of drainage, blood pressure elevated from 64/33 mm Hg to 85/77 mm Hg. CPD indicates controlled pericardial drainage.
Operative Approach
All of the patients underwent emergency aortic repair. The aortic repairs were conducted via a median sternotomy, using full heparinization, cardiopulmonary bypass, and profound hypothermic circulatory arrest with antegrade cerebral perfusion. Arterial cannulation was performed via the femoral artery in all cases. Once a nasopharyngeal temperature of 18° centigrade had been reached, cardiopulmonary bypass was discontinued and circulation was arrested. Antegrade perfusion of the left subclavian artery, left common carotid artery, and innominate artery was started by means of direct cannulation into the true lumen from inside the aortic arch, and the antegrade perfusion flow was maintained. The intimal tear was isolated to the proximal transverse aortic arch in 14 cases and a hemiarch replacement performed. Four cases of total arch replacement were performed when the intimal tear involved the distal transverse arch. After completion of the distal arch reconstruction, antegrade cerebral perfusion was discontinued, and cardiopulmonary bypass flow was resumed with systemic warming initiated until body temperature was at 35° centigrade.

Statistical Analysis
Data collection and analysis were approved by Kobe Red Cross Hospital/Hyogo Emergency Medical Center and Seirei-Mikatagahara General Hospital Committee for the Protection of Human Subjects. Analysis was completed retrospectively. Data were collected from chart reviews by the authors (T.T. and T.H.). Categorical variables are described as numbers and percentages, and continuous variables are described as mean±SD. A paired t test was performed to assess the difference in systolic blood pressure between pre-CPD and post-CPD, with P<0.05 considered statistically significant. Data analyses were performed with SPSS software (version 17.0; SPSS, Inc, Chicago, IL).

Results
Preoperative patient characteristics, effect of CPD, procedures, and operative mortality and morbidity are listed in Table. The mean age of the patients was 75.0±8.0 years (ranging from 58 years to 89 years), and 33% were men. Thirteen patients required endotracheal intubation and ventilation, because of poor oxygenation, before arriving at our hospital or in the emergency department. Two patients required cardiopulmonary resuscitation because of pulseless electric activity in the emergency department, and CPD was performed.

Findings of the thoracic computed tomography scan on admission showed that the maximum thickness of PE was 13.4±9.7 mm (ranging from 1.7 to 27.7 mm). These findings demonstrated that even a little increase in intrapericardial volume caused critical cardiac tamponade. Systolic blood pressure before CPD was 64.3±8.2 mm Hg (ranging from 50 to 80 mm Hg). Blood pressure was elevated significantly when compared with systolic BP before CPD (P<0.0001) in all of the cases after CPD; systolic blood pressure after CPD was 94.8±10.5 mm Hg (ranging from 82 to 120 mm Hg) and the increase in systolic pressure was 30.5±11.7 mm Hg (Figure 3). The total volume of aspirated PE ranged from 10 to 130 mL (40.1±30.6 mL), and 10 of 18 patients required only ≤30-mL volume of aspiration to improve their blood pressure. There were no major complications related to CPD, and none of the patients died preoperatively.

Replacement of the entire aortic arch was performed in 4 patients, and replacement of the hemiarch was performed in 14 patients (Table). Postoperatively, 3 patients died and overall in-hospital mortality was 16.7%. The causes of death included rupture of the descending aorta on the fifth day after surgery, renal and hepatic failure on the 24th postoperative day, and pneumonia on the 55th postoperative day. There was no mortality related to preoperative CPD.

Discussion
Pericardial fluid collection is a frequent complication of AADA. Most commonly, the transudation of fluid across the thin wall of an adjacent false lumen into the PS leads to a hemodynamically insignificant PE, which is present in one third of patients.10 The incidence of cardiac tamponade was reported in 8%-31% of patients with AADA.1,10-15 Cardiac tamponade is one of the important risk factors in poor outcomes. According to Rampoldi et al,1 prolonged hypotension from

<table>
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<th>Characteristic</th>
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<tbody>
<tr>
<td>Age, y</td>
<td>75.0±8.0</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>6 (33)</td>
</tr>
<tr>
<td>Shock on arrival, n (%)</td>
<td>18 (100)</td>
</tr>
<tr>
<td>CPR required, n (%)</td>
<td>2 (11)</td>
</tr>
<tr>
<td>Endotracheal intubation required, n (%)</td>
<td>13 (72)</td>
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<tr>
<td>Controlled pericardial drainage performed, n (%)</td>
<td>18 (100)</td>
</tr>
<tr>
<td>Thickness of PE, mm</td>
<td>13.4±9.7</td>
</tr>
<tr>
<td>Set-up time, min</td>
<td>5.3±1.8</td>
</tr>
<tr>
<td>Systolic BP before CPD, mm Hg</td>
<td>64.3±8.2</td>
</tr>
<tr>
<td>Total volume of aspiration, mL</td>
<td>40.1±30.6</td>
</tr>
<tr>
<td>Volume of aspiration ≤30 mL, n (%)</td>
<td>10 (56)</td>
</tr>
<tr>
<td>Systolic BP after CPD, mm Hg</td>
<td>94.8±10.5†</td>
</tr>
<tr>
<td>Increase in systolic BP, mm Hg</td>
<td>30.5±11.7</td>
</tr>
<tr>
<td>Time interval between CPD and surgery, min</td>
<td>58.3±25.0</td>
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†P<0.0001 when as compared with systolic BP before CPD.
admission to the operating room is associated with fatal outcomes in >40% of patients. Gilon et al.12 reported that inhospital mortality from AADA with cardiac tamponade was 54%, which was more than twice the mortality rate without cardiac tamponade (24.6%; P<0.0001). In addition, when cardiac tamponade occurs in patients with AADA, many patients die before reaching the hospital and before diagnosis is made. Pericardiocentesis has been proven to be a safe and effective procedure in the treatment of cardiac tamponade caused by various underlying diseases.7 However, in the case of cardiac tamponade in the context of AADA, indications for pericardiocentesis are still a matter of controversy. Isselbacher et al.8 reported the following catastrophic results: in 6 patients presenting with cardiac tamponade in the context of AADA on arrival to the hospital or during emergency department evaluation, 4 patients underwent pericardiocentesis and 3 patients died after pericardiocentesis. Because the remaining 2 patients, who did not have pericardiocentesis, survived, they speculated that pericardiocentesis induced intensified bleeding and extension of the dissection of the aorta.8 They stated that blood pressure in 4 patients before pericardiocentesis was 90 to 130 mm Hg, and blood pressure after pericardiocentesis was elevated to between 90 and 170 mm Hg. Volume of fluid that was aspirated among the patients was 0, 100, 250, and 300 mL, respectively. The only patient to survive had a blood pressure reading after pericardiocentesis of 100/50 mm Hg, with a volume of liquids that was aspirated of 0 mL. Therefore, they postulated that cardiac tamponade might have a role to play in minimizing the progression of aortic dissection by lowering blood pressure and reducing the cardiac ejection function. Elevated blood pressure and the dislodging of a thrombus from the PS during pericardiocentesis might cause a closed communication to reopen.8 Failure of the method of Isselbacher et al.8 might be caused by excessive high pressure, which causes rupture or more extension of dissection not only by open communication.

Our series of pericardial drainage procedures provided a better outcome compared with the study by Isselbacher et al.8 That is because the indication of pericardiocentesis was from patients who displayed hypotension after intravenous fluid resuscitation and with an average systolic blood pressure before drainage of 64.3 mm Hg, which was lower than that cited by Isselbacher et al.8 Drainage of PE cited in our study was more finely controlled, and the total volume of fluids that were aspirated was ≈40 mL. That included 10 of the 18 patients and required a volume of aspiration of only ≈30 mL, again, much smaller than those in the study by Isselbacher et al.8 As a result, the elevation of systolic blood pressure was ≈30 mm Hg. In addition, systolic blood pressure after drainage was ≈95 mm Hg in our patients and was achieved without inducing fatal bleeding from the aorta.

From the aspect of physiological changes produced by tamponade, the amount of aspirated blood should be small enough to stabilize circulation of critical cardiac tamponade. The normal PS has a steep pressure-volume curve.16 Rapidly increasing pericardial fluid first reaches the limit of the pericardial reserve volume and then quickly exceeds the limit of parietal pericardial stretch, causing a steep rise in pressure. This becomes even steeper as smaller increments in fluid cause a disproportionate increase in pericardial pressure.16 The end result of an increase in pericardial pressure is an impediment caused by the diastolic filling of the ventricles, resulting in a precipitous decrease in cardiac output and arterial blood pressure and hemodynamic collapse.15 Therefore, draining a small volume of fluid in the PS results in a drastic decrease in pericardial pressure and in rapid clinical and hemodynamic improvement.17

CPD can be performed safely and prompt applied in the emergency department while the patient is awaiting surgery. Importantly, CPD has a closed drainage system through an indwelling 8F pigtail catheter, so the volume of fluids that are aspirated can be easily controlled by a milliliter, while keeping attention so that blood pressure does not rise too high. A few precautions must be taken during CPD treatment. In all of our cases, the catheter is inserted percutaneously from either the fourth or fifth intercostal space and on the left midclavicular line under ultrasound guidance. Care must be taken to avoid pneumothorax, injury to the heart and coronary vessels, or the insertion of the catheter into the clot. Ultrasound guidance is quite effective in allowing us to place the catheter in the exact space. In our experience, no pneumothorax complications or injuries to the heart were recorded.

Several previous studies reported the effectiveness of pericardiocentesis for cardiac tamponade complicating AADA.18–20 In the majority of those studies, however, routine pericardiocentesis was considered a harmful technique for patients with cardiac tamponade in the context of AADA and recognized as a major contraindication.7–9 Even when blood pressure cannot be elevated after massive intravenous volume infusion and the patient may not survive until reaching the operating room, an attempt to resuscitate the patient with pericardiocentesis is warranted and might indeed be successful.18,19 In recent published guidelines, pericardiocentesis is to be performed by withdrawing just enough fluid to restore perfusion in patients with hemopericardium and cardiac tamponade who cannot survive until surgery.10 Our cases suggested the usefulness of CPD for critical cardiac tamponade. However, in cases of an acute accumulation of the hemopericardium from rupture of the false lumen, CPD could not improve the circulation because of the drainage tube’s small diameter. In such cases, cardiopulmonary resuscitation is mandated, and open pericardial drainage from subxyphoidal approach must be challenged if pulseless electric activity continues before emergency mobilization to the operating room for aortic repair.

Conclusions

Cardiac tamponade is associated with fatal outcomes in patients with type A aortic dissection and is considered an important risk factor. CPD should be one of the treatment options to improve hemodynamic instability in patients with cardiac tamponade that complicates AADA. An improvement in the patient’s preoperative state may lead to improve outcomes of AADA with cardiac tamponade.

Acknowledgments

We gratefully acknowledge the support of the emergency department of Japanese Red Cross Kobe Hospital and Hyogo Emergency Medical Center. We thank the following colleagues for their support: Haruki Nakayama, MD; Akihiro Usui, MD; Shigenari Matsuyama, MD; Sinichi Nakayama, MD.
Disclosures

None.

References


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Circulation. 2012;126:S97-S101
doi: 10.1161/CIRCULATIONAHA.111.082685
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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