Cardiac Tamponade Complicating Proximal Aortic Dissection

Is Pericardiocentesis Harmful?

Eric M. Isselbacher, MD; Joaquin E. Cigarroa, MD; Kim A. Eagle, MD, FACC

**Background** Cardiac tamponade frequently complicates acute proximal aortic dissection and is one of the most common causes of death from aortic dissection. Well-defined strategies for the management of acute aortic dissection now exist; however, little is known about how best to manage the hemopericardium that may complicate it.

**Methods and Results** Using a computer-based review, we retrospectively identified 10 patients presenting to our hospital over a 13-year period who were diagnosed with both aortic dissection and cardiac tamponade. All 10 had proximal dissections. Three of the 10 presented as the sudden onset of fatal electromechanical dissociation, 6 presented with hypotension, and 1 was normotensive on presentation. Of the 7 hypotensive or normotensive patients diagnosed with cardiac tamponade, 4 underwent successful pericardiocentesis while awaiting surgery. At time intervals of 5 to 40 minutes after their pericardiocenteses, 3 of the 4 patients experienced sudden onset of electromechanical dissociation and death; the fourth patient survived and underwent surgical repair. Of the 3 hypotensive or normotensive patients who had either no pericardiocentesis or an unsuccessful pericardiocentesis, all 3 underwent successful surgical repair and survived.

**Conclusions** In this study, patients with an aortic dissection complicated by cardiac tamponade have an early mortality of 60%. While 3 of the 10 died from electromechanical dissociation immediately upon presentation, the 3 other deaths all occurred shortly after successful pericardiocentesis, a procedure undertaken to stabilize them. While the number of patients in this series is small, the observations do raise the possibility that in patients with cardiac tamponade complicating aortic dissection pericardiocentesis could be harmful rather than beneficial. Possible mechanisms for why the performance of pericardiocentesis might destabilize such patients are proposed. (Circulation. 1994;90:2375-2378.)

**Key Words** • aorta • tamponade • pericardiocentesis • pericardium

A acute aortic dissection has an early mortality as high as 1% per hour,1 with nearly two thirds of deaths in cases of proximal dissection resulting from the extension of the dissection into the aortic root producing hemopericardium and cardiac tamponade.2,3 Extensive clinical investigation has resulted in a well-defined strategy for prompt treatment of aortic dissection, which has greatly improved survival rates. Despite advances in treatment for aortic dissection, little is known about how best to manage the hemopericardium that frequently complicates proximal aortic dissection and may lead to death.4,5 Since pericardiocentesis is considered the standard treatment for patients suffering from cardiac tamponade, it is not surprising that it has been used as an interim treatment for patients with cardiac tamponade complicating aortic dissection while they await definitive surgical repair.5,7 However, some authors have raised concerns that performing pericardiocentesis in such cases may in fact be dangerous—possibly precipitating hemodynamic collapse and death5—and should therefore be avoided. To assess the utility and safety of pericardiocentesis in the management of cardiac tamponade complicating aortic dissection, we reviewed the care and outcome of all patients at our institution who presented over the past 13 years with this condition.

**Methods** We identified all patients presenting with acute aortic dissection and suspected or documented cardiac tamponade admitted to the Massachusetts General Hospital during the period from January 1980 through September 1993. A computer-based review of all discharge diagnoses was performed with the inclusion criteria of (1) the diagnosis of aortic dissection and (2) the diagnosis of cardiac tamponade or the performance of pericardiocentesis. Demographic information, clinical and hemodynamic data, the performance and results of diagnostic studies, and information on each patient’s hospital course and in-hospital (early) mortality were collected retrospectively by review of the medical record.

**Results** A total of 148 patients were identified with the diagnosis of acute aortic dissection over a 13-year period, and 10 of the patients were also diagnosed as having cardiac tamponade. All 10 had proximal aortic dissections that were documented with either aortography or computed tomographic (CT) scanning. In all cases, the diagnosis of cardiac tamponade was made by either echocardiography, the finding of equal pressures at right heart catheterization, opening of the chest during cardiopulmonary resuscitation, or on postmortem examination.

The Table provides a summary of the signs, symptoms, and findings that suggested the diagnosis of cardiac tamponade, the time at which cardiac tampon-
Summary of Symptoms, Blood Pressure, Management, and Outcomes of the 10 Patients With Cardiac Tamponade Complicating Aortic Dissection

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, y</th>
<th>Sex</th>
<th>When Tamponade First Presented</th>
<th>Presence of Tamponade Suggested by:</th>
<th>BP When Tamponade Presented</th>
<th>BP After IV Fluid Resuscitation</th>
<th>Pericardiocentesis Attempted</th>
<th>Volume of Fluid Aspirated (mL)</th>
<th>BP After Pericardiocentesis</th>
<th>In-hospital Survival, (Time of Death if After Pericardiocentesis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57</td>
<td>F</td>
<td>2 Days after aortic dissection diagnosed*</td>
<td>Equalization of pressures at right heart catheterization followed by EMD</td>
<td>EMD</td>
<td>EMD</td>
<td>No</td>
<td>...</td>
<td>...</td>
<td>Died</td>
</tr>
<tr>
<td>2</td>
<td>78</td>
<td>M</td>
<td>Postoperative day 6 after cardiac surgery</td>
<td>Sudden onset of EMD</td>
<td>EMD</td>
<td>EMD</td>
<td>No</td>
<td>...</td>
<td>...</td>
<td>Died</td>
</tr>
<tr>
<td>3</td>
<td>52</td>
<td>M</td>
<td>9th Day after aortic dissection diagnosed*</td>
<td>Sudden onset of EMD</td>
<td>EMD</td>
<td>EMD</td>
<td>Yes</td>
<td>0</td>
<td>EMD</td>
<td>Died</td>
</tr>
<tr>
<td>4</td>
<td>63</td>
<td>F</td>
<td>During emergency room evaluation</td>
<td>Recurrence of chest pain followed by hypotension</td>
<td>60/palp</td>
<td>70/palp</td>
<td>No</td>
<td>...</td>
<td>...</td>
<td>Survived</td>
</tr>
<tr>
<td>5</td>
<td>60</td>
<td>M</td>
<td>During emergency room evaluation</td>
<td>Recurrence of chest pain followed by hypotension</td>
<td>80/palp</td>
<td>100/60</td>
<td>No</td>
<td>...</td>
<td>...</td>
<td>Survived</td>
</tr>
<tr>
<td>6</td>
<td>73</td>
<td>F</td>
<td>On arrival to emergency room</td>
<td>Hypotension</td>
<td>80/palp</td>
<td>120/70</td>
<td>Yes</td>
<td>100 mL</td>
<td>170/80</td>
<td>Died (5 minutes)</td>
</tr>
<tr>
<td>7</td>
<td>83</td>
<td>M</td>
<td>On admission</td>
<td>Hypotension and cardiomegaly on chest radiograph</td>
<td>80/palp</td>
<td>90/palp</td>
<td>Yes</td>
<td>300 mL</td>
<td>90/palp</td>
<td>Died (20 minutes)</td>
</tr>
<tr>
<td>8</td>
<td>59</td>
<td>F</td>
<td>On admission</td>
<td>Hypotension</td>
<td>70/palp</td>
<td>100/50</td>
<td>Yes</td>
<td>0</td>
<td>100/50</td>
<td>Survived</td>
</tr>
<tr>
<td>9</td>
<td>61</td>
<td>M</td>
<td>2 Weeks after aortic dissection occurred*</td>
<td>Acute onset of hypotension</td>
<td>60/palp</td>
<td>165/105</td>
<td>Yes</td>
<td>1200 mL</td>
<td>170/83</td>
<td>Survived</td>
</tr>
<tr>
<td>10</td>
<td>65</td>
<td>M</td>
<td>On admission</td>
<td>Low cardiac output and high central venous pressure</td>
<td>130/80</td>
<td>130/80</td>
<td>Yes</td>
<td>250 mL</td>
<td>170/80</td>
<td>Died (40 minutes)</td>
</tr>
</tbody>
</table>

BP indicates blood pressure; EMD, electromechanical dissociation.
*After the diagnosis of aortic dissection, surgery was delayed in patient 1 due to a variety of preoperative issues and delayed in patient 3 due to a complicating stroke. In patient 9 (presenting with chest pain), the diagnosis of aortic dissection was unsuspected until 6 days after pericardiocentesis (which was itself performed 14 days after the original onset of chest pain).

ade was diagnosed, each patient’s presenting blood pressure and subsequent blood pressure after intravenous fluid resuscitation, the results of pericardiocentesis (if performed), and the hospital course. Pericardiocentesis, when performed, was done so in an effort to improve the hemodynamic consequences of cardiac tamponade while such patients awaited definitive therapy for aortic dissection. A more detailed description of one patient (patient 10) follows as an illustrative example. A 65-year-old man with long-standing hypertension presented to an outside hospital with the acute onset of tearing anterior chest pain radiating to his back. A contrast-enhanced CT scan was negative for aortic dissection. On the fourth hospital day he developed recurrent chest pain accompanied by hypotension, briefly requiring vasopressors for hemodynamic support. A transthoracic echocardiogram was performed, which revealed an intimal flap in the ascending aorta consistent with aortic dissection together with a large pericardial effusion with signs of cardiac tamponade. He was transferred to Massachusetts General Hospital, where aortography (Figure) revealed a type I aortic dissection (involving the innominate and right common carotid arteries with total occlusion of the left renal artery) as well as mild aortic insufficiency. It was believed that his acute renal failure would increase his
Cardiac Tamponade Complicating Aortic Dissection

Aortogram in the left oblique view confirming the presence of a proximal aortic dissection. The ascending aorta is dilated, and both true (T) and false (F) lumens are readily identified. The intimal flap, seen here as a thin radiolucent line, is found also to extend into the innominate artery.

surgical risk, so before undertaking direct aortic repair, an attempt was made to improve his hemodynamics and renal perfusion by performing pericardiocentesis. Right heart catheterization confirmed the presence of cardiac tamponade, and at pericardiocentesis, 250 mL of dark, nonclotting blood was removed, producing a prompt rise in blood pressure from 130/80 to 170/80 mm Hg (nitroprusside was therefore begun to control this hypertension). A pigtail catheter was left in place in the pericardial space. He was transferred to the coronary care unit, where, upon arrival, he suffered a cardiac arrest with electromechanical dissociation; it was then noted that the pigtail catheter had begun actively draining bright red blood (different from the darker blood observed at pericardiocentesis). Cardiopulmonary resuscitation was unsuccessful, and the patient died 40 minutes after the pericardiocentesis.

As shown in the Table, in 3 of the 10 study patients, cardiac tamponade presented as the sudden onset of fatal electromechanical dissociation; 6 others presented with mild to moderate hypotension, and 1 normotensive patient presented only with jugular venous distention. Of the 6 patients presenting with hypotension, 2 remained hypotensive after intravenous fluid resuscitation, although both patients had normal mental status.

Of the 7 hypotensive or normotensive patients diagnosed with cardiac tamponade, 4 underwent successful pericardiocentesis. At intervals of 5, 20, and 40 minutes, respectively, after their pericardiocenteses, 3 of the 4 patients suffered sudden electromechanical dissociation and death. The fourth suffered no complications before successful surgical repair (with surgery taking place 6 days later due to a late diagnosis of the then unsuspected aortic dissection). In 1 other patient, pericardiocentesis was attempted unsuccessfully, and the patient then underwent successful surgical repair of the dissection.

The final 2 patients with cardiac tamponade were not subjected to pericardiocentesis but were instead taken urgently to the operating room for direct aortic repair. The decision to take patients directly to surgery rather than first performing pericardiocentesis reflected the preferences of the individual cardiologist and cardiac surgeon involved in each case. All 4 patients who underwent surgical repair of their aortic dissections survived the hospitalization.

The mortality among all 10 patients with aortic dissection complicated by cardiac tamponade was 60%. The mortality among those patients presenting with electromechanical dissociation was 100% (3 of 3); the early mortality among those presenting hypotensive or normotensive was 43% (3 of 7). Of the latter group, the early mortality for those undergoing successful pericardiocentesis was 75% (3 of 4), whereas none of those having either no pericardiocentesis or an unsuccessful pericardiocentesis before surgery died (3 of 3).

Discussion

While cardiac tamponade occurs in a minority of patients suffering acute aortic dissection, it is the most common cause of death among these patients.\(^2^3\) In the present study, it is associated with an early mortality of 60% in patients surviving long enough to reach the hospital. While 3 of our 10 patients died from electromechanical dissociation immediately upon presentation of cardiac tamponade, the other 7 remained stable enough to allow consideration of several therapeutic options. The two patients taken directly to the operating room for aortic repair did well. However, in 5 other patients, efforts to stabilize the patients before surgery through pericardiocentesis to relieve cardiac tamponade resulted in unexpected death in 3, which may have been directly precipitated by successful pericardiocentesis. While the number of patients in this study is small, the observations do raise the possibility that pericardiocentesis could be harmful rather than beneficial among patients with cardiac tamponade complicating aortic dissection.

Although pericardiocentesis is safe and effective in treating cardiac tamponade of other causes, particular aspects of the pathophysiology of aortic dissection complicated by hemopericardium could explain the poor outcomes among the patients we studied. The hemopericardium complicating proximal aortic dissection develops as a result of blood leaking into the pericardial space from the false lumen as it dissect back to the aortic root. If such a leak were to become a free communication with the aorta, then the marked elevation of intrapericardial pressure would quickly compress the cardiac chambers and prevent filling, thus resulting in electromechanical dissociation. Pericardiocentesis would probably be futile in such patients because free communication with the false lumen would prevent adequate drainage of the pericardial space.
Because many patients with hemopericardium complicating aortic dissection do not present with electromechanical dissociation, it must be that in such cases, the communication between the pericardial space and the aorta is transient rather than continuous. The mechanisms of the transient nature of such communications are unknown. It may be that the communication between the false lumen closes off due to the intimal flap or local tissues acting as a valve or that thrombus forms in the false lumen or at the site of communication and closes the leak. The hemodynamic effect from cardiac tamponade may help reduce the risk of further bleeding into the pericardial space through a reduction in cardiac output, aortic pressure, and dP/dT. In effect, the tamponade itself reduces further leakage into the pericardium by reducing the pressure gradient between the false lumen and the pericardial space, and perhaps this allows for a tissue valve effect and/or thrombus to seal off the communication. Meanwhile, both the fall in blood pressure and the reduction of dP/dT would help minimize the chance that the aortic dissection might propagate further.

Although hemopericardium may stabilize for a brief time, such stability must be viewed as an extremely tenuous state. Performing pericardiocentesis in this setting would likely reverse the stabilizing hemodynamic consequences of cardiac tamponade. An acute rise in blood pressure would increase the pressure gradient between the false lumen and the pericardial space, which could dislodge a thrombus and cause a closed communication to reopen, with recurrence of bleeding and cardiac tamponade. Furthermore, the acute rise in systemic blood pressure and dP/dT might cause propagation of the aortic dissection such that the false lumen dissects further retrograde into the aortic root producing free communication with the pericardial space.

Such a course of hemodynamic improvement followed by acute decompensation after pericardiocentesis is consistent with the events described in the patient described above. A very similar patient was reported by Coplan et al., who also performed pericardiocentesis in a patient with cardiac tamponade complicating a proximal aortic dissection: After successful pericardiocentesis, the patient had an acute rise in systemic blood pressure that was quickly followed by precipitous hypotension, electromechanical dissociation, and death.

This retrospective study confirms that cardiac tamponade complicating acute aortic dissection is associated with a high early mortality. Prompt treatment is certainly indicated, but the findings presented here suggest that performing pericardiocentesis to stabilize a hypotensive patient in this setting may instead precipitate hemodynamic collapse and death. When cardiac tamponade is discovered in a patient with aortic dissection, it should be considered a surgical emergency, and pericardiocentesis should be avoided while every effort is be made to proceed as urgently as possible to the operating room for direct repair of the aorta with intraoperative drainage of the hemopericardium. However, if a patient’s blood pressure is so low that it compromises cerebral or vital organ perfusion, there may simply not be enough time to wait for surgery in order to save the patient, and in this setting, urgent pericardiocentesis would still be warranted. A prudent strategy in such a case might be to aspirate only enough pericardial fluid to raise the patient’s blood pressure to the lowest acceptable level, thereby minimizing the chance of precipitating a rapid increase in blood pressure and dP/dT, which could prompt a catastrophic recurrence of hemorrhage or induce further extension of the dissection.

References
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