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Early Postoperative Acute Aortic Dissection, the Leading Cause of Sudden Death after Cardiac Surgery? Critical Role of the Computed Tomography Scan

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ABSTRACT

Iatrogenic acute aortic dissection (AOD) is known to occur during cardiac surgery or cardiac catheterization, whereas delayed AOD usually happens up to several years after an uneventful operation. Both entities usually are easily recognized, and their management is well described in the literature. Conversely, early postoperative AOD has not been described with any frequency in the literature, leading one to believe that once surgery is terminated, AOD is unlikely to occur and account for any early postoperative mortality or morbidity. We present our recent experience with 4 patients who sustained early postoperative AOD and whose diagnoses were facilitated by computed tomography (CT) scanning of the chest. Early postoperative acute AOD may not be uncommon and may account for more disasters and deaths than are acknowledged in the literature. Diagnosis is made expeditiously if such AOD is suspected and a CT scan of the chest is done. Prevention may be based on avoiding the manipulation of the ascending aorta and a tighter control of hypertension in the immediate postoperative period. The treatment of this AOD entity is not very difficult and is within the reach of every trained cardiac surgeon.

INTRODUCTION

Iatrogenic acute aortic dissection (AOD) is known to occur during cardiac surgery or cardiac catheterization, whereas delayed AOD usually happens up to several years after an uneventful operation. Both entities usually are easily recognized, and their management is well described in the literature. Conversely, early but not intraoperative AOD has not been described with any frequency in the literature, leading one to believe that once surgery is terminated, AOD is unlikely to occur and account for any early postoperative mortality or morbidity. To illustrate this latter entity, we present our recent experience with 4 patients who sustained early postoperative AOD and whose diagnoses were facilitated by computed tomography (CT) scanning of the chest.

CASE REPORTS

Case 1

E.D., a 74-year-old man with a history of hypertension and recurrent angina, underwent off-pump coronary bypass (OPCAB) surgery on August 1, 2002, 4 days after a myocardial infarction. This surgery consisted of the sequential grafting of a left internal thoracic artery to the left anterior descending coronary artery (LAD) and the diagonal branch and separate reversed saphenous vein grafts (SVG) to the posterior descending branch of the right coronary artery and the marginal branch of the circumflex artery. The intraoperative and immediate postoperative courses were uneventful except for a prolonged episode of atrial fibrillation, for which the patient was treated with full heparinization. On August 8, 2002, he suddenly developed paresis of the lower extremities while walking and had to be returned to bed. Although the patient complained of mild chest discomfort, his chest radiographic and electrocardiographic results remained unchanged, and the results of tests for chemical markers of myocardial infarction were negative. His blood pressure and neurologic status remained stable. Transthoracic echocardiography (TTE) results showed a moderate anterior pericardial hematoma at the base of the heart without signs of tamponade. Heparin treatment was discontinued, and a CT scan of the chest was ordered. The CT scan was done with intravenous contrast and the pulmonary embolism protocol. This technique is similar to the standard CT scan of the thorax; however, the rate of injection of the contrast medium is 2.5 mL/s (instead of the usual 2.0 mL/s) for a total volume of 100 mL. Image acquisition began approximately 15 to 20 seconds after the injection had started. In addition, initial 3-mm axial reconstructions were obtained through the pulmonary vasculature, followed by 8-mm axial reconstructions throughout the entire chest. A dissection flap was noted that involved the ascending aorta, the aortic arch, and the descending thoracic aorta (Figures 1 and 2). Contrast was noted in both true and false lumens. Note was also made of a small linear flap involving the origin of the left subclavian artery that was consistent with an extension into the origin of this vessel. There was a pericardial effusion of

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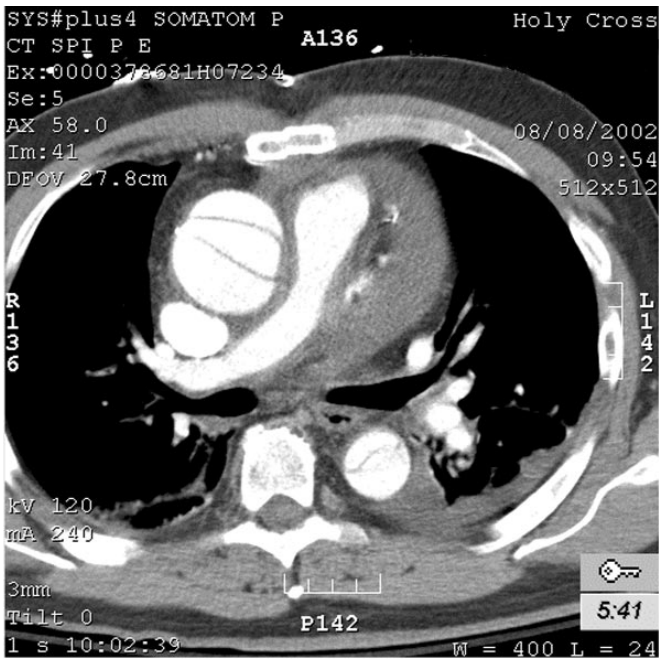

Figure 1. Type A aortic dissection extending to the entire thoracic aorta in case no. 1.

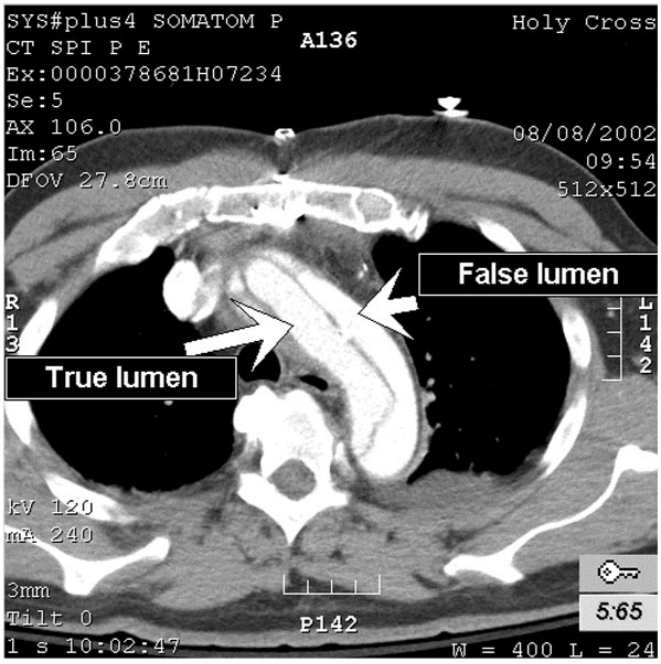

Figure 2. Aortic arch involvement in case no. 1.

significant size. These findings were diagnostic of Stanford type A or DeBakey type I acute AOD.

The patient underwent immediate reexploration when a significant amount of fresh clots was found in the pericardium. The aorta was the site of an extensive subadventitial hematoma originating near the proximal anastomosis of the vein graft to the posterior descending coronary artery (PDA). This graft was thrombosed, and all other grafts were patent. There was no active bleeding from any suture line. With the patient under profound hypothermic circulatory arrest, an exploration of the aortic intima confirmed that the dissection originated at the proximal anastomosis of the vein graft to the PDA. There were no intimal tears in the aorta proper. An ellipse of aortic wall measuring 4 ± 2.5 cm and encompassing the orifices of the 2 vein grafts was excised. After application of BioGlue (CryoLife, Kennesaw, GA, USA) between the surrounding dissected layers of aorta, the defect was replaced with an ellipse of Hemashield graft material (Meadox, Oakland, NJ, USA). The vein graft to the PDA was disconnected distally from the coronary artery, declotted thoroughly, and reanastomosed to the PDA. Both vein grafts were then reattached to the Hemashield graft (Figure 3). Patient recovery was uneventful. Repeat CT scanning at 6 months indicated a persistent double lumen in the descending thoracic aorta without significant encroachment on the true lumen. The aortic diameter remains normal. Hypertension has been well controlled with medical therapy.

Case 2

C.L., an 88-year-old woman, presented with a history of longstanding treated hypertension, recurrent episodes of angina at rest, and shortness of breath on exertion. In addition to extensive triple-vessel disease, the patient was found to have hypertrophic nonobstructive cardiomyopathy and an ejection fraction of 60%. On July 12, 2002, she underwent on-pump coronary bypass (ONCAB) surgery consisting of sequential SVGs to the LAD, the PDA, and the first and second marginal branches of the circumflex coronary artery. Two proximal anastomoses were done with the Symmetry automatic device (St. Jude Medical, St. Paul, MN, USA) because of the finding of significant diffuse calcification along the entire length of the ascending aorta. The third vein was anastomosed in a piggyback fashion to the hood of one of these veins. The patient was discharged on July 18 in good condition and on atenolol therapy. She returned to her cardiologist a week later complaining of increased shortness of breath and edema of the lower extremities. TTE results

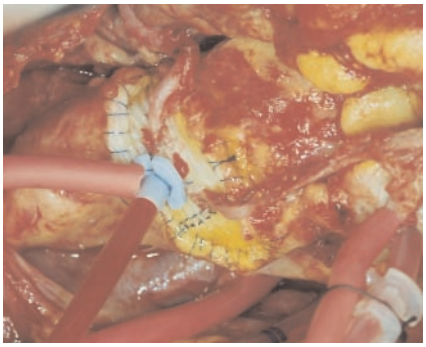

Figure 3. After repair with elliptical graft and reimplantation of vein grafts.

Table 1. Iatrogenic Intraoperative Aortic Dissection

- Side-biting aortic clamp
- Aortic cross-clamp
- Femoral or aortic cannulation site
- Cardioplegia needle site
- Aortotomy site
- Proximal venous anastomotic site (direct or using automatic devices)

showed a moderate pericardial effusion, an ejection fraction greater than 60%, concentric left ventricular hypertrophy with nonobstructive hypertrophic cardiomyopathy, and moderately severe aortic insufficiency that was not present preoperatively. A CT scan of the chest showed a typical appearance of dissection involving the ascending aorta. The flap extended from the aortic root to the top of the ascending aorta but did not involve the transverse portion of the arch. There was also no evidence of extension in the origins of the great vessels. Contrast was noted in both true and false lumens. The descending thoracic aorta was not involved with the dissection. The appearance was typical of a Stanford type A or a DeBakey type II acute AOD. Surgery was recommended, but the patient declined. She died suddenly at home a few days later, presumably from a ruptured aorta.

Case 3

S.B., an 85-year-old woman with a history of longstanding treated hypertension, severe aortic stenosis, and mitral regurgitation, underwent aortic valve replacement, mitral valve repair with ring, and coronary bypass with SVGs to the LAD and the right coronary artery on October 15, 2002. After a prolonged and complicated recovery, the patient was transferred to a rehabilitation unit where she continued to manifest uncontrolled hypertension. Although a nuclear renal scan suggested renal artery stenosis, selective angiography findings did not confirm this suspicion. A recent finding of hoarseness and paralysis of the right vocal cord and diaphragm with worsening shortness of breath led to a CT scan of the chest. We were surprised to find that the scan demonstrated a Stanford type A or DeBakey type II acute AOD. The dissection flap was noted as a curvilinear line within the ascending aorta. Contrast was noted in both true and false lumens. The dissection did not extend into the arch, nor did it involve the origins of the great vessels or the descending thoracic aorta. Because of the patient's poor overall condition, we elected to treat her conservatively. The patient died suddenly 9 weeks after her original surgery, presumably from a ruptured aorta.

Case 4

E.M., a 77-year-old woman with hypertension, underwent OPCAB surgery on August 18, 2001, consisting of a sequential grafting of a free right internal thoracic artery to the LAD and diagonal branch and an SVG to the PDA. Blood pressure control during the application of the partially occluding aortic clamp for the performance of the proximal anastomoses was erratic with extremes of 240 mm Hg and 40 mm Hg. However, the procedure was completed, and the patient returned to the intensive care unit without any evident deleterious complication of this problem. Four days later, the patient was started on oral anticoagulants for treatment of atrial fibrillation. Within 24 hours, she became extremely short of breath and hypotensive, and a chest radiograph indicated whitening of the right chest cavity, an observation compatible with the presence of a large hemothorax. TTE results confirmed good left ventricular function, the absence of a hemopericardium, and the presence of a large pleural hematoma on the right side. The patient underwent thoracoscopic drainage of the hemothorax, at which time all grafts and anastomoses were explored and appeared to be intact. On September 2, 2001, the patient again became suddenly hypotensive and short of breath, and a chest radiograph showed the recurrence of a large right hemothorax. Results of transesophageal echocardiography (TEE) performed in the operating room confirmed an acute Stanford type A or DeBakey type II dissection originating at the anastomosis of the SVG of the PDA to the aorta. Repair consisted of tube graft replacement of the ascending aorta with reimplantation of the grafts under profound hypothermic circulatory arrest. Although the patient survived the surgery initially, she died a few days later with multiple systems failure.

DISCUSSION

AOD is known to occur spontaneously in patients with chronic hypertension, Marfan syndrome, and other collagen diseases. It has been induced by the abuse of certain drugs (cocaine, methylenedioxymethamphetamine). It has been known to occur late (up to several years) after uneventful cardiac surgery (Stranger 2002). One of its most challenging variants, however, is the iatrogenic AOD occurring during surgery (Still 1992) as a result of aortic injury (Table 1). It is then easily recognized and can be addressed immediately with a variety of surgical techniques (Blakeman 1988). Iatrogenic AOD occurring during cardiac catheterization (Carter 1994, Dunning 2000, Yip 2001) is also recognized immediately, although its management is not necessarily as straightforward. Management is observation if the AOD is limited and surgery if the AOD is extensive, is progressive, or results in the closure of a coronary artery that cannot be reversed by stenting.

More subtle in its symptomatology and more difficult to recognize is acute AOD occurring early after cardiac surgery (Lam 1977, Gillinov 1999). Unrecognized, such AOD may result in unexplained postoperative death (in the hospital or at home), which is commonly attributed to "massive pulmonary embolism" in the absence of autopsy. In our institution (600 cardiac surgeries per year), an average of 2 to 3 sudden deaths occur every year after cardiac surgery, usually involving coronary revascularization and occurring with both ONCAB and OPCAB. Because of the serious medicolegal situation in Florida, autopsies are usually not performed, and most deaths are classified under the massive pulmonary embolism category. The real cause of death remains unknown (Table 2). The surgeries for the 4 cases presented here were performed by 4 different senior surgeons. Two surgeries were OPCAB, and 2 were

Table 2. Sudden Death after "Uneventful" Cardiac Surgery

- Acute myocardial infarction (graft occlusion, coronary embolism)
- Cardiac tamponade (removal of temporary pacing wires, continued post operative bleeding, ruptured type A aortic dissection)
- Severe hypoxemia (unrecognized patent foramen ovale or pneumothorax)
- Ventricular arrhythmias
- Acute anemia (ruptured ventricle or aortic aneurysm)
- Massive pulmonary embolism

ONCAB (one with concomitant valve surgery). The correct diagnosis was missed by TTE in all cases and was confirmed preoperatively only by a CT scan of the chest. Although eventually responsible for 2 delayed deaths after patient discharge from the hospital (cases 2 and 3), cardiac tamponade usually was not sudden, thus allowing some time for the clinician to reach the diagnosis. The earliest symptoms were not dissimilar to those occurring in "medical" AOD, namely, chest pain, paraparesis of the lower extremities, and shortness of breath. Physical findings were misleading except for unexplained hypotension and a new murmur of aortic insufficiency in one patient. TTE results were nonspecific in that they confirmed the presence of a pericardial or pleural hematoma, a not so unusual finding after cardiac surgery, but indicated no evidence of pericardial tamponade. CT scanning of the chest was in our experience the key diagnostic tool in establishing the correct diagnosis (Archer 1986). In most institutions, this study can be completed in less than 15 minutes at any time of the day, whereas TEE (Katz 1993) may not be available or feasible on the ward for an uncomfortable or unstable patient. Once the diagnosis of AOD is confirmed, surgery needs to be performed expeditiously, depending on the findings, and consists of tube graft or patch graft replacement of the aorta after gluing the dissected layers together with BioGlue and then reimplanting the vein conduits on the graft.

The pathophysiology of early postoperative AOD is only conjectural, although it may be quite similar to that occurring intraoperatively. Recent attention, however, has been placed on the role of the proximal vein-aorta anastomosis in the initiation of the dissection, particularly when automatic anastomotic devices are used for its performance (Endo 2002). Such anastomoses have been described as having been blown off or pulled off the aorta. These reports are still sketchy, and, as Dr. Robert Frater commented, "by and large you are going to find that anything that has happened in off-pump CABG surgery has already happened many times in cardiac surgery in general as well as CABG using extracorporeal circulation" [Frater 2002]. Because AOD has also occurred after conventional anastomoses, including those described in this report, it is difficult at this time to blame the automatic connector system for this complication.

More plausible is the role of the aortic clamp applied during the performance of the proximal anastomoses. Although not proven statistically, it is conceivable that there may be a higher incidence of early AOD in OPCAB (Chavanon 2001) than in ONCAB, presumably due to the application of the partially occluding aortic clamp with pulsatile aortic flow. This theory has prompted the recommendation to use padded or soft-jaw clamps and systolic pressures not higher than 75 mm Hg during the performance of the proximal anastomosis in OPCAB, whereas a single continuous cross-clamp period is recommended in ONCAB. Clearly, in situ arterial grafts that do not necessitate the creation of an arterial inflow obviate clamping the aorta and should reduce the incidence of this complication.

CONCLUSION

Despite being the most common and probably one of the most studied and standardized procedures in surgery, coronary revascularization continues to be plagued by a number of complications occurring during or even after a seemingly uneventful operation. Technical problems that are obvious during the performance of the surgery are usually easier to handle than sudden postoperative events occurring in the stepdown units. Some of these problems, such as cardiac tamponade after removal of temporary pacing wires in anticoagulated patients, are preventable. Most of these problems, however, are not preventable but could be addressed appropriately if recognized early. One such entity is early postoperative acute AOD, which, as described in this report, may not be that uncommon and may account for more disasters and deaths than are acknowledged in the literature. Its diagnosis is made expeditiously if AOD is suspected and a CT scan of the chest is done. Its prevention may be based on avoiding the manipulation of the ascending aorta and a tighter control of hypertension in the immediate postoperative period. The treatment of this AOD is not very difficult and is within the reach of every trained cardiac surgeon.

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