to minimize the risk of migration, and the endoleak was induced by aneurysmal configuration.

In conclusion, a two-staged combined surgical and endovascular approach with the use of a new multi-branched prosthesis may be effectively and safely used in management of high-risk extensive thoracic and suprarenal abdominal aortic aneurysm.

References


Impella Used for Hemostasis by Left Ventricular Unloading, in a Case With Left Ventricular Posterior Wall Rupture

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Left ventricle wall rupture is a feared complication in mitral valve surgery. We report a combined mitral valve anuloplasty and coronary artery bypass grafting procedure with severe, life-threatening bleeding complication due to left ventricular posterior wall rupture. The patient was successfully treated with a temporary left ventricular assist device to decompress the left ventricle in an attempt to minimize the bleeding, as the patient’s condition did not allow standard repair of the left ventricle.


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The Impella Recover (Impella CardioSystems AG, Aachen, Germany) ventricular assist device is a miniaturized blood pump for unloading of the left or right ventricle, or both. The pump incorporates a rotor driven by an electromagnetic motor, a driving console, and an inflow cannula. The console allows management of the pump speed in nine gradations and displays the pressure gradient between the left ventricle (LV) and the ascending aorta. An inflow cannula is inserted transvascularly into the LV. The Impella Recover (Impella CardioSystems AG) provides a continuous flow of 4.0 to 4.5 L/min. It aspirates blood from the LV and expels it in the ascending aorta. The Impella has been successfully used in settings of postcardiotomy heart failure [1] and cardiogenic shock [2].

We believe that this is the first report describing the use of the Impella left ventricular assist device (LVAD) in an attempt to unload the LV in a patient with a left ventricular posterior wall rupture, but without postcardiotomy shock.

A 68-year-old man with diabetes mellitus and chronic obstructive pulmonary disease with a posterior wall myocardial infarction 18 years ago had dyspnea develop in the last year. During hospitalization due to a subendocardial myocardial infarction, three-vessel disease was diagnosed. Furthermore, a mitral insufficiency and pulmonary artery hypertension were found on echocardiography. The left atrium was enlarged, the mitral annulus was dilatated, and the posterior wall of the left ventricle was akinetic. The magnetic resonance imaging showed calcifications in the posterior wall of the left ventricle.

The patient was scheduled for surgery. After division of pericardial adhesions, a standard cannulation of the aorta and both cavas was performed. Myocardial revascularization was accomplished with a saphenous vein graft to the third marginal branch and jump Anastomoses to the second and first marginal branches and then to the first diagonal branch. The left internal mammary artery was anastomosed to the left descending coronary artery before opening the left atrium. A slightly undersized Carpentier-Edwards Physio ring (Edwards Laboratories, Irvine, CA) was used for the mitral anuloplasty. During reperfusion, a small, almost punctual bleeding from the myocardium (2 cm away from a marginal branch anastomosis) was noticed. A patched suture was placed over the bleeding, which at that moment was believed to be an instrumental injury. After weaning from cardiopulmonary bypass, the bleeding restarted and further patched sutures were placed. The area was covered with Floseal (Fusion Medical Technologies Inc, Mountain View, CA), and a Tachosil patch (Nycomed, Roskilde, Denmark) was placed before closure. The patient was reoperated on three times for tamponade or bleeding during the first 24 hours, but as soon as the patient’s blood pressure rose, the bleeding restarted. Then we realized that the bleeding was due to a left ventricular posterior wall rupture. At
this time we considered that the patient’s condition did not allow for a re-atriotomy. Despite absence of postcardiotomy shock, we decided to unload the left ventricle with an Impella. After a test run outside the patient, the Impella was introduced into the left ventricle through a Hemashield graft (Boston Scientific Corp, Natick, MA) sutured to the ascending aorta under echocardiographic control. The bleeding from the posterior wall ceased and the hemodynamics were stable. The chest was left open and 24 hours later the chest was bandaged with a vacuum-assisted closure for maximal protection against bacterial contamination. Antibiotic prophylaxis was administered according to normal postoperative routine; initially Cloxacinil (Astra Zeneca, Södertälje, Sweden) was used and then Levofloxacine (Aventis Pharma, Helsinki, Finland) at the reoperations. After the implantation, the Impella was run at full speed during the whole treatment period. No systemic anticoagulation was used, except for heparin sulphate, which was used for lubrication of the device. Activated thromboplastin time was at a level of 35 to 40 seconds.

The central circulation was monitored with a Swan-Ganz catheter with continuous measurements of cardiac output and mixed venous saturation (SvO₂) (Edwards Lifesciences, Irvine, CA). The left atrial pressure was measured as the true filling pressure of the LV. Intermittent echocardiographic examinations of the pump position and the unloading of the LV were undertaken. Pharmacological treatment with milrinone and adrenaline [3–4] was used to achieve the hemodynamic goals; left atrial pressure was as low as possible at 26, 11, and 26, before, during, and after the LVAD treatment, respectively; mean arterial blood pressure was 60 to 80 mm Hg; mixed venous saturation (SvO₂) was greater than 60%; cardiac index was greater than 3 L/min/kg; and hourly diuresis was greater than 2 mL/kg. The systemic vascular resistance was in the range of 540 to 740 dynes/cm². Noradrenaline or phenylephrine was administered through a left atrial catheter to maintain an adequate mean arterial blood pressure [5]. These levels were accepted as long as the shed mediastinal blood losses were within acceptable ranges. On postoperative day 4, the patient could successfully be weaned from the LVAD during careful monitoring of the bleeding as well as invasive and noninvasive hemodynamic measurements. After LVAD removal and chest closure, the blood losses were within acceptable ranges and the chest tubes could be removed after an additional 2 days. The vasoactive drugs could be withdrawn 9 days postoperatively.

The patient required massive transfusions of erythrocytes and plasma as well as maximal pharmacological therapy in attempts to stop the bleeding, mainly during the period prior to implantation of the Impella. Due to several periods of hypovolemic shock with resuscitation, the low mean arterial blood pressure during LVAD treatment and massive transfusions leading to an extreme over-hydration with a cumulative fluid balance that was 21 L on day 1 and 19 L on day 7, multiple organ failure developed in the patient. Due to the transient renal insufficiency, the patient had intermittent hemodialysis.

Pneumonia with Stenotrophomonas maltophilia and a postoperative cholecystitis developed in the patient. The patient was treated with antibiotics and drainage of the gallbladder. However, he did not develop any wound infection. The patient was treated for 13 days in the intensive care unit and was thereafter discharged to the intensive care unit at the referring hospital. He was rehabilitated, discharged home, and at his 6-month follow-up he reported a full recovery without sequel.

Comment

In this case, the true origin of bleeding was not recognized either during the primary operation or during the first two reoperations. This was attributed to the fact that the first bleeding spot was situated no less than 6 cm from the atrioventricular groove. Hence, the standard procedure for repair of the posterior wall rupture was not applicable. Prolonged bleeding and periods of pericardial tamponade made the situation desolate and the improvisation with left ventricular unloading had an immediate and dramatic effect on the bleeding, which terminated momentarily.

The aim of the LVAD and pharmacological therapy was to keep the LV pressure as low as possible. In combination with the echocardiographic findings, we believe that the left atrial pressure and systemic vascular resistance indicated a relative proper unloading effect of the left ventricle. One can argue that the left atrial pressure levels were in the high range during the pump treatment, but this can be partially explained by the decreased compliance due to the surgical trauma and the edema in the myocardium. Due to the massive transfusions, we expected increasing levels of the pulmonary pressures and vascular resistance with impairment of the right ventricular function. However, the monitoring of the global circulation indicated a proper right ventricular function, as well as the blood flow from the LVAD.

The Impella system is relatively noninvasive compared with other LVADs for temporary use; it unloads the failing ventricle satisfactorily and generates an adequate blood flow to the systemic circulation.

Left ventricular unloading may be considered in selected cases of left ventricular posterior wall rupture if the patient’s condition disqualifies him for standard treatment with endocardial patching of the rupture.

References


Chronic Expanding Hematoma in Sternum Resected 5 Years After Median Sternotomy

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Chronic expanding hematomas occur at various locations in the body; however, their occurrence in the sternum has not been reported yet. We report a patient with chronic expanding hematoma in the sternum 5 years after undergoing a median sternotomy for cardiac surgery. Although preoperative biopsy specimens did not lead to a definitive diagnosis, we could not rule out the possibility of a malignant tumor because of the expanding and infiltrative behavior of the hematoma. We performed a sternectomy and reconstructed the chest wall using artificial materials.


Chronic expanding hematomas (CEHs), initially identified by Reid and Kommareddi [1], occur at various locations in the body. Their occurrence has been reported in thoracic cavities [2–4]. However, CEHs in the sternum have not been reported yet. It is difficult to differentiate CEHs from malignant tumors because CEHs sometimes behave in an invasive manner similar to malignant tumors. Furthermore, malignant tumors occasionally contain a large amount of blood. We describe a patient with CEH in the sternum that was resected 5 years after he underwent a median sternotomy.

A 79-year-old man who had undergone a median sternotomy for cardiac surgery, aorta replacement for aortic arch aneurysm, 5 years ago was followed up. He took some medications for hypertension and hepatitis C, but did not take anticoagulants during the follow-up period. He felt something unusual in the precordial region, which was determined to be an erythematous mass surrounded by redness (Fig 1). Computed tomography (CT) of the chest showed a round mass shadow obliterating the sternum (Fig 2). The mass in the current CT was larger than that obtained in the CT 1 year ago, and had extended into the surrounding sternal bone. Although the sternal wires used during the previous cardiac surgery obstructed the view, chest magnetic resonance imaging showed that the mass was filled with fluid. To differentiate between a CEH and a malignant tumor, we performed a needle biopsy of the mass. Cytological examination revealed only the blood component with no evidence of malignant or inflammatory cells. Blood chemistry analysis showed mild anemia and a mild hepatic dysfunction. We could not reach a definite diagnosis and completely ruled out malignancy. Furthermore, there was a synthetic graft under the expanding mass. Therefore, we believed it was appropriate to surgically remove the mass.

The skin was incised at a sufficient distance from the mass. The sternal bone was cut from the manubrium at the sternum angle, and the second to sixth ribs were amputated bilaterally at the costochondral junctions. We reconstructed the chest wall with a Gore-Tex sheet (W. L. Gore & Assoc, Flagstaff, AZ) and Bard mesh (C.R. Bard Inc, Murray Hill, NJ), we placed two Kirschner wires (Mizuho Co Ltd, Tokyo, Japan) above the sheets, and we hardened them with cranioplastc cement (Johnson & Johnson K.K., Tokyo, Japan) to form a structure similar to the sternal bone (Fig 3). The operation lasted 247 minutes, and the total blood loss was 640 mL.

Macroscopic observation showed that the mass was hollow and filled with old blood. Pathologic examination revealed the presence of dense fibrous tissues integrated with bone beams surrounding the hematoma, hemosiderin deposits, lymphocyte infiltration, fibroblast proliferation, and foreign-body granulomas, including foreign-body giant cells. There was no evidence of malignancy.

Although the chest had to be drained for approximately 2 weeks postoperatively, the patient was discharged 48 days after surgery without evidence of a flail chest. At the 1-year follow-up, there was no sign of recurrence.

Comment

Although CEHs have been reported, the underlying mechanism for their formation is still unclear. One possibility is that cellular breakdown products of leukocytes, erythrocytes, hemoglobin, platelets, and fibrin inside the clot cause a fibroblastic reaction. Continued inflammation increases the permeability of the vascular wall resulting in the bleeding of the damaged capillaries beneath the fibrous capsule [5].

It is very difficult to differentiate CEHs from malignant tumors. The medical history of the patient is important, because most cases with CEH have a history of trauma or surgery. In our case, there had been a previous surgery in the sternal region; therefore, we should have thought

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