

Penetrating Injury to the Proximal Descending Aorta: Can we do Better in the Endovascular Era?

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INTRODUCTION

Penetrating thoracic aortic injuries are very rare, comprising only 1% of all thoracic vascular injuries and 13% of penetrating injuries to the thoracic aorta [1,2]. These injuries are usually fatal on the scene and have a very high mortality rate, 55%, even among the few who are alive when they arrive at the emergency room (ER) [3]. Most patients who reach the ER are in shock due to intrathoracic hemorrhage and as such should be transferred immediately to the operating room (OR). However, most trauma patients around the world are treated in hospitals which lack cardiac and vascular surgeons who are familiar with this complicated area of the aorta. Endovascular treatment, either as temporary bleeding control or as a definitive measure, seems a promising option. We hereby report a case of a patient with several stab wounds, to the thoracic inlet, with

proximal descending aortic injury. The clinical course and therapeutic dilemmas are discussed.

Case Description

A 17-year old, previously healthy, male was found by Magen David Adom technicians (Israeli Emergency Medical Technicians) on scene lying in a puddle of blood. Three stab wounds to the left chest cavity were noted – one in the thoracic inlet, above the left clavicle, one below the left nipple, 4th intercostal space, and the third on the posterior axillary line on the 9th intercostal space. Needle decompression was performed on both sides of the chest, and the patient was transferred to the ER of a nearby level 2 trauma center.

On arrival to the trauma bay, the patient was intubated and ventilated. His blood pressure and pulse oximetry were not measurable and only filiform rapid carotid pulse was palpated. Breath, as well as heart sounds, were normal. On physical examination, three stab wounds to the left thoracic inlet and bilateral needle thoracostomy were noted. Chest drains were inserted simultaneously to both sides of the chest, without any evidence of pneumothorax or hemothorax on any side. Focused assessment sonography for trauma (FAST) did not reveal any sign of pericardial or peritoneal fluid. Two units of O-positive packed cells (PC) and one liter of crystalloids were immediately administered and the patient's blood pressure was raised to 90/60. Suddenly a massive arterial bleeding arising from the left thoracic inlet wound was noticed. The bleeding was controlled by direct digital pressure and the patient was transferred to the OR, 35 minutes after his admission.

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Figure 1 Extravasation from the proximal descending aorta, distal to the LSCA orifice (LSCA - Lt. Subclavian artery), despite manual compression. The big white arrow shows extravasation, black arrows show the tract of bleeding, and the small white arrow shows fingers compression.

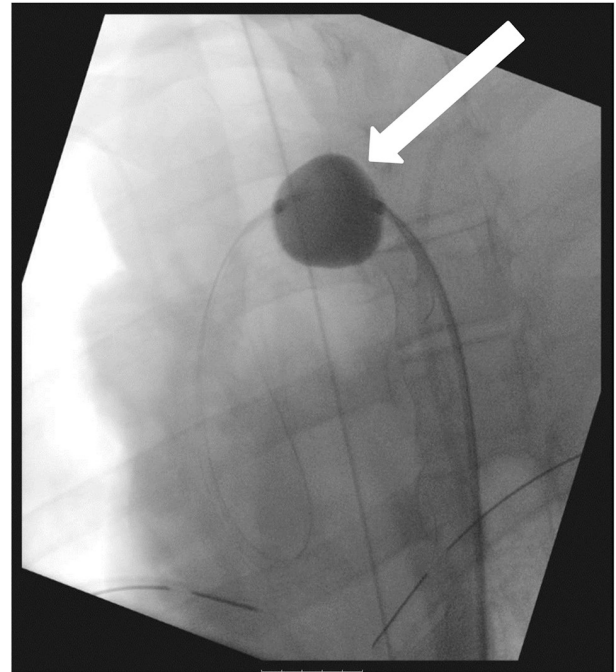


Figure 2 Occluding balloon located in the distal aortic arch. The white arrow shows the inflated balloon.

On arrival to the OR, the patient's blood pressure was 120/74 mmHg. Vasopressors were not used at this stage. On-table angiography was performed, via the right common femoral artery. Extravasation originating a few millimeters distal to the left subclavian artery (LSCA) orifice was noted. No bleeding was noticed from the LSCA or the left common carotid artery (Figure 1). The vascular surgeon's decision was to treat the lesion by using a stent graft (SG). The SG, which was unavailable on the shelf, was ordered with an estimated arrival time of one hour. However, the patient's condition deteriorated gradually despite continuous administration of blood products. At that time, the patient received eight units of PC, eight units of fresh frozen plasma and eight units of platelets. A decision to treat the patient by open surgical repair was taken due to a steep hemodynamic deterioration. The decision for open surgical repair was made and a 12 French intra-aortic balloon (Reliant Balloon Catheter to Expand Vascular Prosthesis, Medtronic, USA) was inserted via the left femoral artery and inflated proximal to the bleeding site in the aortic arch as pre-incision preparation for proximal control (see Figure 2). This procedure only required femoral sheath changes from 6 French to 12 French and the balloon insertion, which took a few minutes. Then an emergency sternotomy with a left "trap-door" extension was performed. On entering the chest, a large mediastinal hematoma was seen. There was no blood in the pleural or pericardial cavities. While trying to get proximal control, the hematoma ruptured and the patient expired almost immediately.

DISCUSSION

Hillel Yaffe Medical Center is a regional level 2 trauma center, serving a catchment area of approximately 600,000 inhabitants. The trauma unit, led by a certified trauma surgeon, admits approximately 180 major trauma cases (ISS 16 and above) annually. Our medical center lacks cardiothoracic surgeons as well as related devices, such as a heart-lung machine.

Thoracic aortic injury is usually fatal, with most patients dying at the scene. The few who survive long enough to reach the hospital are kept alive by contained hematoma, which prevents massive uncontrollable bleeding to the pleural cavity with immediate death ensuing. According to the accepted treatment protocols, most trauma surgeons would consider ER thoracotomy as the initial step of dealing with a patient presenting in extremis or without vital signs in the setting of penetrating thoracic trauma such as in this case. The decision of the very experienced trauma surgeon, who managed this case, not to follow the strict rules and not to perform ER thoracotomy was because the thorax did not drain blood. This fact led to the assumption that the cause of the hemodynamic instability was secondary to mediastinal hematoma. In such a scenario, opening the chest would most probably lead to relief of the tamponade effect and secondary rupture of the contained hematoma. This will invariably lead to the patient's death. Even if this is not the case, putting a clamp on the descending aorta will increase the afterload and as such will increase the risk of secondary hematoma rupture, which once again would lead to the same conclusion.

The option of treating the aortic injury directly would not have been the smartest thing to do without proximal or distal control. In order to gain proximal and distal control, the best option would be a clamshell thoracotomy which will almost invariably lead to the rupture of the hematoma and the patient's death. It seems that if the patient is likely to survive, it would be with a minimal procedure that will lead to the best consequences, which in the current era is the endovascular option.

Inserting an occlusion balloon as a temporary measure seems to be the most logical thing to do in this scenario. This is a common practice undertaken by vascular surgeons dealing with ruptured abdominal aortic aneurysms. Therefore, if it works for them why should it not work in this case? Furthermore, there was no indication to inflate the balloon on this occasion as the patient was stable, and it was left there for safety if needed until the SG arrived. In the pre-endovascular era, the only therapeutic option was open surgical repair. Such cases require very high surgical competence, of extremely experienced cardiac and vascular surgeons, and in many cases, mainly when the proximal aorta is involved, the application of a heart–lung machine, and probably temporary or permanent cardiopulmonary bypass [4]. Worldwide, most patients with aortic injury reach hospitals with no such facilities. The evolution of endovascular surgery has made a dramatic change in patients' prognosis. Endovascular repair of thoracic aortic injuries is entirely different than opening the patient's chest in order to repair injuries. Endovascular treatment allows an experienced team of trauma and vascular surgeons in cooperation with an interventional radiologist to deal with injuries which were once only dealt with by cardiothoracic surgeons.

One should be familiar with hemodynamics and not fall into the trap of figures. A patient with a consistent systolic blood pressure of 70 mmHg is someone that the treating physician should try to keep as such and not attempt to normalize the figures. In our opinion, the chance of the patient's survival, while waiting for an SG to be delivered, are much higher in such a scenario of controlled hypotension than when the patient is given vasopressors and blood in order to see appealing figures on the screen. Some studies indicate that the patients who are most likely to benefit from hypotensive resuscitation are those in hemorrhagic shock caused by uncontrolled sources of bleeding [5,6]. Particularly, in the case of large vessel penetration injury, this strategy may prevent rupture of a contained hematoma which is the only thing keeping the patient alive. Using this strategy requires strict monitoring and assessment of end organ perfusion. However, Carrick et al. in a randomized study on 168 trauma patients treated by two resuscitation strategies, did not find significant differences in acute myocardial infarction and stroke rates, as well as in incidence of acute renal failure [7].

Temporary measures such as balloon occlusion of the tear, even using two balloons, proximally and distally to

the tear, should be kept in mind as a bailout procedure when necessary. If the balloon can occlude the root of the LSCA (Zone "0" occlusion), this maneuver should be used for hemorrhage control. We may only assume why the balloon occlusion did not work in this particular case. There are several potential reasons. First, the balloon probably migrated downstream due to aortic pressure and as a result could not occlude the LSCA. Second, even if the balloon was properly located, the aortic arch is not a flexible area and, therefore, the chances of occlusion are reduced.

In our case, the resuscitative endovascular balloon occlusion of the aorta (REBOA) site confirmation during the operation was based on no inflow; just an enormous amount of backflow which led to the patient's death almost immediately after the hematoma was entered. This lesson was learned and we are currently using a double balloon technique, both proximal and distal to the injury site.

Our case demonstrated a new possibility in dealing with an aortic injury that was previously considered almost unsalvageable. There is no question that open surgery remains the treatment of choice. However, development of an endovascular trauma management approach opens new treatment horizons, such as temporary balloon occlusion and the use of endovascular SG. The balloon occlusion, which we believe should be based on the use of two occlusion balloons, is the preliminary step of hemorrhage control until an SG is available for definitive treatment. Even for those scheduled for surgery, using balloons may be very helpful as a temporary control measure.

We believe that future research will confirm the "viability" of our pioneer approach.

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