A ruptured thoracic aorta is a rare but life-threatening condition requiring immediate intervention. Possible causes of a ruptured thoracic aorta are ruptured descending thoracic aneurysms, acute type B aortic dissections, penetrating aortic ulcers, and traumatic thoracic aortic injuries (TTAI). Thoracic endovascular aortic repair (TEVAR) offers an effective treatment for these acute thoracic aortic pathologies, and the endovascular approach appears to reduce the morbidity and mortality rates compared with traditional open surgery. At many institutions, TEVAR has become the preferred treatment for acute thoracic aortic disease.

However, endovascular repair of these acute aortic pathologies is still associated with the occurrence of endoleak in 5% to 30% of cases. The majority of these endoleaks are proximal type I endoleaks, and urgent reintervention is often needed. Adequate endograft sizing using preoperative computed tomography angiography (CTA) is thought to be important in minimizing risks of endoleak and other endograft-related complications and for improving long-term outcomes after endovascular repair.

Patients with thoracic aortic rupture are often admitted with considerable blood loss or hypovolemic shock. Although peripheral vasoconstriction is a well-known response to hypovolemic shock to maintain perfusion of the heart and brain, the effects of hypovolemia on the aortic dimensions remain unclear. Temporary changes in the aortic diameter during blood loss could lead to incorrect aortic measurements on preoperative CTA, inadequate endograft sizing, and increased risks of endograft-related complications after TEVAR for thoracic aortic emergencies.

Effects of Hypovolemia on Aortic Dimensions

We performed several research projects to study the effects of hypovolemia on the aortic dimensions and the potential implications for the endovascular management of thoracic aortic rupture. In the first project, we retrospectively investigated aortic changes in trauma patients who were admitted with hemodynamic instability (mean arterial pressure [MAP] < 95 mm Hg and a heart rate > 100 bpm). All trauma patients with these vital signs who had undergone a CT of the thorax and abdomen both at admission and at another time (control CT scan) were included for analysis. All CT examinations were blinded, and differences in aortic diameter between the initial CT scans obtained in the trauma bay and the control CTs were compared. This comparison revealed that these trauma patients had significantly smaller aortic diameters at...
admission when they were hemodynamically unstable, compared with the control CT scans. The decrease in aortic diameter was observed at all evaluated levels, and the inferior vena cava was also significantly smaller at admission compared with the control CT. A subanalysis of the patients who had more severe hemodynamic instability (a heart rate ≥ 130 bpm) showed that the mean difference in aortic diameter between the two different scans was even larger. At the level of the mid-descending thoracic aorta and the abdominal aorta, the aortic diameter was on average 13% smaller at admission when hemodynamically unstable compared with the control CT scans (Figure 1). Our hypothesis for these remarkable findings was that the decreased pressure on the aortic wall due to hypovolemic shock and endogenous production of vasoconstrictors may have caused the decrease in aortic diameter in these trauma patients.

Because this retrospective study had several limitations, we also performed several pig experiments to further investigate the exact effects of blood loss on the aortic dimensions. The circulating blood volume of seven Yorkshire pigs was gradually lowered in 10% increments. At 40% blood loss, an endograft was deployed in the descending thoracic aorta followed by gradual fluid resuscitation. Potential changes in aortic diameter during the experiment were recorded using intravascular ultrasound (IVUS). We observed that during blood loss, the aortic diameter gradually decreased in all subjects at the level of the ascending, descending, and abdominal aorta (Figure 2). The ascending aortic diameter decreased on average by 38% after 40% blood loss (range, 24%–62%), the descending thoracic aorta by 32% (range, 18%–52%), and the abdominal aorta by 28% (range, 15%–39%). Linear regressions analysis showed that the degree of blood loss was highly correlated with the descending thoracic aortic diameter. There was also a strong correlation between the blood pressure during the experiment and the aortic diameter in the Yorkshire pig, whereas the heart rate was less reliable for predicting the aortic diameter. Once the animals received fluid resuscitation, the aortic diameters quickly regained their initial size. Two of the seven Yorkshire pigs developed endoleaks during fluid resuscitation, whereas no abnormalities were observed after initial endograft deployment at 40% blood loss (Figure 3).

Theoretically, these aortic changes could take place in all patients with considerable blood loss, but we expect that these changes will be most extreme in young TTAI patients. Because aortic compliance decreases with age due to a loss of elasticity and increased pra-
In the presence of aortic calcification, we believe that aortic changes may be less dramatic in patients with ruptured aortic aneurysms who are typically much older than the average trauma patient. The aortic changes during blood loss and fluid resuscitation may have implications for the endovascular management of thoracic aortic rupture.

**Implications for TEVAR of Thoracic Aortic Rupture**

Endograft sizing for TEVAR is usually performed using the preoperative CTA scan; however, if the diameter of the descending thoracic aorta is considerably smaller at admission due to blood loss, physicians may undersize the endograft. This mismatch between the aortic diameter and the endograft could theoretically result in increased risks of endoleak or other endograft-related complications such as endograft migration after TEVAR.

Physicians performing TEVAR should be aware that changes in aortic diameter due to hypovolemia could take place, and there may be two options to adjust for this phenomenon.

First, because the actual aortic diameter measurements may be larger than observed on preoperative CTA, physicians could consider slightly increasing the percentage of oversizing for the endograft (Figure 4). Because of the many variables that may affect the aortic dimensions, it is difficult to provide a recommendation with regard to the exact percentage of oversizing needed in hypovolemic patients. Based on the finding that the mid-descending thoracic aorta of trauma patients with a pulse > 130 bpm and a MAP < 95 mm Hg was on average 13% smaller on the CT at admission, physicians could consider applying approximately 20% to 30% oversizing in TTAI patients with similar vital signs, instead of the standard 10% to 20% oversizing. Caution is needed when increasing the percentage of oversizing during TEVAR because some reports suggest that this may increase the risk of endograft collapse, an uncommon but serious complication of TEVAR.

An alternative to increasing the percentage of oversizing could be performing additional imaging after fluid resuscitation for more adequate aortic measurements. Several recent studies have suggested that delaying TEVAR for patients with TTAI improves survival, and this approach may also be beneficial for correct endograft sizing. Additional aortic imaging could be performed using either a second CT scan or using IVUS during the endovascular procedure. IVUS offers the benefit of real-time sizing during the endovascular procedure when the patient may be better resuscitated than during the CT scan at admission. Therefore, the aortic measurements on IVUS during endovascular repair may be more reliable, and regular use of IVUS during TEVAR of TTAI may be recommended. However, the strategy to delay the endovascular procedure could be lethal in hypovolemic patients with active bleeding. Some studies suggest that fluid resuscitation should be limited in patients with hemorrhagic shock due to trauma or ruptured aneurysm because fluid restriction may limit internal bleeding and its associated loss of platelets and clotting factors.

Further research is warranted to better understand the aortic dynamics during hypovolemia to more accurately predict the normal aortic measurements in hypovolemic patients requiring TEVAR. A better understanding of the
aortic dynamics may eventually result in a reduction of endograft-related complications and further improvements of the prognosis of patients with thoracic aortic rupture.

CONCLUSIONS

Endovascular repair is emerging as the preferred treatment for patients with thoracic aortic rupture. Hypovolemic shock could result in decreased aortic dimensions, which could lead to undersizing of the endograft and an increased risk of endoleak during TEVAR. Therefore, increased oversizing of the endograft or additional aortic imaging after fluid resuscitation may be required in hypovolemic patients with thoracic aortic rupture.

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