

CARDIOVASCULAR SURGERY UPDATE

Acute Aortic Syndromes: Newer Developments

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ulcer; aortic hematoma*

ABBREVIATIONS

AADA = acute aortic dissection type A
IMH = intramural hematoma
PAU = penetrating atherosclerotic ulcer
TEVAR = thoracic endovascular aortic
repair

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INTRODUCTION

Acute aortic syndrome is a modern term that describes the acute presentation of patients with characteristic “aortic” pain caused by one of the life-threatening thoracic aortic conditions including aortic dissection, intramural hematoma and penetrating atherosclerotic ulcer. This entity involves acute lesions of the aorta involving the tunica media and can be distinguished by their etiopathogenesis and characteristic appearance using various diagnostic imaging modalities. Recent advances in imaging techniques and therapeutic interventions have increased awareness of these pathological conditions and emphasized the importance of early diagnosis and treatment. Each of these syndromes also shares the Stanford classification that defines the location and extent of aortic involvement. Stanford **type A** begins in the ascending aorta, while **type B** originates distal to the left subclavian artery to involve the descending thoracic aorta. This classification system is popular since it directs different management strategies and correlates with patient prognosis. Aortic dissections can also be classified as *acute* or *chronic*, depending on whether the dissection is less or greater than 2 weeks old.

Classic aortic dissections are the most common cause of acute aortic syndromes (70%).¹ Aortic dissection is the most common aortic catastrophe occurring two to three times more frequently than abdominal aortic rupture.

A) TYPE A AORTIC DISSECTION

Acute aortic dissection type A (AADA) remains a life threatening medical condition requiring emergent surgical therapy.^{1,2} Despite improvements in diagnostics, medical therapy and surgical technique, patient mortality and morbidity remain high. Standard treatment in the setting of AADA is the replacement of the ascending aorta with resection of the entry site, often in combination with an open distal anastomosis or hemiarch replacement, during a period of circulatory arrest with implementation of adjunct neuroprotective strategies such as cerebral perfusion and hypothermia. However, this treatment leaves the downstream aorta untouched and a residual dissection membrane remains in up to 70% of patients treated for AADA. The risk of progressive dilation with possible need for aortic re-intervention over the long-term remains. Due to this risk, a more aggressive approach with complete arch replacement and possible stenting of the proximal descending aorta via an antegrade approach has been adopted by a number of clinical institutions worldwide to better obliterate the false lumen and thus reduce the incidence of late aortic complications. Other groups, however, have demonstrated an increased risk of mortality and morbidity when exacc

aorta with a suitable form of frozen elephant trunk.tensive surgery involving the aortic arch and the downstream aorta is implemented, thus recommending a more conservative approach to the treatment of AADA patients.

Analysis of the German Registry for Acute Aortic Dissection type A (GERAADA) gave the opportunity to compare the surgical outcomes of patients with AADA treated by total arch replacement and those of hemiarch replacement, with respect to early mortality, and onset of new neurological and malperfusion deficit. In view of these data, it may be concluded that more extensive treatment with total arch replacement and possibly adjunct therapy of the descending aorta can be performed in the setting of the AADA at an acceptable operative risk comparable to the standard treatment with replacement of the ascending aorta. Immediate postoperative complications are higher, however 30-day mortality and the onset of new neurological and malperfusion deficit show no significant difference. In the absence of pre-existing neurological deficits, subgroup analysis demonstrates a higher mortality for patients treated with total arch replacement. Long-term results are presently not obtained by the registry data, and are clearly necessary to justify the necessity of possible aortic re-intervention for patients treated for AADA by the differing surgical approaches. According to the current Mayo clinic perspective, a surgeon should deal with the arch in the manner that is safest in his hands at the time of the acute event, as long as he is prepared to address the thor

B) TYPE B AORTIC DISSECTION

Acute type B aortic dissection (identified within 2 weeks of symptom onset), as described using the Stanford classification, involves the aorta distal to the left subclavian artery and accounts for 25%-40% of all aortic dissections.³ The traditional treatment paradigm of medical management for uncomplicated acute type B dissection and open surgical intervention for early or late complications of type B dissection is currently undergoing a period of evolution as a result of the influence of minimally invasive thoracic endovascular aortic repair options. *Thoracic endovascular aortic repair (TEVAR)* has replaced open surgical repair as the preferred treatment for complicated acute type B aortic dissection, and may also prove beneficial for prophylactic repair of uncomplicated acute type B dissection for high-risk patients. A number of studies have suggested several subsets of high risk uncomplicated patients who may benefit from early TEVAR. Specific predictors of early or late adverse events identified in multiple studies include an initial aortic diameter >4 cm with a patent false lumen, an initial false lumen diameter >22 mm in the proximal descending aorta, recurrent/refractory pain or hypertension, or intramural hematoma with development of a penetrating atherosclerotic ulcer in the proximal descending aorta.

INTRAMURAL HEMATOMA

Aortic intramural hematoma (IMH) account for up to 20% of all cases of acute aortic syndromes and represent a variant dissection characterized by the absence of an intimal flap, reentrant tear, or double channel with false lumen flow. Intramural hematomas often occur in patients with severe atherosclerotic disease in which penetrating aortic ulcers or atherosclerotic plaques rupture causing intimal injury with blood entering the media.

TYPE A IMH

The recommended treatment for patients with type A IMH is prompt surgical intervention. Proximal IMH are independently associated with potential progression to dissection, aneurysm and rupture as well as poor clinical outcomes. The risk of a nonsurgical approach to type A IMH demonstrates an early mortality of 55% compared with 8% following surgical repair. However, for patients with significant comorbidities and uncomplicated type A IMH (no dissection or intimal tear, thickness less than 11 mm, aortic diameter less than 50 mm), medical treatment with follow-up imaging and timed surgical intervention has been recommended.

TYPE B IMH

The management of IMH involving the descending aorta (type B) is similar to that recommended for type B dissections. The current literature supports medical management. However, if complications arise (ulceration, expansion and dilatation), endograft placement may be considered although limited data exists. Endografts may cause erosion of the intima during the acute phase. IMH of the descending aorta have been associated with an in-hospital mortality rate of 10%, similar to that of type B aortic dissection, further emphasizing the importance of correct diagnosis and proper treatment.

PENETRATING ATHEROSCLEROTIC ULCER

The term penetrating atherosclerotic ulcer (PAU) describes a condition in which ulceration of an atherosclerotic lesion penetrates the intima and extends into the media eroding the inner elastic layer of the aortic wall. Penetrating atherosclerotic ulcers are focal lesions most often located in the descending thoracic aorta, which correlates with a greater disease burden in that region. These patients tend to be older with severe systemic atherosclerosis but without connective tissue diseases. Multiple PAUs are often found in a single patient. Imaging of PAU most often reveals extensive atherosclerosis with severe intimal calcification and plaque. A crater or extravasation of contrast is often visualized.

There are multiple important factors to identify when diagnosing PAU. One must determine the number (single or multiple), location (type A or B), and associated complications (IMH, dissection, pseudoaneurysm, and rupture). Type A PAU should be treated surgically. Medical therapy is indicated in stable patients with type B PAU. Very few centers advocate any surgical intervention in uncomplicated type B patients because there is a high risk of organ failure and poor prognosis due to the high likelihood of extensive atherosclerotic disease. For patients with symptomatic or progressive disease, focal PAU in the descending aorta are ideal targets for endograft placement. Criteria for endograft placement in the acute setting include pain and rupture and in the chronic case indications include recurrent pain, aortic diameter greater than 55 mm, and increase in size greater than 10 mm per year.

CONCLUSION

Significant advances have been made in the diagnosis and management of acute aortic dissections over the past

two decades, including surgical techniques for the ascending aorta dissections and percutaneous repair techniques for the descending aorta syndromes. These advancements have led to a better understanding of aortic pathology and led to the discovery of variants that are collectively termed acute aortic syndrome. Despite a persistent level of uncertainty in the diagnosis and management of this lethal disorder, advances are being made and patient outcomes are improving.

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