Cardiovascular Care for Older Adults

Vascular disease in the older adult

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1 Introduction

Among older adults, incidence and prevalence of aortic disease, peripheral arterial disease (PAD), and venous thromboembolism (VTE) increase as age-related alterations in vascular structure and function are compounded by longer exposure to cardiovascular disease (CVD) risk factors. This review highlights the unique presentations and treatment strategies for aortic dissection, abdominal aortic aneurysm (AAA), PAD, and VTE in older adults.

Dai, et al.[1] described some of the distinctive physiologic aspects of cardiovascular aging. Arterial changes include: increased calcium deposition, collagen content and collagen cross-linking; increased vessel diameter and outward remodeling; increased reactive oxygen species and a pro-inflammatory state; and increased apoptosis of vascular smooth muscle cells. Other changes include: alterations in endothelial function, neurohormonal regulation and renal function; and venous stasis with laxity in large venous valves. The cumulative impact of these vascular processes lead to unique physiology and vulnerabilities of older adults, which are commonly manifest in target organ damage and vascular disease.

2 Aortic diseases

2.1 Aortic dissection

Aortic diseases are usually asymptomatic and often not detected until an acute and often catastrophic complication arises. Acute aortic syndromes include aortic dissections, intramural hematomas and penetrating atherosclerotic ulcers.

Most patients present with chest pain (80%) that is more commonly anterior (71%) than posterior (32%), but older adults are relatively more likely to present with syncope, stroke, or congestive heart failure rather than pain.[2] Aortic dissection evolves from medial degeneration, which is characterized by disruption and loss of elastic fibers, increased deposition of proteoglycans, and loss of smooth muscle cells in the aortic media. Whereas Marfan syndrome commonly underlies aortic dissection in the young, in older adults, hypertension, atherosclerosis and iatrogenic etiologies tend to be the more common causal factors.[3]

Stanford type A aortic dissections, which include all dissections involving the ascending aorta, are severe and life-threatening, with an ominous prognosis if left untreated. In the International Registry of Acute Aortic Dissection, 32% of patients with type A aortic dissection were ≥ 70 years of age.[3] Older adults with type A aortic dissections are more likely than younger patients to present with hypotension, which is a particularly threatening sign. While older adults are less likely than younger patients to be managed surgically, surgical outcomes for uncomplicated type A aortic dissection are acceptable in the older adult population.[4] When denied surgery, in-hospital mortality rates for octogenarians are 45%–62%,[3] whereas, surgical intervention was associated with a 63% one year survival rate in a series of octogenarians with uncomplicated aortic dissection.[4] On the other hand, complicated aortic dissections, which present with neurologic deficits, mesenteric ischemia or cardiopulmonary resuscitation, carry a very high surgical risk and might be best managed medically.[4]

Type B aortic dissections do not involve the ascending aorta and nonsurgical treatment is usually recommended.[2] Medical stabilization with intravenous β-blocker therapy to a goal heart rate less than 60 beats/min and systolic blood pressure of 100 to 120 mmHg and prompt cardiac surgical consultation are the initial strategies. Endovascular stent grafting or open repair is reserved for patients with high risk of rupture.[1]
2.2 Abdominal aortic aneurysm

The prevalence of AAA is five times more common in men than women and increases with age, rising from 1.3% in men and 0 in women aged 45 to 54 years up to 12.5% in men and 5.2% in women aged 75 to 84 years.[5] AAA results from medial degeneration that reduces the integrity of the aortic wall and is defined by an abdominal aorta diameter > 3 cm, with rates of growth that are generally 1–5 mm per year. While often asymptomatic, presenting symptoms of AAA in older adults can be quite varied and include failure to thrive due to mesenteric ischemia or acute renal failure due to atherosclerotic processes or thromboembolic disease.

Rupture is the most widely recognized complication of AAA and carries a staggering high mortality rate of up to 90%.[5] Risk of rupture is directly related to aneurysm size, such that eventual rupture risk is 20% for AAA > 5 cm, 40% for AAA > 6 cm, and > 50% for AAA > 7 cm; while it is quite low for AAA < 4 cm. Other complications of AAA include thromboembolic ischemic events and the compression of or erosion into adjacent structures.

The US Preventive Services Task Force recommends screening with abdominal duplex ultrasonography for AAA in all men aged 65 to 75 years old who have ever smoked and selectively in men who have never smoked.[6] There is insufficient evidence to support screening ultrasound studies in women; however, studies in women who have ever smoked have been underpowered to definitively draw conclusions. Risk factors for AAA that might augment interest in screening include older age, smoking history, having a first-degree relative with an AAA, and any history of CVD, hyperlipidemia, obesity or hypertension. If the abdominal aorta measures < 3 cm in individuals between ages 65–75 years old, there is no need for additional ultrasound studies. For AAA of 3–5.4 cm, ultrasound surveillance at a frequency of every 3 to 12 months is recommended. For AAA ≥ 5.5 cm in diameter or that have a rapid growth rate (> 1 cm per year), angiography with computed tomography or magnetic resonance is warranted for size confirmation and these patients should be evaluated for repair.

Medical therapy for AAA includes tobacco cessation, blood pressure control, and statin therapy. A meta-analysis of observational comparative studies of statin therapy versus placebo or no statin in patients with AAA < 5.5 cm suggested a slowing of the growth rate for statin therapy that was more effective as the baseline diameter increased.[7] As with all forms of CVD, AAA demands a secondary level of prevention and comprehensive risk factor modification.

Endovascular or open surgical repair is indicated for patients with AAA ≥ 5.5 cm or that have a rapid growth rate, with the great majority of older adults currently treated with endovascular aortic repair (EVAR). By 2010, EVAR accounted for 74% of all AAA repairs in the United States.[8] Randomized, controlled trials and large observational studies demonstrate an early survival benefit with EVAR as compared with open surgical repair that persists for up to three years.[9] The overall perioperative mortality for EVAR was 1.6% versus 5.2% with open repair, with a 3-fold relative risk reduction for EVAR that was consistent through all age groups and included a large number of octogenarians.[9] Comparatively, the risk of mortality following repair of a ruptured AAA is upwards of 50%, making elective AAA repair a useful intervention in most older adults.

3 Peripheral arterial disease

3.1 Epidemiology

PAD is common in older adults, with a prevalence that rises steeply with age to nearly 25% of adults ≥ 80 years old in the United States.[10] In a large database of over 3.6 million screened subjects, the prevalence of any peripheral vascular disease (defined as PAD, AAA or carotid stenosis) increased from 1 in 50 to nearly 1 in 3 for the 40–50 year old and 91–100 year old age groups, respectively.[11] While age is an independent risk factor, cigarette smoking, diabetes, hypertension and hypercholesterolemia, in that order, are important modifiable risk factors for PAD.

Despite its high prevalence, only about 10% of people with PAD have the classic symptoms of intermittent claudication, while 40% are asymptomatic and the remaining 50% have leg symptoms different from classic claudication.[12,13] This observation might be more pronounced in older adults where only 6.3% of those in the Rotterdam Study with PAD reported symptoms of intermittent claudication.[14] Higher prevalence of comorbidities such as neuropathy, osteoarthritis, spinal stenosis, chronic lung disease and congestive heart failure, which might elicit indistinguishable symptoms and might impair mobility, could explain why claudication is underappreciated by older adults.

PAD is an important independent determinant of overall CVD risk, as patients with PAD are at an increased risk of systemic cardiovascular events as well as limb-related morbidity. Patients with PAD often have concomitant cerebrovascular and ischemic heart disease. In the Framingham study, a low ankle brachial index (ABI), less than ≤ 0.90, was associated with approximately a three-fold increased 10 year cardiovascular mortality after adjusting for the Framingham risk score.[15]
3.2 Diagnosis and evaluation

A diagnosis of PAD can be established with modern noninvasive vascular techniques, including ABI and toe brachial indices, segmental pressure measurements, pulse volume recordings, duplex ultrasound imaging, and exercise testing. The ABI is the standard measurement that provides objective data as a diagnostic and prognostic tool. The ABI is obtained by measuring systolic blood pressure in both brachial arteries and both posterior tibials and dorsalis pedis arteries after the patient has been at rest in a supine position for 10 min. The higher brachial pressure is used as the denominator, and left and right ABIs are calculated using the higher ankle pressure on each side. Due to the reflected wave, ankle pressures should be 10 to 15 mmHg higher than brachial pressures in healthy individuals, and the normal ABI is > 1.00. Guidelines support ABI measurements in all new patients with PAD, and in individuals with exertional leg symptoms or non-healing wounds who are ≥ 70 years or who are ≥ 50 years with a history of smoking or diabetes.

In older adults, arterial stiffening can lead to a falsely elevated ABI and an ABI > 1.30 may be consistent with PAD in this population. In fact, there is a reversed J-shaped distribution for CVD risk, with increased cardiovascular mortality for both low ABI (≤ 0.90) and high ABI (> 1.40). Also, in the most severe form of PAD, critical limb ischemia (CLI), ABI measurements can be unreliable. In both of these populations, the toe brachial index should used to establish the presence of lower extremity PAD, and toe brachial index values < 0.70 are considered diagnostic. This test is performed by placement of a small cuff on the proximal portion of the great toe with use of a plethysmographic detection device to assess return of toe pulsatility. Additional diagnostic imaging, including duplex ultrasound, computed tomographic angiography and magnetic resonance angiography are useful to diagnose anatomic location and the presence of significant stenosis in patients with lower extremity PAD, as well as to plan interventions.

3.3 Treatment

The optimal management of PAD requires a comprehensive treatment strategy that includes both lifestyle changes, such as smoking cessation and exercise, as well as optimal medical therapy to a secondary level. The treatment recommendations for optimal therapy of PAD are similar in older adults and younger persons, yet older adults are less likely to receive it. Smoking is the most important risk factor for PAD, yet older adults are less likely to be referred for comprehensive smoking cessation interventions. Hypertension affects 65% of USA adults ≥ 60 years of age, yet control of hypertension is achieved in only 50.5% according to the National Health and Nutrition Examination Survey 2011 to 2012. Statin therapy may improve symptoms of intermittent claudication and should be administered with high-intensity therapy to most patients with PAD, although moderate-intensity therapy may be more appropriate for many patients older than 75 years who do not tolerate higher intensity dosing.

Therapies recommended specifically for older adults with PAD include supervised exercise training, angiotensin converting enzyme (ACE) inhibitors, antplatelet agents, and drugs to increase walking distance such as cilostazol. Supervised exercise training includes 30–45 min sessions of walking to near-maximal pain in sessions at least three times per week for a minimum of 12 weeks. The ACE inhibitor ramipril has been associated with improved maximal walking distance in several studies and may have particular benefit. Aspirin is recommended in all patients with PAD to reduce the risk of CVD events, and clopidogrel can be used as an effective alternative. Cilostazol, although contraindicated in patients with heart failure, improves maximal walking distance and quality of life scores on the Short Form 36, performing similarly in those less than and over 65 years of age.

Endovascular therapy (atherectomy, angioplasty, and stenting) or surgical peripheral arterial bypass is indicated for those with lifestyle-limiting claudication if there is not an adequate response to exercise and pharmacologic interventions and for those with CLI. Owing to increased prevalence of diabetes and chronic kidney disease, the risk for contrast-induced nephrotoxicity is increased for older adults and should be assessed with the patient and family prior to angiography. An evaluation of perioperative cardiovascular risk and anatomic determinants are important to deciphering the risks and benefits of open surgical versus endovascular repair for older adults. A recent analysis of over 640,000 admissions from the Nationwide Inpatient Sample demonstrated that for patients with CLI, endovascular revascularization procedures are on the rise and have been associated with decreasing in-hospital mortality and major amputation rates, as well as length of stay and cost of hospitalization, compared with surgery. In general, although high-level evidence demonstrating the superiority of one method over the other is lacking, an endovascular first approach may be advisable in patients with significant comorbidity.

Amputation is indicated if tissue loss is beyond salvage, if functional limitation provides no reason for limb salvage, or for relief of severe ischemic pain despite attempted revascularization. As older adults are less likely to adapt to prosthetic devices and limb loss, amputation can often be-
come a decision point for nursing home placement. Amputation also reduces independence and has been associated with a high mortality rate in all adults. With improved adherence to optimal medical therapy and revascularization techniques, amputation rates in the United States declined by about 25% between 2000 and 2008. However, the older adult population continues to be over-represented in the amputation statistics, likely related to late recognition of PAD.

4 Venous thromboembolism

4.1 Epidemiology

Incidence rates for deep venous thrombosis (DVT) and pulmonary embolus (PE) increase exponentially with age for both men and women. The physiologic basis for this observation appears related to alterations in thrombotic factors and laxity in large venous valves. The incidence of VTE rises from a rate of < 5 per 100,000 per year among children to the range of 450 to 600 per 100,000 per year (about 0.5% per year) among individuals ≥ 80 years old, with a sharp incline in risk after the 6th decade. Also, PE comprises a larger percent age of all VTE events as age increases.

Similar to other topics in this series, older adults are less likely than younger adults to present with typical symptoms. For DVT, this might be due to a relatively lower incidence of isolated calf DVT in older adults who are more likely to have proximal disease. Also, comorbidities may mask or confuse symptoms of VTE. Older adults with PE are more likely than younger adults to present with cough and syncope and less likely to present with pleuritic chest pain. PE in particular requires a high index of suspicion and should be considered when older patients are admitted with shortness of breath that poorly responds to therapy aimed at an alternative or presumptive diagnosis.

VTE should be considered a chronic disease with likelihood of episodic recurrence. Without long-term anticoagulation, about 30% of patients develop a recurrence within 10 years. Age, prior VTE, active cancer, recent surgery or immobilization, central venous catheters or transvenous pacemaker, infection, thrombophilia, kidney disease, and leg paresis from neurological disease are all independent predictors of VTE events. Additionally, age is an important risk factor for major bleeding complications of anticoagulation. An international registry of patients with acute VTE treated with vitamin K antagonist therapy and followed for 3 months identified a major bleeding risk of 3.4% and 2.1% for patients ≥ 80 years and < 80 years, respectively.

4.2 Treatment

The most important intervention for treatment of VTE in older adults is prevention. Early mobilization and use of sequential compression devices or thromboprophylaxis in hospitalized older adults is paramount. Clinical guidelines support use of low-molecular-weight heparin or low-dose unfractionated heparin during the period of immobilization or acute hospital stay for acutely ill hospitalized medical patients, or intermittent pneumatic compression and/or compression stockings for those at high risk for major bleeding.

Once a VTE event has been diagnosed, anticoagulation therapy should be administered. For acute PE, risk stratification requires assessment of hemodynamic instability and evidence of right heart strain. For older adults with acute PE associated with hypotension, systemically administered thrombolytic therapy is recommended; and, in selected patients with acute PE who deteriorate or have a high bleeding risk, catheter-assisted thrombus removal or catheter-based thrombolysis can be considered. Newer guidelines also now recommend long-term anticoagulation therapy with dabigatran, rivaroxaban, apixaban, or edoxaban over vitamin K antagonist therapy when no cancer is present; and, in selected patients with acute PE who deteriorate or have a high bleeding risk, catheter-assisted thrombus removal or catheter-based thrombolysis can be considered. Newer guidelines also now recommend long-term anticoagulation therapy with dabigatran, rivaroxaban, apixaban, or edoxaban over vitamin K antagonist therapy when no cancer is present; and, in selected patients with acute PE who deteriorate or have a high bleeding risk, catheter-assisted thrombus removal or catheter-based thrombolysis can be considered.
year in the very old (about 10-fold higher than the incidence in a 50 year old adult), predominantly in the setting of immobility. Aggressive prophylaxis through sequential compression devices and early mobilization can decrease exposure to the higher bleeding risks associated with anticoagulation in older adults.

Acute PE presents more frequently as syncope in older adults and is usually best treated with anticoagulation, with thrombolysis or surgical/catheter-based embolectomy reserved for hemodynamic instability.

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