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Mini Review

Evolving Approaches to Abdominal Aortic Aneurysms: A Short Review

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Abstract

Abdominal Aortic Aneurysm (AAA) remains a significant cause of morbidity and mortality, particularly in older adults. Over the past two decades, advances in imaging, risk stratification, and treatment modalities have transformed the management of AAA. This short review highlights the current understanding of AAA pathophysiology, summarizes screening and surveillance strategies, and discusses the evolution of surgical approaches, particularly the shift from open surgical repair to Endovascular Aneurysm Repair (EVAR). Ongoing innovations and unresolved challenges are also briefly addressed.

Introduction

Abdominal Aortic Aneurysm (AAA) is a localized dilatation of the abdominal aorta that exceeds 3.0 cm in diameter or is more than 50% larger than the normal diameter of the adjacent segment. It is a potentially life-threatening condition due to its often silent progression and the high mortality associated with rupture. AAAs are among the most common aneurysmal diseases, accounting for the majority of aortic aneurysms diagnosed in clinical practice [1].

The natural history of AAA is variable. Many cases remain asymptomatic for years and are discovered incidentally through imaging performed for unrelated conditions. However, once the aneurysm reaches a critical size, the risk of rupture increases significantly, with fatal outcomes in up to 90% of cases if emergency surgical repair is not immediately available [2]. Ruptured AAA is responsible for an estimated 1% – 2% of all deaths in men over the age of 65 in developed countries, making it a significant public health concern [3].

Traditionally, Open Surgical Repair (OSR) was the only definitive treatment option for AAA. Over the past three decades, however, the advent of Endovascular Aneurysm Repair (EVAR) has revolutionized management strategies. EVAR offers a less invasive alternative with reduced perioperative morbidity and shorter recovery time, although it brings unique challenges in terms of long-term surveillance and reintervention [4] (Table 1).

The management of AAA has continued to evolve with advances in imaging technology, device design, and patient selection strategies. In addition, increased awareness and implementation of screening programs in at-risk populations have led to earlier detection and more elective repairs, which are associated with better outcomes [5].

Given the significant changes in diagnostic and therapeutic approaches to AAA over recent decades, a concise synthesis of current practices is essential. This short review aims to highlight the evolving trends in AAA management, focusing on epidemiology, screening protocols, pathophysiological mechanisms, and the shift from open surgical repair to

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this article is not exhaustive, and selection bias may have influenced the emphasis on certain themes. Additionally, rapid advances in endovascular technologies may limit the long-term relevance of some findings discussed (Table 2).

Epidemiology and risk factors

Abdominal Aortic Aneurysms (AAAs) predominantly affect older adults, with a strong male predominance. The estimated global prevalence of AAA ranges from 1.3% to 8.9%, with variation depending on population demographics, screening practices, and definitions used [6]. In men over 65 years, the prevalence has historically been reported at approximately 4% - 7%, while in women of the same age group, it is considerably lower, typically under 1% [7]. However, when present in women, AAAs tend to rupture at smaller diameters and are associated with higher mortality [8] (Table 3).

Table 1: Comparison of Open Surgical Repair (OSR) and Endovascular Aneurysm Repair (EVAR).

Feature	Open Surgical Repair (OSR)	Endovascular Aneurysm Repair (EVAR)
Invasiveness	High	Minimally invasive
Anesthesia required	General	Local/regional or general
Hospital stay	Longer (7–10 days)	Shorter (2–3 days)
Perioperative mortality	~4–5%	~1–2%
Durability	High (often lifelong)	Lower; requires ongoing surveillance
Reintervention rate	Low	Higher (due to endoleaks or migration)
Suitable for complex anatomy	Yes	Limited (unless using fenestrated/branched EVAR)
Follow-up requirements	Minimal after recovery	Lifelong imaging follow-up required

Table 2: Risk Factors for Abdominal Aortic Aneurysm.

Risk Factor	Type	Evidence/Comments
Male sex	Non-modifiable	4–6× higher risk than females
Age > 65 years	Non-modifiable	Incidence increases with age
Smoking (current/ former)	Modifiable	Strongest risk factor; dose-dependent effect
Family history of AAA	Non-modifiable	2–4× increased risk; suggests genetic predisposition
Hypertension	Modifiable	Associated with increased wall stress
Atherosclerosis	Modifiable	Common coexisting condition
Hyperlipidemia	Modifiable	Less directly correlated than in CAD
Diabetes mellitus	Modifiable	Paradoxically associated with lower AAA risk

Table 3: Surveillance Recommendations Based on Aneurysm Diameter.

Aneurysm Diameter	Recommended Follow-up Interval	Comments
<3.0 cm	No follow-up	Considered normal
3.0–3.9 cm	Every 2–3 years	Low risk of rupture
4.0–4.9 cm	Every 12 months	Intermediate risk
5.0–5.4 cm	Every 6 months	Higher risk; consider timing for intervention
≥5.5 cm (men) / ≥ 5.0 cm (women)	Elective repair recommended	Based on guideline thresholds

Aging is one of the most significant risk factors for AAA development, likely due to cumulative vascular damage and degenerative changes in the aortic wall. The incidence of AAA increases sharply after the age of 65 and continues to rise with age [9]. Male sex is a well-established risk factor, with men being up to four times more likely to develop AAA than women [10]. Other non-modifiable risk factors include a family history of AAA, which confers a two- to fourfold increased risk, suggesting a genetic predisposition [11].

Among modifiable risk factors, cigarette smoking is the most important and consistent contributor. Current or former smokers have a substantially higher risk of developing AAA and are more likely to experience faster aneurysm growth and rupture [12]. A dose-response relationship has been observed, with risk increasing with the duration and intensity of smoking. Other cardiovascular risks factors such as hypertension, dyslipidemia, and atherosclerosis are also associated with AAA formation, although their role is less pronounced compared to their impact on occlusive arterial disease [13].

Interestingly, patients with diabetes mellitus appear to have a paradoxically lower incidence of AAA, a phenomenon that remains poorly understood but may be related to alterations in matrix remodeling or vessel wall inflammation [14].

Over the past two decades, the prevalence of AAA appears to be declining in some regions, likely due to decreased smoking rates and increased screening awareness [15]. Nevertheless, AAA remains a significant cause of morbidity and mortality, particularly in aging populations. Population-based screening programs, especially in men aged 65 and older who have ever smoked, have proven effective in reducing aneurysm-related deaths through early detection and elective repair.

Pathophysiology

Abdominal Aortic Aneurysms (AAAs) result from a complex interplay of inflammatory, proteolytic, and biomechanical processes that lead to progressive weakening of the aortic wall. Chronic inflammation in the aortic media and adventitia promotes infiltration of macrophages, T-lymphocytes, and neutrophils, which release cytokines and matrix-degrading enzymes. Key among these is matrix metalloproteinases (MMP-2 and MMP-9), which degrade elastin and collagen in the extracellular matrix, compromising structural integrity. Simultaneously, smooth muscle cell apoptosis reduces the regenerative capacity of the vessel wall and impairs extracellular matrix maintenance. Oxidative stress and reactive oxygen species further exacerbate vascular injury and inflammation. Hemodynamic forces, especially in the infrarenal aorta, where flow turbulence and wall tension are greatest, contribute to aneurysm formation and expansion. Genetic factors and familial clustering suggest inherited susceptibility, with several loci associated with extracellular matrix remodeling and inflammatory pathways. Together, these mechanisms lead to progressive aortic dilation, increased wall stress, and eventual risk of rupture.

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Surveillance and screening

Ultrasound is the gold standard for AAA screening due to its high sensitivity, non-invasiveness, and cost-effectiveness [10]. Current guidelines recommend one-time screening for men aged 65–75 years who have a history of smoking [11]. Surveillance frequency depends on aneurysm size:

- 3.0–3.9 cm: every 2–3 years
- 4.0–4.9 cm: annually
- 5.0–5.4 cm: every 6 months

Intervention is typically considered for aneurysms >5.5 cm in men or >5.0 cm in women, or if rapid expansion (>0.5 cm in 6 months) occurs [12].

Evolving surgical approaches

Open Surgical Repair (OSR)

Open surgical repair, involving replacement of the diseased aorta with a synthetic graft, was the standard for decades. It provides durable results, especially for patients with low surgical risk and favorable anatomy [13].

Endovascular Aneurysm Repair (EVAR)

EVAR, introduced in the 1990s, has rapidly become the preferred option in many cases. The technique involves deployment of a stent-graft through the femoral arteries to exclude the aneurysm sac from systemic circulation [14]. EVAR is associated with lower perioperative mortality and shorter hospital stay but requires ongoing surveillance due to risks of endoleaks and graft migration [15].

Current trends

There has been a notable global shift toward EVAR in elective AAA repair. In many high-income countries, EVAR now accounts for over 75% of interventions [16]. Complex anatomies once considered unsuitable for EVAR are now being managed with fenestrated and branched stent-grafts [17].

Anesthesia considerations

Anesthesia approaches for AAA repair vary depending on the chosen surgical technique and patient comorbidities. Open surgical repair typically requires general anesthesia due to the extensive nature of the procedure. In contrast, Endovascular Aneurysm Repair (EVAR) can often be performed under regional or even local anesthesia with sedation, which has been associated with reduced hemodynamic stress and shorter recovery times. Anesthesia choice should be individualized, with multidisciplinary input from vascular surgeons and anesthesiologists to optimize perioperative outcomes.

Challenges and future directions

Despite its advantages, EVAR has limitations in terms of long-term durability and the need for secondary interventions [18]. Late complications such as endoleaks, sac enlargement, and device migration remain concerns [19]. Emerging areas of interest include:

- Personalized stent-graft design and 3D printing for complex anatomies [20]
- Adjunctive pharmacotherapy to slow aneurysm growth [21]
- Machine learning models for rupture risk stratification [22]
- Use of intravascular imaging (e.g., IVUS, OCT) during EVAR to enhance precision [23-25].

Conclusion

The management of AAA has evolved significantly over the past few decades, with a major shift toward minimally invasive endovascular techniques. While EVAR has improved perioperative outcomes, long-term follow-up, and individualized decision-making remain essential. Continued innovation in imaging, devices, and predictive analytics holds promise for further improving patient outcomes.

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