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ABDOMINAL AORTIC ANEURYSM: CURRENT INSIGHTS INTO RISK FACTORS, PATHOGENESIS, DIAGNOSTIC STRATEGIES, AND THERAPEUTIC APPROACHES

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ABSTRACT
Abdominal aortic aneurysm (AAA) is a progressive vascular disorder characterized by abnormal dilatation of the abdominal aorta, defined as a diameter of ≥3 cm or more than 50% above normal. It is a potentially life-threatening condition due to the risk of rupture, leading to severe internal bleeding and high mortality. AAA commonly affects elderly individuals, especially males, and is strongly associated with risk factors such as smoking, hypertension, atherosclerosis, dyslipidemia, and genetic predisposition. The pathogenesis involves inflammation, extracellular matrix degradation, oxidative stress, and vascular smooth muscle cell loss, resulting in weakening of the aortic wall. Most AAAs remain asymptomatic and are often detected incidentally through imaging techniques such as ultrasonography and CT scan. . Management strategies depend on aneurysm size, growth rate, and patient risk factors, ranging from regular surveillance to surgical interventions such as open surgical repair and endovascular aneurysm repair (EVAR). Early screening and appropriate therapeutic interventions are essential to reduce morbidity and mortality associated with AAA.

KEYWORDS: *Abdominal Aortic Aneurysm (AAA), Aortic Dilatation, Vascular Disease, Pathophysiology, Risk Factors, Diagnosis, Management*

INTRODUCTION

Abdominal aortic aneurysm (AAA) is an abnormal, focal dilatation of the abdominal aorta and is defined as an aortic diameter of 3 cm or greater or an increase of more than 50% compared to the normal vessel diameter. It represents a potentially life-threatening condition that requires careful monitoring or intervention depending on aneurysm size, rate of growth, and associated symptoms. AAAs are considered true aneurysms because they involve all three layers of the arterial wall—intima, media, and adventitia—and most commonly occur in the infrarenal segment of the aorta, often extending into the iliac arteries.

AAA aneurysms are relatively common, particularly in older populations, affecting approximately 2–8% of men over the age of 65 years, and are about five times more common in men than in women. The clinical course of AAA is often silent, with many cases detected incidentally during imaging for unrelated conditions. However, the condition carries a significant risk of rupture, which increases with aneurysm size. The annual risk of rupture is less than 1% for aneurysms smaller than 5.5 cm, approximately 10% for aneurysms measuring 5.5–7 cm, and up to 33% for aneurysms larger than 7 cm. Rupture of an AAA is associated with extremely high mortality, ranging from 85% to 90%, making it a major cause of sudden death in elderly individuals. Globally, aortic aneurysms accounted for over 168,000 deaths in 2013, reflecting a substantial increase over previous decades. [1-2]

RISK FACTORS AND AETIOLOGY

The development of an abdominal aortic aneurysm is influenced by a combination of genetic, environmental, and cardiovascular risk factors. Although the precise initiating mechanism responsible for aneurysm formation is not fully understood, several well-established factors contribute to weakening of the aortic wall and progression of aneurysmal dilation. These factors influence both the initiation of aneurysm formation and the rate of aneurysm growth and rupture. [3-5]

- **Age and Sex:** Age is one of the most important determinants in the development of AAA. The incidence of AAA increases markedly with advancing age, particularly after the age of 65 years. Epidemiological studies have shown that the annual incidence of AAA rises dramatically from less than 1 case per 100,000 individuals in people aged 40–44 years to more than 160 cases per 100,000 individuals in those aged 75–79 years. This substantial increase reflects age-related degenerative changes in the aortic wall, including loss of elastin, increased collagen degradation, and reduced regenerative capacity of vascular smooth muscle cells. Sex also plays a significant role in the epidemiology of AAA. The condition occurs significantly more frequently in men than in women, with men exhibiting a risk that is approximately four to six times higher. In women, the development of AAA is generally delayed until later in life, possibly due to the protective effects of estrogen before menopause. However, when aneurysms occur in women, they tend to have a higher risk of rupture at smaller diameters.
- **Smoking:** Smoking is the most important modifiable risk factor for AAA, with smokers having a three to twelve times higher risk than non-smokers. Tobacco exposure promotes aneurysm formation by increasing oxidative stress, inflammation, and degradation of elastin in the aortic wall. The risk rises with longer duration of smoking and declines gradually after cessation,

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although it may persist for years. Experimental studies also show that tobacco smoke accelerates elastin degradation and aneurysm enlargement, while promoting immune reactions and vascular damage in the aortic wall.

- **Hypertension:** Hypertension is an important risk factor for the development and progression of AAA. Elevated blood pressure increases mechanical stress on the arterial wall, promoting degeneration of the aortic structure, disruption of elastic fibers, and weakening of the vessel wall. Studies show that individuals with hypertension have a higher risk of developing AAA and may also have an increased risk of aneurysm rupture due to greater tensile stress on the weakened aorta. Hypertension further contributes to aneurysm progression by stimulating matrix degradation and inflammatory processes through activation of matrix metalloproteinases and related signaling pathways.
- **Dyslipidaemia:** Abnormal lipid metabolism is also associated with the development of AAA. Elevated levels of low-density lipoprotein cholesterol (LDL-C) have been linked to an increased risk of aneurysm formation, whereas higher levels of high-density lipoprotein cholesterol (HDL-C) appear to have a protective effect. HDL cholesterol may protect against aneurysm development through its anti-inflammatory and antioxidant properties. Although dyslipidemia is strongly associated with the presence of AAA, current evidence suggests that lipid abnormalities may not significantly influence aneurysm growth rate or rupture risk. Nevertheless, dyslipidemia contributes to vascular damage and atherosclerotic changes that may predispose individuals to aneurysm formation.
- **Family History and Genetic Factors:** Genetic predisposition plays a significant role in the development of AAA. Approximately 6–20% of patients with AAA have a positive family history, and first-degree relatives of affected individuals have roughly double the risk of developing the condition compared with the general population. Studies of twins have provided further evidence of genetic influence, showing markedly increased concordance rates among monozygotic twins. Despite strong epidemiological evidence supporting heritability, specific genetic mutations responsible for most cases of AAA have not yet been clearly identified. However, rare hereditary connective tissue disorders such as Marfan syndrome and Ehlers–Danlos syndrome are strongly associated with aneurysm formation due to structural defects in connective tissue proteins.
- **Atherosclerosis and Cardiovascular Disease:** AAA frequently occurs in individuals with advanced atherosclerosis, leading to the long-standing assumption that aneurysms are caused by atherosclerotic degeneration of the aortic wall. Atherosclerosis may contribute to aneurysm formation by causing mechanical weakening of the vessel wall and reducing blood supply to the aortic media through obstruction of the vasa vasorum. However, the relationship between atherosclerosis and AAA is complex. While many patients with AAA exhibit atherosclerotic changes, not all individuals with severe atherosclerosis develop aneurysms. Therefore, atherosclerosis is now considered a contributing factor rather than the primary cause of aneurysm development.
- **Other Contributing Factors:** Several additional conditions may increase the risk of developing AAA. Chronic obstructive pulmonary disease (COPD) has been associated with AAA, possibly due to shared risk factors such as smoking and systemic inflammation. Long-term alcohol consumption and inflammatory vascular diseases may also contribute to vascular wall damage. Other rare causes include infections, trauma, arteritis, and degenerative changes such as cystic medial necrosis.

PATHOPHYSIOLOGY

Abdominal aortic aneurysm is a complex vascular disorder characterized by progressive dilation of the abdominal aorta due to structural weakening of the vessel wall. The pathophysiology of AAA involves a multifactorial interplay between genetic susceptibility, biochemical processes, inflammatory responses, and biomechanical forces. These processes collectively lead to the degeneration of the aortic wall, resulting in aneurysmal dilation and an increased risk of rupture. The normal abdominal aorta gradually increases in diameter with age. After the age of 50 years, the average diameter of the infrarenal aorta is approximately 1.5 cm in women and 1.7 cm in men. An aortic diameter of 3 cm or greater is considered diagnostic of an AAA, even in asymptomatic individuals. Approximately 90% of AAA occur in the infrarenal portion of the aorta, indicating that this region is particularly susceptible to aneurysmal degeneration. The formation and progression of AAA are primarily associated with the destruction of the structural components of the aortic wall, particularly elastin and collagen. These proteins form the extracellular matrix (ECM) of the aortic wall and are responsible for maintaining its elasticity and tensile strength. Degeneration of the medial layer of the aorta leads to progressive widening of the vessel lumen and loss of mechanical stability. [6-8]

Degradation of Extracellular Matrix: The extracellular matrix of the aortic wall provides the structural framework that enables the vessel to withstand continuous hemodynamic pressure. Elastin provides elasticity and recoil, while collagen contributes to tensile strength. In AAA, progressive proteolytic degradation of elastin and collagen weakens the vessel wall. This degradation is largely mediated by a group of proteolytic enzymes known as matrix metalloproteinases (MMPs), along with other proteases such as serine proteases and plasmin. These enzymes are produced by inflammatory cells, including macrophages and neutrophils, as well as by vascular smooth muscle cells within the aortic wall. Under normal physiological conditions, MMP activity is balanced by tissue inhibitors of metalloproteinases (TIMPs), which regulate extracellular matrix remodeling. However, in AAA this balance shifts toward increased protease activity and decreased inhibitor activity, leading to progressive destruction of the structural matrix components of the aortic wall. The degradation of elastin is particularly significant because elastin is the principal load-bearing component of the aortic wall. Loss of elastin reduces the elastic recoil of the vessel and allows progressive dilation under arterial pressure.

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Elastin Fragmentation and Medial Degeneration: Histological examination of aneurysmal aortic tissue reveals several characteristic structural changes, including fragmentation of elastin fibers, thinning of the medial layer, and loss of vascular smooth muscle cells. The medial layer normally contains concentric layers of elastin and smooth muscle cells that allow the aorta to maintain its structural integrity. In the infrarenal aorta, the number of elastin layers is naturally lower compared with the thoracic aorta. This anatomical feature makes the infrarenal region particularly vulnerable to structural damage and aneurysm formation. Degeneration of elastin fibers and thinning of the medial layer significantly compromise the mechanical strength of the vessel wall, allowing progressive dilation of the aortic lumen.

Inflammatory and Immune Mechanisms: Inflammation plays a central role in the pathogenesis of AAA. Aneurysmal tissue typically exhibits chronic inflammatory infiltration, particularly in the adventitial and medial layers of the aortic wall. The infiltrating cells include macrophages, lymphocytes, neutrophils, and mast cells. These inflammatory cells release a variety of pro-inflammatory cytokines and mediators, including interleukin-1 (IL-1), interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis factor-alpha (TNF-α). These mediators stimulate the production of matrix metalloproteinases and other proteolytic enzymes that degrade extracellular matrix components. In addition to innate immune responses, adaptive immune mechanisms also contribute to AAA development. The adventitia may develop organized lymphoid structures containing B cells and T cells, which generate antibodies against modified self-proteins produced during oxidative and proteolytic injury. These immune responses further perpetuate inflammation and tissue damage within the aortic wall.

Loss of Vascular Smooth Muscle Cells: Vascular smooth muscle cells (VSMCs) are essential for maintaining the structural and functional integrity of the aortic wall. They synthesize extracellular matrix components such as elastin and collagen and participate in vessel wall repair. In AAA, a significant reduction in VSMCs occurs due to apoptosis, oxidative injury, and detachment from the extracellular matrix. Proteolytic enzymes and oxidative stress damage the cellular environment, leading to VSMC death. The loss of these cells reduces the ability of the aortic wall to repair itself and maintain structural stability, thereby accelerating aneurysm progression.

Oxidative Stress: Oxidative stress is another important factor in the pathogenesis of AAA. It results from an imbalance between the production of reactive oxygen species (ROS) and the antioxidant defense mechanisms of the tissue. Reactive oxygen species generated by inflammatory cells and trapped red blood cells within the aneurysm promote oxidative damage to proteins, lipids, and DNA. Oxidative stress also activates matrix metalloproteinases and contributes to the degradation of collagen fibers in the aortic wall. Furthermore, oxidative modification of proteins can create new antigenic determinants that stimulate immune responses, thereby amplifying inflammation within the aneurysm.

Hemodynamic and Biomechanical Stress: Mechanical forces exerted on the aortic wall significantly influence aneurysm development and progression. According to Laplace's law, the tension in the vessel wall is directly proportional to the radius of the vessel and the internal pressure, and inversely proportional to wall thickness. As the aneurysm enlarges and the wall becomes thinner, the tensile stress on the vessel wall increases, creating a cycle of progressive dilation. The infrarenal aorta is exposed to unique hemodynamic conditions due to its proximity to the iliac bifurcation. Turbulent blood flow, pressure fluctuations, and reflected pulse waves in this region increase mechanical stress on the vessel wall, further promoting aneurysm formation and expansion.

Role of Intraluminal Thrombus: Many AAA contain a multilayered intraluminal thrombus (ILT). Rather than acting as a protective barrier, the thrombus contributes to disease progression. The ILT contains inflammatory cells, platelets, fibrin, and trapped red blood cells, which release proteolytic enzymes, oxidant molecules, and inflammatory mediators. These substances diffuse into the underlying aortic wall, promoting extracellular matrix degradation and vascular smooth muscle cell death. The ILT also limits oxygen diffusion to the aortic wall, creating a hypoxic environment that further weakens the vessel structure.

CLINICAL PRESENTATION

AAA are frequently described as a silent vascular disorder because most cases remain asymptomatic until the aneurysm enlarges significantly or ruptures. In many individuals, AAAs are discovered incidentally during diagnostic imaging procedures such as ultrasonography, computed tomography, or magnetic resonance imaging performed for unrelated abdominal complaints. Because aneurysms often enlarge gradually over several years without producing obvious symptoms, early detection frequently occurs during evaluation for other medical conditions. Although the majority of AAAs are asymptomatic, some patients may experience nonspecific symptoms as the aneurysm enlarges. These symptoms usually arise from progressive dilation of the aortic wall or compression of surrounding anatomical structures. Patients may report persistent or intermittent abdominal discomfort, lower back pain, flank pain, or groin pain. The pain may radiate to adjacent areas such as the lower back, legs, or scrotum depending on the structures affected. In some cases, patients may notice a pulsating sensation in the abdomen, which reflects the abnormal expansion of the aorta.

On physical examination, the most characteristic clinical finding is a pulsatile abdominal mass, typically located around or above the umbilicus. In certain cases, turbulent blood flow within the aneurysm may produce an audible bruit during auscultation. Abdominal palpation can aid in the detection of larger aneurysms and does not increase the risk of rupture when performed carefully. The sensitivity of palpation increases with aneurysm size, being relatively low in smaller aneurysms but significantly higher in those

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exceeding 5 cm in diameter. However, abdominal obesity and increased body habitus can reduce the accuracy of palpation, limiting its usefulness as a screening method.

As the aneurysm expands further, symptoms may become more pronounced and may indicate impending rupture. Patients with enlarging aneurysms often develop sudden, severe, and persistent pain in the abdomen, lower back, flank, or groin. This pain results from rapid stretching of the aortic wall or irritation of surrounding tissues. In some individuals, the expanding aneurysm may compress adjacent organs, producing symptoms such as early satiety, nausea, vomiting, urinary complaints, or venous thrombosis. Compression of nearby nerves may also produce isolated groin pain or radiating pain to the lower extremities.

In rare cases, aneurysms may produce distal embolic phenomena due to the release of thrombotic or atheromatous material from the aneurysm wall. This can lead to ischemic changes in the toes or feet, sometimes presenting as livedo reticularis or "blue toe syndrome." Additionally, thrombosis within the aneurysm may result in reduced blood flow to the lower extremities, causing symptoms such as claudication, numbness, or loss of motor function.

The most dramatic clinical presentation occurs when an aneurysm ruptures. Ruptured AAA is a life-threatening emergency and is typically characterized by sudden onset of severe abdominal or back pain accompanied by hypotension and signs of circulatory collapse. Patients may present with tachycardia, dizziness, syncope, or altered consciousness due to massive internal bleeding. In some cases, physical examination may reveal a pulsatile abdominal mass along with signs of haemorrhagic shock.

Rupture may lead to bleeding into either the retroperitoneal space or the peritoneal cavity. Retroperitoneal bleeding may produce characteristic signs such as Grey Turner sign, which appears as bruising along the flanks, and Cullen sign, which presents as periumbilical ecchymosis. Because bleeding may initially be contained within the retroperitoneal space, patients may appear relatively stable in the early stages before rapidly deteriorating. Mortality associated with ruptured AAA is extremely high, with many patients dying before reaching the hospital. [9-10]

COMPLICATIONS

Abdominal aortic aneurysm can lead to several serious and potentially life-threatening complications if left untreated. The most severe complication is aneurysm rupture, which results in massive internal bleeding into the retroperitoneal space or the peritoneal cavity. Rupture is associated with extremely high mortality, with many patients dying before reaching the hospital due to rapid hypovolemic shock and cardiovascular collapse. As the aneurysm enlarges, the weakened aortic wall becomes increasingly susceptible to rupture because of rising mechanical stress.

Another important complication is peripheral embolization, which occurs when thrombotic material or fragments of atherosclerotic plaque detach from the aneurysm wall and travel through the bloodstream to distal arteries. These emboli can obstruct small vessels in the lower extremities, leading to ischemic manifestations such as pain, discoloration of the toes, livedo reticularis, or the characteristic "blue toe syndrome." In some cases, embolization may result in tissue necrosis or gangrene if blood supply is severely compromised.

AAA may also cause acute aortic occlusion due to thrombosis within the aneurysm sac. This condition can abruptly interrupt blood flow to the lower extremities, resulting in severe leg pain, loss of pulses, numbness, and motor weakness. If not promptly treated, acute aortic occlusion can lead to irreversible limb ischemia and possible amputation. Large aneurysms may also compress surrounding structures, leading to complications such as venous thrombosis, gastrointestinal symptoms, urinary disturbances, or nerve compression.

In rare instances, the aneurysm may erode into adjacent organs and form abnormal vascular connections. Rupture into the inferior vena cava can produce an aortocaval fistula, resulting in high-output heart failure, leg swelling, abdominal bruit, and renal dysfunction. Similarly, rupture into the duodenum can create an aortoduodenal fistula, which may initially present with a small gastrointestinal bleed followed by a catastrophic hemorrhage. Because of these potentially fatal complications, early detection and timely management of AAA are essential to prevent severe morbidity and mortality. [11]

DIAGNOSIS

The diagnosis of AAA is based on clinical suspicion supported by imaging studies. In many cases, AAA is identified incidentally during imaging performed for other abdominal complaints. Clinical examination may provide initial clues, but imaging techniques are essential for confirming the diagnosis, determining aneurysm size, evaluating anatomical features, and planning appropriate treatment. [12-13]

Clinical Diagnosis: Clinical diagnosis of AAA often begins with a thorough medical history and physical examination. Patients at increased risk typically include individuals older than 60–65 years with a history of smoking, hypertension, chronic obstructive pulmonary disease, peripheral vascular disease, or a family history of aneurysm. During physical examination, the most important finding is a pulsatile abdominal mass, usually located in the upper abdomen near the umbilicus. Palpation of the abdomen may reveal an expansile pulsation corresponding to the dilated aorta. However, the sensitivity of physical examination varies widely and depends on the size of the aneurysm, the experience of the examiner, and the patient's body habitus. Obesity and abdominal distension can make palpation difficult, and therefore physical examination alone cannot reliably establish the diagnosis. Laboratory investigations do not specifically diagnose AAA but may be useful in evaluating the patient's general health and identifying associated conditions. Tests such as complete blood count, metabolic panels, and inflammatory markers may be performed to assess operative risk and detect concurrent disease processes.

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Ultrasonography: Abdominal ultrasonography is widely considered the primary diagnostic and screening tool for AAA. It is a non-invasive, safe, inexpensive, and highly accurate imaging technique with a sensitivity approaching 100% for detecting infrarenal aneurysms. Ultrasonography allows visualization of the aortic diameter and can accurately measure the maximal size of the aneurysm. It is particularly useful for screening high-risk populations and monitoring small aneurysms that do not yet require surgical intervention. Bedside ultrasonography can also detect free intraperitoneal fluid in cases of suspected rupture. However, the accuracy of ultrasound may be limited by the presence of bowel gas, obesity, or poor acoustic windows.

Computed Tomography (CT): It is one of the most sensitive imaging techniques for diagnosing AAA, with nearly 100% sensitivity. CT provides detailed cross-sectional images that allow accurate measurement of aneurysm diameter, evaluation of the extent of the aneurysm, and identification of involvement of nearby structures or branch vessels. CT angiography is particularly valuable for preoperative planning, as it provides precise information regarding the aneurysm neck, renal arteries, iliac vessels, and surrounding anatomy. This information is essential for determining whether a patient is suitable for endovascular aneurysm repair (EVAR) or open surgical repair. CT is also highly effective in detecting retroperitoneal hemorrhage in cases of suspected aneurysm rupture. Despite its advantages, CT scanning has some limitations, including exposure to ionizing radiation, the use of contrast agents that may affect renal function, and higher cost compared with ultrasonography.

Plain Radiography: Plain abdominal radiographs may occasionally reveal the outline of an aneurysm if the aortic wall is calcified. Calcification along the aneurysmal wall can produce a visible curvilinear shadow on X-ray images. However, this finding is present in fewer than half of cases, making plain radiography relatively insensitive for diagnosing AAA. Because of its limited diagnostic value, radiography is generally not recommended as a primary imaging modality for evaluating suspected aneurysms.

Magnetic Resonance Imaging (MRI): It provides high-quality images of the aorta and surrounding structures without the use of ionizing radiation. MRI is particularly useful in patients who cannot undergo CT scanning due to allergy to iodinated contrast agents or impaired kidney function. It offers excellent visualization of vascular structures and branch vessels. However, MRI is less commonly used in emergency situations because it requires a stable patient, is relatively expensive, and is not always readily available.

Angiography: Conventional angiography was historically used to evaluate AAAs, but its use has declined with the availability of advanced CT imaging techniques. Angiography may underestimate aneurysm size because thrombus within the aneurysm can create the appearance of a normal lumen. Currently, angiography is mainly used during endovascular procedures to guide stent graft placement. Because it is invasive and carries risks such as bleeding or embolization, routine use of angiography for diagnosis is not recommended.

Functional Imaging: Recent advances have introduced functional imaging techniques, such as positron emission tomography (PET) and specialized MRI methods, to evaluate biological processes involved in aneurysm progression. PET imaging using fluorodeoxyglucose (18F-FDG) can detect areas of increased metabolic activity associated with inflammation within the aneurysm wall. These techniques may help identify aneurysms that are at higher risk of rapid growth or rupture, although their clinical application remains limited.

Size Classification of AAA: Abdominal aortic aneurysms are commonly classified according to their maximum aortic diameter. A normal abdominal aorta typically measures about 2 cm in diameter, and an aneurysm is defined when the aortic diameter exceeds 3 cm or increases by more than 50% of its normal size. Aneurysms measuring 3–5 cm are generally considered moderate in size, whereas those exceeding 5–5.5 cm are classified as large and carry a significantly higher risk of rupture.

Advanced Risk Assessment: In addition to measuring aneurysm diameter, newer computational and biomechanical approaches have been developed to evaluate rupture risk more accurately. These methods analyse parameters such as peak wall stress, mean wall stress, and rupture risk indices using three-dimensional imaging data obtained from CT scans. Such techniques help estimate the mechanical forces acting on the aneurysm wall and may provide a more individualized assessment of rupture risk. However, these approaches are still evolving and are not yet widely used in routine clinical practice.

DIFFERENTIAL DIAGNOSIS

The symptoms of AAA, particularly during expansion or rupture, may resemble several other conditions. Severe abdominal or back pain may be mistaken for kidney stones, musculoskeletal back pain, or ureteric colic, leading to possible misdiagnosis. Common gastrointestinal conditions that may mimic AAA include acute gastritis, peptic ulcer disease, appendicitis, diverticulitis, gallstones, and bowel obstruction. Urinary conditions such as urinary tract infections and pyelonephritis can also present with similar abdominal or flank pain. In addition, serious conditions like myocardial infarction and mesenteric ischemia may present with abdominal discomfort and should be considered in the differential diagnosis. Therefore, imaging techniques such as ultrasound or CT scan are essential for confirming the diagnosis of AAA. [14]

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TREATMENT AND MANAGEMENT

The management of AAA is primarily determined by the size of the aneurysm, the presence of symptoms, the rate of aneurysm expansion, and the patient's overall clinical condition. Treatment strategies include conservative management with regular surveillance, control of modifiable cardiovascular risk factors, and surgical repair. Intervention is usually recommended when the aneurysm diameter exceeds 5.5 cm in men or about 5.0 cm in women, when the aneurysm enlarges rapidly (more than 0.5 cm within six months or 1 cm per year), or when the patient develops symptoms related to the aneurysm. [15-17]

Conservative Management and Surveillance: Small, asymptomatic aneurysms are generally managed through careful observation and modification of risk factors. Regular surveillance with imaging techniques such as abdominal ultrasonography or computed tomography is performed to monitor aneurysm size and detect any progression. Lifestyle changes, particularly smoking cessation, are strongly encouraged because smoking significantly accelerates aneurysm expansion and increases the risk of rupture. Control of blood pressure is also essential to reduce mechanical stress on the weakened aortic wall. Patients with aneurysms measuring 3–4 cm in diameter are typically monitored periodically, while those with aneurysms between 4 and 5.4 cm require more frequent imaging follow-up, often every 6 to 12 months. Medical management focuses on treating associated cardiovascular risk factors such as hypertension, dyslipidemia, and atherosclerosis. Although no pharmacological therapy has been proven to directly slow aneurysm growth, medications such as statins and antihypertensive agents may reduce cardiovascular morbidity and improve overall patient outcomes.

Surgical Management: Surgical intervention is recommended when the risk of aneurysm rupture exceeds the risk associated with surgery. Two principal surgical techniques are used for AAA repair: open surgical repair and endovascular aneurysm repair (EVAR).

- Open Surgical Repair: Open surgical repair has long been considered the standard and definitive treatment for AAA. In this procedure, a large abdominal or retroperitoneal incision is made to expose the affected portion of the aorta. The aneurysmal segment is then replaced with a synthetic vascular graft, commonly composed of Dacron or polytetrafluoroethylene (PTFE). During the procedure, the aorta is temporarily clamped to control blood flow while the graft is positioned and secured. Although open repair provides durable long-term results, it is a major surgical operation associated with significant recovery time. Patients usually require intensive postoperative monitoring, and complete recovery may take several weeks to months.
- Endovascular Aneurysm Repair (EVAR): Endovascular aneurysm repair is a less invasive alternative to open surgery and has become widely used in suitable patients. In this technique, a stent graft is introduced through the femoral arteries using a catheter-based approach and positioned within the aneurysmal segment of the aorta. The stent graft redirects blood flow through the graft lumen, thereby reducing pressure on the weakened arterial wall and lowering the risk of aneurysm rupture. EVAR offers several advantages, including reduced perioperative mortality, shorter hospital stay, decreased intensive care requirements, and faster postoperative recovery. However, the procedure is not appropriate for all patients because its feasibility depends on the anatomical characteristics of the aneurysm. Furthermore, individuals who undergo EVAR require lifelong imaging surveillance to detect complications such as endoleaks, graft migration, or device failure.

Management of Ruptured AAA: A ruptured AAA represents a life-threatening emergency that requires immediate surgical intervention. Initial management focuses on rapid resuscitation, stabilization of the patient, and urgent surgical repair using either open surgery or EVAR, depending on anatomical suitability and patient stability. Controlled or permissive hypotension is often maintained during early management to minimize bleeding until definitive surgical control of the aorta is achieved.

Follow-Up and Long-Term Care: Patients who undergo AAA repair require long-term follow-up to evaluate graft integrity and detect potential complications. Periodic imaging with ultrasound or CT scanning is performed to ensure that the aneurysm sac remains stable and that no leakage occurs around the graft. Long-term management also includes strict control of cardiovascular risk factors, particularly smoking cessation, blood pressure management, and lipid control, to improve overall prognosis and reduce the risk of future vascular complications.

PROGNOSIS

The prognosis of abdominal aortic aneurysm largely depends on whether the aneurysm ruptures and the timing of surgical intervention. Once rupture occurs, the prognosis is extremely poor. More than 50% of patients die before reaching the hospital, and mortality remains high even among those who receive emergency treatment. Postoperative mortality for ruptured AAA is typically greater than 40%, and survival decreases rapidly with ongoing bleeding and shock. Factors associated with higher mortality include advanced age (over 80 years), female sex, preoperative cardiac arrest, severe blood loss, and the need for extensive transfusion.

In contrast, the prognosis is significantly better when AAA is detected and repaired electively before rupture. Elective surgical repair has a much lower mortality rate, generally between 1% and 6%, and approximately 70% of patients survive for at least five years after repair. Long-term survival after successful repair is mainly influenced by coexisting medical conditions such as chronic obstructive pulmonary disease, cardiovascular disease, and peripheral vascular disease.

Traditionally, the risk of rupture has been assessed based on the maximum diameter of the aneurysm, with aneurysms larger than 5.5 cm considered at high risk. However, studies have shown that some smaller aneurysms may still rupture while some larger aneurysms remain stable. Therefore, newer approaches to risk assessment are being explored, including evaluation of aneurysm wall stress, growth rate, geometric characteristics, and biomechanical modelling using imaging data. [18-19]

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CONCLUSION

AAA is a potentially life-threatening condition that often remains asymptomatic until rupture, making early detection and monitoring essential. Timely intervention and risk factor management significantly improve outcomes and reduce the high mortality associated with rupture.

REFERENCES

1. *Tarafdar, S. A., & Gannon, M. X. (2017). Abdominal aortic aneurysm. InnovAiT Education and Inspiration for General Practice, 10(5), 290–296. https://doi.org/10.1177/1755738017693654*
2. *Shaw, P. M., Loree, J., & Oropallo, A. (2025, January 19). Abdominal aortic aneurysm. StatPearls - NCBI Bookshelf. https://www.ncbi.nlm.nih.gov/books/NBK470237/*
3. *Kessler, V., Klopf, J., Eilenberg, W., Neumayer, C., & Brostjan, C. (2022). AAA revisited: A comprehensive review of risk factors, management, and hallmarks of pathogenesis. Biomedicines, 10(1), 94. https://doi.org/10.3390/biomedicines10010094*
4. *Aggarwal, S., Qamar, A., Sharma, V., & Sharma, A. (2011). Abdominal aortic aneurysm: A comprehensive review. Experimental and clinical cardiology, 16(1), 11–15.*
5. *Powell, J. T., Sweeting, M. J., Brown, L. C., Gotensparre, S. M., Fowkes, F. G., & Thompson, S. G. (2011). Systematic review and meta-analysis of growth rates of small abdominal aortic aneurysms. The British journal of surgery, 98(5), 609–618. https://doi.org/10.1002/bjs.7465*
6. *Kuivaniemi, H., Ryer, E. J., Elmore, J. R., & Tromp, G. (2015). Understanding the pathogenesis of abdominal aortic aneurysms. Expert review of cardiovascular therapy, 13(9), 975–987. https://doi.org/10.1586/14779072.2015.1074861*
7. *Patel, M., Braga, D., Money, B., Pirela, A., Zybulewski, A., Olivieri, B., & Beasley, R. (2021). Pathogenesis of Abdominal Aortic Aneurysm. In Cardiovascular Risk Factors in Pathology. IntechOpen. https://doi.org/10.5772/intechopen.91670*
8. *Khoury, M. K., Stranz, A. R., & Liu, B. (2020). Pathophysiology of Aortic Aneurysms: Insights from Animal Studies. Cardiology and Cardiovascular Medicine, 04(04), 498–514. https://doi.org/10.26502/fccm.92920146*
9. *Kumar, Y., Hooda, K., Li, S., Goyal, P., Gupta, N., & Adeb, M. (2017). Abdominal aortic aneurysm: pictorial review of common appearances and complications. Annals of translational medicine, 5(12), 256. https://doi.org/10.21037/atm.2017.04.32*
10. *Anagnostakos, J., & Lal, B. K. (2021). Abdominal aortic aneurysms. Progress in Cardiovascular Diseases, 65, 34–43. https://doi.org/10.1016/j.pcad.2021.03.009*
11. *Al-Zoubi, N. A., Mahafdah, M. R., & Albawaih, O. (2021). Chronic Contained Rupture of the Abdominal Aortic Aneurysm. Open access emergency medicine : OAEM, 13, 439–443. https://doi.org/10.2147/OAEM.S327922*
12. *Mandika, S. (2011). Diagnosis of Aortic Aneurysm. In Diagnosis, Screening and Treatment of Abdominal, Thoracoabdominal and Thoracic Aortic Aneurysms. InTech. https://doi.org/10.5772/17503*
13. *Schwartz, S. A., Taljanovic, M. S., Smyth, S., O'Brien, M. J., & Rogers, L. F. (2007). CT findings of rupture, impending rupture, and contained rupture of abdominal aortic aneurysms. AJR. American journal of roentgenology, 188(1), W57–W62. https://doi.org/10.2214/AJR.05.1554*
14. *Azhar, B., Patel, S. R., Holt, P. J., Hinchliffe, R. J., Thompson, M. M., & Karthikesalingam, A. (2014). Misdiagnosis of ruptured abdominal aortic aneurysm: systematic review and meta-analysis. Journal of endovascular therapy : an official journal of the International Society of Endovascular Specialists, 21(4), 568–575. https://doi.org/10.1583/13-4626MR.1*
15. *Moll, F., Powell, J., Fraedrich, G., Verzini, F., Haulon, S., Waltham, M., Van Herwaarden, J., Holt, P., Van Keulen, J., Rantner, B., Schlösser, F., Setacci, F., & Ricco, J. (2011). Management of Abdominal Aortic Aneurysms Clinical practice Guidelines of the European Society for Vascular Surgery. European Journal of Vascular and Endovascular Surgery, 41, S1–S58. https://doi.org/10.1016/j.ejvs.2010.09.011*
16. *Metcalfe, D., Holt, P. J., & Thompson, M. M. (2011). The management of abdominal aortic aneurysms. BMJ (Clinical research ed.), 342, d1384. https://doi.org/10.1136/bmj.d1384*
17. *Hallett J. W., Jr (2000). Management of abdominal aortic aneurysms. Mayo Clinic proceedings, 75(4), 395–399. https://doi.org/10.4065/75.4.395*
18. *Johansen, K., Kohler, T. R., Nicholls, S. C., Zierler, R. E., Clowes, A. W., & Kazmers, A. (1991). Ruptured abdominal aortic aneurysm: the Harborview experience. Journal of vascular surgery, 13(2), 240–247.*
19. *Schermerhorn, M. L., Buck, D. B., O'Malley, A. J., Curran, T., McCallum, J. C., Darling, J., & Landon, B. E. (2015). Long-Term Outcomes of Abdominal Aortic Aneurysm in the Medicare Population. The New England journal of medicine, 373(4), 328–338. https://doi.org/10.1056/NEJMoa1405778*