NEUROVASCULAR PATHOPHYSIOLOGY AND CONTEMPORARY MANAGEMENT OF INTRACRANIAL ANEURYSMS: A COMPREHENSIVE CLINICAL REVIEW

Thalapalli Sangeeth Kumar, Syeda Nishat Fathima
Department of Pharmacology, Jayamukhi College of Pharmacy, Narsampet, Warangal-506332, Telangana, India

Abstract:

Cerebral aneurysms are localized dilatations of cerebral arteries caused by structural weakness of the vessel wall, affecting approximately 3–5% of the global population. Their development is driven by a multifactorial interplay of hemodynamic stress, endothelial dysfunction, chronic inflammation, vascular smooth muscle cell remodelling, genetic susceptibility, and modifiable risk factors such as hypertension and smoking. Although many aneurysms remain asymptomatic, rupture can result in aneurysmal subarachnoid haemorrhage, a life-threatening neurological emergency associated with high morbidity and mortality. Diagnosis relies on multimodal neuroimaging, including computed tomography, CT angiography, magnetic resonance angiography, and digital subtraction angiography, the diagnostic gold standard. Management strategies encompass microsurgical clipping, endovascular coiling, adjunctive medical therapy, and comprehensive rehabilitation, with treatment tailored according to aneurysm characteristics and patient factors. This review provides a concise overview of the classification, epidemiology, pathophysiology, clinical manifestations, diagnostic approaches, contemporary management, and prognosis of cerebral aneurysms, highlighting current evidence to support optimal neurovascular care and clinical decision-making.

Keywords: Intracranial aneurysm; Cerebral aneurysm; Subarachnoid haemorrhage; Endovascular therapy; Microsurgical clipping; Flow diversion; Neuroimaging; Vascular biology; Inflammation; Precision medicine.

INTRODUCTION:

An intracranial aneurysm, commonly referred to as a cerebral aneurysm, is a cerebrovascular abnormality characterized by a localized dilation of a cerebral artery resulting from structural weakness of the vessel wall. Cerebral aneurysms are classified based on size, morphology (shape), rupture status, location, and etiology, each of which has important implications for prognosis and management. [1]

- Cerebral aneurysms are commonly categorized by size, which is a critical determinant of rupture risk. Small aneurysms typically measure less than 10–15 mm in diameter, while larger aneurysms are subdivided into large (10–25 mm), giant (>25 mm), and super-giant (>50 mm) lesions. The likelihood of rupture increases significantly with size, particularly in aneurysms exceeding 10 mm, making size-based classification essential for clinical decision-making and treatment planning.
- Based on morphology, aneurysms are divided into saccular and non-saccular types. Saccular aneurysms, also known as berry aneurysms, are the most common form and appear as rounded outpouchings arising from arterial bifurcations. These are frequently associated with risk factors such as hypertension, smoking, connective tissue disorders, and conditions like Autosomal Dominant Polycystic Kidney Disease. In contrast, fusiform aneurysms involve circumferential dilation of the entire vessel wall and are often

related to atherosclerosis and chronic vascular degeneration. These aneurysms have a relatively lower but continuous risk of rupture. Other non-saccular variants include dissecting aneurysms, which result from a tear in the arterial wall, and dolichoectatic aneurysms, characterized by elongation and tortuosity of arteries.A distinct subtype includes microaneurysms, particularly the Charcot–Bouchard Aneurysm, which occur in small penetrating vessels such as the lenticulostriate arteries of the basal ganglia. These are strongly associated with chronic hypertension and are a well-recognized cause of intracerebral hemorrhage.

- Clinically, aneurysms are also classified based on rupture status into ruptured and unruptured aneurysms. Ruptured aneurysms typically present with subarachnoid hemorrhage and require urgent intervention, whereas unruptured aneurysms may be incidentally detected and managed based on risk assessment.
- Another important classification is based on anatomical location within the cerebral circulation. Approximately 90% of aneurysms occur in the anterior circulation (e.g., anterior communicating artery and middle cerebral artery), while the remaining 10% are found in the posterior circulation, including the basilar and vertebral arteries. Posterior circulation aneurysms are generally associated with a higher risk of complications and poorer outcomes.
- Finally, aneurysms may also be categorized according to etiology, including atherosclerotic, mycotic (infectious), traumatic, congenital, and hemodynamic (flow-related) origins. In addition, angioarchitectural features such as neck size and dome-to-neck ratio are crucial in determining suitability for endovascular or surgical treatment.

EPIDEMIOLOGY:

Cerebral aneurysms are relatively common vascular abnormalities, with a global prevalence of approximately 3–5% in the general population. Autopsy and imaging studies suggest that prevalence may vary depending on detection methods, ranging from 1% to 10%. In the United States, this corresponds to nearly 10–12 million individuals, with an annual incidence of about 1 per 10,000 persons, translating to approximately 27,000 new cases each year. Despite advances in medical imaging, the overall frequency of aneurysms has remained relatively stable over time.[2,3]

The occurrence of cerebral aneurysms shows a clear age-related pattern, most commonly affecting individuals between 30 and 60 years, with peak clinical manifestation around 55–60 years of age. These aneurysms are rare in pediatric populations, accounting for only about 2% of cases. Gender distribution is generally equal in younger individuals; however, after the age of 50, there is a female predominance, with ratios approaching 2:1. This shift is thought to be related to declining estrogen levels, which reduce collagen content and weaken vascular integrity.

The incidence of aneurysmal subarachnoid hemorrhage (SAH), the most severe complication, is estimated at 10–12 cases per 100,000 individuals annually worldwide. Certain populations, particularly those in Finland and Japan, exhibit higher rupture rates, although this does not necessarily reflect a higher prevalence of aneurysms but rather increased susceptibility to rupture.

From an anatomical perspective, aneurysms most commonly occur in the anterior circulation, particularly at the anterior communicating artery and middle cerebral artery. Gender-specific patterns have also been observed, with men more frequently affected in the anterior cerebral regions, while women more commonly develop aneurysms at the internal carotid–posterior communicating artery junction. Giant aneurysms are significantly more common in women.

Aneurysmal SAH contributes to approximately 0.4–0.6% of all-cause mortality, with substantial clinical impact. Among patients with rupture, mortality rates approach 20–50%, and an additional 30–40% of survivors experience significant neurological morbidity. Prognosis worsens with increasing age, female gender, and severity of hemorrhage.

AETIOLOGY:

Intracranial aneurysms are multifactorial vascular abnormalities that arise due to a complex interaction of acquired, genetic, and structural factors affecting the integrity of cerebral arterial walls. Most aneurysms are considered acquired lesions and are commonly associated with modifiable risk factors such as hypertension, cigarette smoking, chronic alcohol consumption, and obesity. Persistent hypertension contributes to increased hemodynamic stress on arterial walls, leading to progressive weakening and dilation. Similarly, smoking induces endothelial dysfunction, oxidative stress, and inflammatory changes that compromise vascular integrity. Substance abuse, particularly cocaine and amphetamines, has also been strongly implicated due to its acute hypertensive effects and vasoconstrictive properties. Additional acquired causes include head trauma and infections, such as mycotic aneurysms resulting from bacterial endocarditis, which can directly damage vessel walls. [4]

Genetic predisposition plays a significant role in the development of intracranial aneurysms, particularly in individuals with a positive family history. The risk is notably higher among first-degree relatives, suggesting a hereditary component. Several inherited disorders affecting connective tissue and vascular structure are strongly associated with aneurysm formation. These include Autosomal Dominant Polycystic Kidney Disease, Marfan Syndrome, and Ehlers-Danlos Syndrome Type IV, all of which are characterized by defects in collagen and elastin, leading to reduced tensile strength of blood vessels. Other conditions such as Neurofibromatosis Type I and Hereditary Hemorrhagic Telangiectasia further contribute to vascular fragility and abnormal vessel formation.

Structural and vascular abnormalities also significantly influence aneurysm development. Conditions such as Arteriovenous Malformation increase blood flow and shear stress on vessel walls, predisposing to aneurysm formation at sites of turbulence. Similarly, Coarctation of the Aorta alters systemic hemodynamics, while disorders like Fibromuscular Dysplasia and Moyamoya Disease contribute to abnormal arterial architecture and compensatory vascular changes. Inflammatory conditions such as cerebral vasculitis further exacerbate endothelial injury and vessel wall weakening.

At the molecular level, genetic studies have identified alterations in genes regulating extracellular matrix composition and vascular tone, including elastin, collagen type I A2, endothelial nitric oxide synthase, and endothelin receptor pathways. Multiple chromosomal loci, such as 1p34–36 and 19q13, have also been implicated, highlighting the polygenic nature of this condition.

CLINICAL PRESENTATION:

The clinical presentation of cerebral aneurysms is highly variable and depends on whether the aneurysm is unruptured, leaking, or ruptured, as well as its size, location, and associated complications. The spectrum ranges from completely asymptomatic cases to sudden catastrophic neurological deterioration. [5,6]

- Unruptured Aneurysms: Most unruptured intracranial aneurysms remain clinically silent and are often discovered incidentally during imaging for unrelated conditions. When symptoms occur, they are typically due to the mass effect of the aneurysm on adjacent neural structures. Patients may present with chronic or intermittent headaches, dizziness, or vague neurological complaints. Larger aneurysms can compress cranial nerves, particularly the oculomotor nerve, leading to ptosis, diplopia, and pupillary dilation. Visual disturbances such as blurred vision or visual field defects may occur if the optic pathways are involved. In some cases, focal neurological deficits including weakness, sensory loss, or speech disturbances may arise due to compression or distal embolization.
- Sentinel (Warning) Leak: A subset of patients experiences a “sentinel headache” days to weeks before a major rupture. This headache is typically sudden and severe but less intense than the classic rupture

headache. It represents a minor leak or small hemorrhage from the aneurysm and serves as an important warning sign. Failure to recognize this stage may result in missed opportunities for early intervention.

- Ruptured Aneurysms and Subarachnoid Hemorrhage: Rupture of a cerebral aneurysm most commonly leads to subarachnoid hemorrhage (SAH), a life-threatening emergency. The hallmark symptom is a sudden, severe "thunderclap headache", often described as the worst headache of the patient's life. The onset is abrupt and may occur during physical exertion or emotional stress. The headache may last from hours to days and is sometimes localized to the side of the aneurysm.This may be accompanied by nausea, vomiting, neck stiffness, photophobia, and varying levels of altered consciousness. A brief loss of consciousness is common due to a rapid rise in intracranial pressure.

Neurological and Systemic Manifestations: Neurological findings depend on the site and extent of hemorrhage. Patients may exhibit focal deficits such as hemiparesis, hemisensory loss, aphasia, or visual field defects due to associated intracerebral hemorrhage or ischemia. Cranial nerve palsies, especially involving the third nerve, are characteristic of aneurysms in specific locations. Signs of meningeal irritation, including neck rigidity and photophobia, are typical of SAH. Additionally, autonomic disturbances such as fever, sweating, and cardiac arrhythmias may occur due to irritation of the hypothalamus and brainstem.

Atypical and Severe Presentations: In some cases, aneurysmal rupture may present with intraparenchymal, intraventricular, or subdural hemorrhage, leading to focal neurological deficits rather than classic SAH features. Giant aneurysms may cause progressive neurological deterioration due to mass effect. Severe cases may present with stupor, coma, or decerebrate posturing, indicating extensive brain injury and poor prognosis.

Grading: To assess prognosis and guide management, clinicians commonly use the Hunt and Hess Grading System, which categorizes patients based on their neurological condition at presentation. This grading system ranges from Grade I to Grade V. Grade I includes patients with mild headache and minimal neck stiffness. Grade II is characterized by severe headache and nuchal rigidity without significant neurological deficits except possible cranial nerve palsy. Grade III involves drowsiness, confusion, or mild focal neurological deficits. Grade IV represents a more severe condition with stupor and moderate to severe hemiparesis. Grade V denotes the most critical stage, with coma and decerebrate posturing, and is associated with a very poor prognosis.

PATHOPHYSIOLOGY:

The pathophysiology of cerebral aneurysms is a complex and multifactorial process involving structural weakness of the arterial wall, abnormal hemodynamic forces, endothelial dysfunction, and chronic inflammation. These mechanisms interact over time, ultimately leading to aneurysm formation, growth, and rupture. [7-9]

At the core of aneurysm development is structural alteration of the cerebrovascular wall. Normally, cerebral arteries consist of three layers: intima, media, and adventitia.The fundamental pathological feature is the disruption of normal vascular wall architecture, particularly involving degeneration of the internal elastic lamina and thinning of the tunica media. Loss of smooth muscle cells and fragmentation of elastic fibers significantly reduce the tensile strength and elasticity of the vessel wall, predisposing it to dilation under continuous hemodynamic stress. These changes are especially prominent at arterial bifurcations, such as those within the Circle of Willis, where turbulent blood flow and structural discontinuities increase susceptibility to aneurysm formation.

Hemodynamic factors play a critical role in both the initiation and progression of aneurysms. Both high and low wall shear stress (WSS) exert critical but distinct influences on the formation, progression, and rupture of cerebral aneurysms. Although the exact mechanisms remain incompletely understood, current evidence

suggests that these opposing hemodynamic forces trigger different pathological pathways. Low WSS is commonly associated with the growth and rupture of larger aneurysms through sustained inflammatory processes. In contrast, high WSS is believed to promote the initiation and rupture of smaller aneurysms via direct injury to the vessel wall, often referred to as a mural response. These hemodynamic disturbances are further exacerbated by systemic risk factors such as hypertension, cigarette smoking, female sex, genetic predisposition, infections, and trauma, all of which contribute to vascular wall vulnerability.Hemodynamic stress leads to endothelial dysfunction and structural damage within the arterial wall, initiating a cascade of inflammatory responses. This process involves the recruitment and activation of immune cells, including T lymphocytes, macrophages, and mast cells, which infiltrate the vessel wall and release a wide range of inflammatory mediators. Key mediators include interleukin-1β, interleukin-6, tumor necrosis factor-α (TNF-α), matrix metalloproteinases (MMP-1, MMP-2, MMP-9), prostaglandin E2, components of the complement system, reactive oxygen species (ROS), and angiotensin II. These substances collectively promote degradation of the extracellular matrix, oxidative stress, and amplification of the inflammatory cascade, ultimately weakening the vascular structure.

A crucial pathological event in aneurysm development is the phenotypic transformation of vascular smooth muscle cells. Under normal conditions, VSMCs in the tunica media maintain a contractile phenotype that supports vascular tone and structural integrity. However, in response to chronic hemodynamic and inflammatory stimuli, these cells migrate into the tunica intima and undergo a functional shift to a pro-inflammatory and synthetic phenotype.This transformation leads to reduced contractility, increased secretion of inflammatory mediators, and abnormal synthesis of extracellular matrix components, particularly collagen.Consequently, the arterial wall undergoes fibrotic remodeling, characterized by disorganized collagen deposition, depletion of functional smooth muscle cells, and progressive thinning of the vessel wall. The degradation of elastin and collagen, combined with impaired repair mechanisms, significantly compromises the mechanical stability of the artery. This structural weakening facilitates aneurysm formation and increases susceptibility to rupture under continuous hemodynamic stress.

Although genetic factors are believed to contribute to aneurysm susceptibility, no single specific gene locus has been definitively identified, highlighting the multifactorial and polygenic nature of the disease. Overall, the interplay between abnormal shear stress, inflammation, and vascular remodeling forms the cornerstone of cerebral aneurysm pathophysiology, providing important insights into potential therapeutic targets and preventive strategies.

Special forms of aneurysms arise through distinct mechanisms. Infectious (mycotic) aneurysms result from septic emboli, often originating from bacterial endocarditis, leading to direct vessel wall destruction. Traumatic aneurysms occur due to vessel injury from head trauma, while neoplastic aneurysms may develop from tumor emboli. Additionally, dolichoectatic aneurysms are often associated with atherosclerosis and cause vessel elongation and tortuosity rather than focal outpouching.

DIAGNOSIS:

The diagnosis of intracranial aneurysms is a structured, multimodal process that integrates clinical suspicion, neuroimaging, and cerebrospinal fluid (CSF) analysis, with the primary objective of detecting the aneurysm and determining rupture status. This distinction is critical, as ruptured aneurysms causing subarachnoid hemorrhage (SAH) constitute a neurological emergency requiring immediate intervention. [10,11]

In patients presenting with acute severe headache suggestive of SAH, noncontrast computed tomography (CT) of the brain is the initial imaging modality of choice. CT is highly sensitive for detecting acute hemorrhage, particularly within the first few hours of symptom onset, where sensitivity approaches 100%. The modality can identify hyperdense blood within the basal cisterns, ventricles, or brain parenchyma. However, the sensitivity of CT declines over time, falling significantly after the first 24 hours, which

necessitates further diagnostic evaluation when clinical suspicion persists.When CT findings are inconclusive, lumbar puncture (LP) serves as the gold standard for confirming SAH. CSF analysis typically demonstrates elevated red blood cell counts that do not diminish across sequential tubes, along with increased opening pressure. The presence of xanthochromia, a yellowish discoloration of CSF resulting from hemoglobin degradation, is a highly sensitive and specific indicator when assessed at least 12 hours after hemorrhage onset. This step is particularly important in cases of delayed presentation or minor leaks.

Following confirmation of hemorrhage, identification and characterization of the aneurysm are achieved using vascular imaging techniques. Computed tomographic angiography (CTA) is widely utilized due to its rapid acquisition, high sensitivity, and ability to provide detailed three-dimensional visualization of aneurysm morphology, including size, neck configuration, and relationship to parent vessels. CTA is especially effective in detecting aneurysms larger than 3 mm and is often used in emergency settings.

Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) offer noninvasive alternatives, particularly useful in evaluating unruptured aneurysms and in screening high-risk populations. Advanced MRI sequences, such as fluid-attenuated inversion recovery (FLAIR), can detect subarachnoid blood, while time-of-flight MRA provides detailed vascular imaging without the need for contrast. However, MRI may be less practical in unstable patients and may have reduced sensitivity for detecting very small aneurysms or calcifications.

The gold standard for definitive diagnosis remains digital subtraction angiography (DSA). This invasive technique involves catheter-based contrast injection and provides superior spatial resolution, allowing precise evaluation of aneurysm anatomy, including small lesions, multiple aneurysms, and vascular variants. DSA is particularly valuable for treatment planning and can detect features such as daughter sacs or vasospasm that may not be evident on noninvasive imaging.

Additional diagnostic modalities may complement evaluation in specific clinical scenarios. Transcranial Doppler ultrasonography is useful for detecting and monitoring vasospasm following SAH, while advanced imaging techniques such as CT perfusion, positron emission tomography (PET), or single-photon emission computed tomography (SPECT) may assess cerebral blood flow and ischemia. Laboratory investigations, including complete blood count, coagulation profile, serum electrolytes, and arterial blood gases, assist in evaluating systemic status and identifying complications.

DIFFERENTIAL DIAGNOSIS:

The evaluation of cerebral aneurysm requires differentiation from several conditions that present with similar symptoms, particularly sudden headache and neurological deficits.Key differentials include Arteriovenous Malformation, which can cause intracranial hemorrhage, and Cerebral Venous Thrombosis, presenting with headache and seizures. Vascular disorders such as carotid or vertebral artery dissection and Fibromuscular Dysplasia may mimic aneurysmal symptoms.Neurological conditions like stroke (ischemic or hemorrhagic) and Moyamoya Disease should also be considered. Sudden severe headache may be seen in Pituitary Apoplexy, while primary headache disorders such as migraine and cluster headache can resemble milder presentations.Rare causes include Vein of Galen Malformation and Primary Angiitis of the Central Nervous System. Accurate diagnosis relies on clinical assessment and imaging. [12]

TREATMENT:

The treatment of intracranial aneurysms represents a critical aspect of modern neurovascular care, requiring a multidisciplinary, patient-specific approach that integrates advances in neurosurgery, interventional neuroradiology, and intensive care medicine. The overarching goal is to prevent rupture in unruptured aneurysms or rebleeding in ruptured cases, while preserving cerebral perfusion and minimizing neurological

morbidity. Treatment strategies broadly include emergency stabilization, definitive aneurysm occlusion (surgical or endovascular), adjunctive medical therapy, and long-term follow-up and rehabilitation. [13-16]

1. Initial Stabilization and Emergency Management: In patients presenting with ruptured aneurysms, typically manifesting as subarachnoid hemorrhage (SAH), immediate management focuses on airway, breathing, and circulation. Patients with decreased consciousness may require intubation and mechanical ventilation. Hemodynamic stabilization is crucial, with careful regulation of blood pressure to prevent rebleeding while maintaining adequate cerebral perfusion. Management of intracranial pressure is essential. Measures include head elevation, osmotic diuretics such as mannitol, and controlled ventilation. In severe cases, external ventricular drainage may be required to relieve hydrocephalus. Intensive care monitoring is mandatory due to the high risk of complications such as vasospasm, electrolyte imbalance, and cardiac arrhythmias. Pharmacological therapy plays a supportive role. The calcium channel blocker Nimodipine is routinely administered to reduce the risk of delayed cerebral ischemia caused by vasospasm. Antihypertensive agents, anticonvulsants (when indicated), analgesics, and antiemetics are also used as part of comprehensive care.

2. Definitive Treatment: It is directed toward the complete and permanent exclusion of the aneurysm from the cerebral circulation, thereby preventing rupture or rebleeding while preserving normal blood flow in the parent vessel and adjacent perforating arteries. This objective can be achieved through two principal modalities: microsurgical clipping and endovascular therapy, both of which have evolved significantly with advances in technology and technique. The choice of treatment is individualized, taking into account aneurysm size, morphology, location, rupture status, and patient-specific factors such as age, comorbidities, and neurological condition.Microsurgical clipping remains a cornerstone of definitive aneurysm management, particularly for anatomically accessible and complex aneurysms. This technique involves performing a craniotomy to directly visualize the aneurysm and placing a metallic clip across its neck to isolate it from the circulation. Clipping provides immediate and durable occlusion, with very low recurrence rates, making it especially suitable for younger patients and those with wide-neck aneurysms or lesions located in the middle cerebral artery. The procedure allows direct inspection of surrounding vessels and neural structures, ensuring preservation of normal circulation. However, it is an invasive intervention associated with risks such as intraoperative rupture, infection, and neurological deficits, and it requires a longer recovery period compared to minimally invasive techniques. Endovascular therapy has emerged as a less invasive and widely preferred alternative, particularly in elderly patients or those with high surgical risk. The most commonly employed method is endovascular coiling, in which platinum coils are delivered into the aneurysm sac via a catheter navigated through the vascular system. These coils induce thrombosis within the aneurysm, effectively occluding it and preventing further blood flow. Endovascular coiling offers significant advantages, including reduced procedural morbidity, shorter hospital stay, and faster recovery. However, it is associated with a higher risk of aneurysm recurrence and recanalization, necessitating long-term imaging follow-up and, in some cases, repeat intervention. To address the limitations of conventional coiling, several advanced endovascular techniques have been developed. Stent-assisted coiling provides structural support for wide-neck aneurysms by placing a stent within the parent artery, preventing coil displacement. Similarly, balloon-assisted coiling involves temporary inflation of a balloon across the aneurysm neck during coil deployment to enhance stability. Another major advancement is the use of flow diverter devices, which are specialized stents that redirect blood flow away from the aneurysm, promoting gradual thrombosis and endothelialization of the vessel wall. These devices are particularly effective for large, giant, or fusiform aneurysms that are difficult to treat with traditional methods. However, they require prolonged antiplatelet therapy, which may increase the risk of hemorrhagic complications.The selection between microsurgical and endovascular approaches is influenced by multiple factors. Clipping is generally favored for younger patients, wide-neck aneurysms, and lesions located in surgically accessible regions, whereas endovascular therapy is preferred for posterior circulation aneurysms, elderly patients, and those

with significant comorbidities. Despite differences in approach, both modalities aim to achieve complete aneurysm occlusion, and outcomes are often comparable when appropriately selected. Timing of definitive treatment is crucial, particularly in cases of ruptured aneurysms. Early intervention, typically within 24 to 48 hours, is recommended to minimize the risk of rebleeding, which is highest during the initial days following rupture. However, in patients with poor clinical status or complications such as vasospasm or cerebral edema, delayed intervention may be considered after stabilization.

3. **Adjunctive Medical Therapy:** It plays a crucial role in the comprehensive management of cerebral aneurysms, particularly in the perioperative and post-rupture period. While definitive treatment aims to secure the aneurysm, medical management focuses on stabilizing the patient, preventing complications, and optimizing neurological recovery. These interventions are typically carried out in an intensive care setting with continuous neurological and hemodynamic monitoring.

- One of the most critical aspects of medical therapy is the management of vasospasm, a common and potentially devastating complication following subarachnoid hemorrhage (SAH). Vasospasm usually occurs between days 3 and 21 after hemorrhage and can lead to delayed cerebral ischemia. The calcium channel blocker Nimodipine is routinely administered, as it has been shown to improve neurological outcomes by reducing the severity of ischemic complications. In addition, induced hypertension is often employed to maintain adequate cerebral perfusion in patients with vasospasm.
- Blood pressure control is another essential component. Before aneurysm occlusion, hypertension is carefully controlled to reduce the risk of rebleeding, while avoiding excessive lowering that may compromise cerebral perfusion. After securing the aneurysm, blood pressure targets may be adjusted to support cerebral blood flow, especially in the presence of vasospasm.
- Seizure management is also important, as seizures may occur following aneurysmal rupture. Antiepileptic drugs are administered in patients with clinical seizures or those at high risk. Routine prophylactic use remains controversial but may be considered in selected cases.
- Pain control and sedation are necessary to reduce patient distress and prevent increases in intracranial pressure. Analgesics and antiemetics are commonly used to manage headache, nausea, and vomiting. Electrolyte balance, particularly sodium levels, must be closely monitored, as hyponatremia is frequently observed due to conditions such as syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt wasting.
- Additional supportive measures include deep vein thrombosis prophylaxis, stress ulcer prevention, and maintenance of adequate nutrition. In cases of hydrocephalus, external ventricular drainage may be required to relieve intracranial pressure. Continuous cardiac monitoring is also necessary, as SAH can be associated with arrhythmias and myocardial dysfunction.

4. **Long-Term Follow-up:** It is essential after both surgical and endovascular treatment to ensure durable aneurysm occlusion and early detection of recurrence or new aneurysm formation. The need for follow-up is particularly important in patients treated with endovascular techniques, as these are associated with higher rates of recanalization. Follow-up typically involves serial imaging studies, including CT angiography (CTA), magnetic resonance angiography (MRA), or conventional angiography. The first follow-up study is usually performed within 6 to 12 months after treatment, with subsequent imaging at regular intervals depending on the clinical scenario. Noninvasive imaging modalities are increasingly preferred for routine surveillance.Patients with a history of aneurysm are also at risk of developing de novo aneurysms, particularly those with genetic predisposition or multiple aneurysms. Therefore, long-term monitoring is recommended, especially in high-risk individuals.Equally important is the management of modifiable risk factors. Strict control of hypertension, smoking cessation, moderation of alcohol intake, and management of comorbid conditions such as diabetes and hyperlipidemia are essential to reduce the risk of recurrence or new aneurysm formation.

5. Rehabilitation: Recovery following cerebral aneurysm rupture or treatment often requires comprehensive rehabilitation, particularly in patients who have experienced neurological deficits. Rehabilitation aims to restore functional independence, improve quality of life, and facilitate reintegration into daily activities.

- **Physical therapy** is fundamental in patients with motor deficits, focusing on improving strength, coordination, and mobility. Techniques include gait training, balance exercises, and muscle strengthening programs. Early mobilization is encouraged to prevent complications such as muscle atrophy and deep vein thrombosis.
- **Occupational therapy** addresses difficulties in performing activities of daily living, such as dressing, eating, and personal hygiene. It also helps patients adapt to physical limitations through assistive devices and environmental modifications.
- **Speech and language therapy** is essential for patients with aphasia, dysarthria, or cognitive-communication impairments. These interventions focus on improving speech, comprehension, and cognitive functions such as memory and attention.
- Neuropsychological support is also important, as many patients experience **cognitive deficits, emotional disturbances, anxiety, or depression** following aneurysm rupture. Counseling and cognitive rehabilitation programs help patients cope with these challenges and improve mental well-being.
- Family education and support play a significant role in rehabilitation, as caregivers are integral to the recovery process. Long-term outcomes depend on multiple factors, including the severity of initial hemorrhage, timing of treatment, and effectiveness of rehabilitation interventions.

PROGNOSIS:

The prognosis of intracranial aneurysms varies widely and depends on whether the aneurysm is unruptured or ruptured, as well as multiple patient- and disease-related factors. Advances in early diagnosis, neuroimaging, and treatment have improved outcomes; however, aneurysmal rupture remains associated with significant morbidity and mortality. [17,18]

In the case of unruptured aneurysms, prognosis is generally favorable, particularly for small lesions. Aneurysms measuring less than 7 mm in diameter have a low annual risk of rupture, often less than 1%, and tend to grow slowly over time. Many of these aneurysms are discovered incidentally during imaging for unrelated conditions. With appropriate risk factor modification—such as blood pressure control and smoking cessation—and regular imaging follow-up using CTA or MRA, most patients can be managed safely. However, certain morphological features, including increased aneurysm height, irregular shape, and unfavorable aspect ratio, may increase the risk of rupture and influence prognosis.

In contrast, the prognosis of ruptured cerebral aneurysms, typically presenting as subarachnoid hemorrhage (SAH), is considerably more serious. Rupture is associated with high early mortality, with approximately 25% of patients dying within the first 24 hours and nearly 50% within the first three months. Even with appropriate treatment, overall mortality may approach 40%, and a significant proportion of survivors experience long-term neurological deficits. The variability in outcomes reflects differences in the severity of hemorrhage, timing of intervention, and occurrence of complications. A key determinant of prognosis is the Hunt and Hess Grading System, with lower grades (I–II) associated with better outcomes and higher grades (IV–V) indicating poor prognosis. Other important factors include age, aneurysm location, extent of hemorrhage, vasospasm, and comorbidities. Early diagnosis and timely intervention significantly improve survival and functional recovery.

CONCLUSION:

Cerebral aneurysms are complex neurovascular lesions whose clinical significance ranges from incidental findings to catastrophic subarachnoid hemorrhage, necessitating a thorough understanding of their

pathogenesis, timely multimodal diagnosis, and individualized treatment through microsurgical or endovascular intervention. A multidisciplinary approach encompassing early intervention, adjunctive medical care, comprehensive rehabilitation, and long-term surveillance remain essential to minimizing morbidity and mortality associated with this potentially life-threatening condition.

REFERENCES:

1. Brisman, J. L., Song, J. K., & Newell, D. W. (2006). Cerebral aneurysms. *The New England journal of medicine*, *355*(9), 928–939. https://doi.org/10.1056/NEJMra052760
2. Caranci, F., Briganti, F., Cirillo, L., Leonardi, M., & Muto, M. (2013). Epidemiology and genetics of intracranial aneurysms. *European Journal of Radiology*, *82*(10), 1598–1605. https://doi.org/10.1016/j.ejrad.2012.12.026
3. Macedo, S., Alves, G., Souza, T., Siqueira, C., Siqueira, S., Siqueira, A., & Oliveira, L. (2009). Epidemiological analysis of patients with cerebral aneurysm submitted for an embolization at São José do Avaí Hospital. *Critical Care*, *13*(Suppl 1), P97. https://doi.org/10.1186/cc7261
4. Deshmukh, A. S., Priola, S. M., Katsanos, A. H., Scalia, G., Costa Alves, A., Srivastava, A., & Hawkes, C. (2024). The Management of Intracranial Aneurysms: Current Trends and Future Directions. *Neurology international*, *16*(1), 74–94. https://doi.org/10.3390/neurolint16010005
5. Cianfoni, A., Pravatà, E., De Blasi, R., Tschuor, C. S., & Bonaldi, G. (2013). Clinical presentation of cerebral aneurysms. *European journal of radiology*, *82*(10), 1618–1622. https://doi.org/10.1016/j.ejrad.2012.11.019
6. Mooij J. J. (2001). Editorial: grading and decision-making in (aneurysmal) subarachnoid haemorrhage. *Interventional neuroradiology : journal of peritherapeutic neuroradiology, surgical procedures and related neurosciences*, *7*(4), 283–289. https://doi.org/10.1177/159101990100700402
7. Huang, Z., & Ling, C. (2025). Pathophysiological mechanisms for intracranial aneurysms. *World Neurosurgery*, *201*, 124273. https://doi.org/10.1016/j.wneu.2025.124273
8. Rodemerk, J., Junker, A., Chen, B., Pierscianek, D., Dammann, P., Oppong, M. D., Radbruch, A., Forsting, M., Maderwald, S., Quick, H. H., Zhu, Y., Jabbarli, R., Sure, U., & Wrede, K. H. (2020). Pathophysiology of intracranial aneurysms. *Stroke*, *51*(8), 2505–2513. https://doi.org/10.1161/strokeaha.120.030590
9. Jung K. H. (2018). New Pathophysiological Considerations on Cerebral Aneurysms. *Neurointervention*, *13*(2), 73–83. https://doi.org/10.5469/neuroint.2018.01011
10. Radić, B., Blažeković, A., Jovanović, I., Jurišić-Kvesić, A., Bilić, E., &Borovečki, F. (2021). DIAGNOSTIC AND THERAPEUTIC DILEMMAS IN THE MANAGEMENT OF INTRACRANIAL ANEURYSMS. *Acta clinicaCroatica*, *60*(4), 758–764. https://doi.org/10.20471/acc.2021.60.04.24
11. Suarez, J. I. (2015). Diagnosis and management of subarachnoid hemorrhage. *CONTINUUM Lifelong Learning in Neurology*, *21*(5), 1263–1287. https://doi.org/10.1212/con.0000000000000217
12. Schill, C. N., Tessier, S., Longo, S., Ido, F., & Nanda, S. (2022). Differential Diagnosis of Multiple Systemic Aneurysms. *Cureus*, *14*(10), e30043. https://doi.org/10.7759/cureus.30043
13. Hackenberg, K. A., & Etminan, N. (2020). Neurovascular disease, diagnosis, and therapy: Brain aneurysms. *Handbook of Clinical Neurology*, *176*, 121–134. https://doi.org/10.1016/b978-0-444-64034-5.00001-8
14. Liang D. (2024). Endovascular treatment of intracranial aneurysms: Past and present. *Journal of cerebrovascular and endovascular neurosurgery*, *26*(3), 249–259. https://doi.org/10.7461/jcen.2024.E2023.09.005
15. Jeong, H. W., Seo, J. H., Kim, S. T., Jung, C. K., & Suh, S. (2014). Clinical Practice Guideline for the Management of Intracranial Aneurysms. *Neurointervention*, *9*(2), 63. https://doi.org/10.5469/neuroint.2014.9.2.63

16. Thompson, B. G., Brown, R. D., Amin-Hanjani, S., Broderick, J. P., Cockroft, K. M., Connolly, E. S., Duckwiler, G. R., Harris, C. C., Howard, V. J., Johnston, S. C., Meyers, P. M., Molyneux, A., Ogilvy, C. S., Ringer, A. J., & Torner, J. (2015). Guidelines for the management of patients with unruptured intracranial aneurysms. *Stroke*, *46*(8), 2368–2400. https://doi.org/10.1161/str.0000000000000070

17. Rice-Canetto, T. E., Ueno, A., Whitney, E., Reier, L., Houston, R., & Siddiqi, J. (2025). A Review of the Current Literature on Cerebral Aneurysms. *Cureus*, *17*(3), e80223. https://doi.org/10.7759/cureus.80223

18. Sanchez, S., Miller, J. M., & Samaniego, E. A. (2023). Clinical scales in aneurysm rupture prediction. *Stroke Vascular and Interventional Neurology*, *4*(1), e000625. https://doi.org/10.1161/svin.123.000625

Copyright & License:
© Authors retain the copyright of this article. This work is published under the Creative Commons Attribution 4.0 International License (CC BY 4.0), permitting unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.