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# Vertebral Artery Dissection (VAD): Causes, Consequences, and Differential Diagnoses

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## Abstract

The vertebral arteries, which supply blood to the brain's posterior circulation, are clinically most significant. A rupture in a major cervicocerebral artery causes blood to penetrate the arterial wall and divide its layers, resulting in stricture or aneurysmal dilation of the vessel. Vertebral artery dissection (VAD) is of particular concern in the patient due to its typical late appearance and association with the risk of chronic neurological damage or even trauma-related death. VAD is one of the leading causes of stroke in patients under 50 years of age and generally manifests as a migraine with and without neck pain.

VAD can be caused by hereditary factors, underlying arteriopathy, or stretching or swelling of the arterial wall. The increasing availability and use of computed tomography and magnetic resonance imaging have contributed to an improved identification rate of what was once a rare injury.

VAD therapy aims primarily to prevent vertebrobasilar ischemia. This objective is accomplished by intravenous heparin anticoagulation with an adequate activated partial thromboplastin time. The activated partial thromboplastin time should be at least double the base value. Other therapeutic options are endovascular revascularization or surgery.

Recent advances in the epidemiology and pathophysiology of VAD, as well as clinical presentations, diagnostic tools, and therapeutic options, are reviewed in this article.

***Keywords:*** *Aortic Aneurysm; Brain Stem Thrombus; Ischemia of the Vertebrobasilar Circulation; String Sign; Subarachnoid Hemorrhage*

## Abbreviations

CAD: Carotid Artery Dissection; CT: Computed Tomography; VA: Vertebral Artery; VAD: Vertebral Artery Dissection

## Introduction

Dissection of the carotid and vertebral arteries, which supply blood to the brain, was identified in the early 1950s [1]. Arterial dissection occurs following a tear when blood rushes through the vascular wall, generally at the endothelium surface. A subintimal dissection results in luminal constriction or occlusion, whereas a subadventitial dissection results in a pseudoaneurysm, which carries a bleeding risk [2]. The average age of most people with dissection is 40 years (range: 30 – 50) [3]. Spontaneous dissection can be because of substantial trauma or seemingly innocuous activities (e.g., heavy exercise) [2].

The first unequivocal example of vertebral artery dissection (VAD) was documented in the 1970s. However, rare cases of carotid dissection were reported earlier. Charles Miller Fisher, a Canadian neurologist and stroke physician at Massachusetts General Hospital, first noticed the "string sign" anomaly in the internal carotid artery on cerebral angiograms of stroke patients in 1971. In 1978, he found that the same anomaly occurred even in the vertebral arteries (VAs) [4–6].

Pathologically, VAD is distinguished by an acute, extensive breakdown of the internal elastic lamella, which separates tunica intima from tunica media in arterioles [7]. It is commonly classified as 'spontaneous' or 'traumatic', with cases involving modest, non-penetrating neck trauma or twists lying in the middle. However, this categorization can be unclear and confusing [2].

Additionally, VAD is categorized into two types: ischemic and hemorrhagic dissection. Ischemia of the vertebrobasilar circulation because of arterial constriction and thrombosis causes ischemic VAD.

Subarachnoid hemorrhage induced by a puncture in an intradural VA dissecting aneurysm causes hemorrhagic VAD [2]. Dissection causes discomfort in the back of the head and neck, often spreading to the orbits. Vertebral dissection can lead to unilateral or bilateral infarction of the occipital lobe or brainstem involvement, resulting in double vision and other eye movement abnormalities [8]. The reported incidence of VAD has tripled since 1994 [9].

VAD-induced cerebral ischemia affects approximately 80% of patients and their work life. Although functional results appear to be favorable in most patients, understanding the historical development of VAD and standards for follow-up care remains unclear [10].

## Discussion (Part 1)

### Vertebral artery

The term 'vertebral' indicates the position of the arteries throughout the vertebrae or backbone. They are a component of the circulatory system [11]. VAs typically begin from the subclavian artery (V1) and ascend vertically through the transverse foramina of the cervical vertebrae (C6–C1), with a lateral deviation between C2 and C1 (V2). The VA forms a mobile "loop" between the C1 transverse process and dura at the foramen magnum. This loop, located beneath the posterior atlantooccipital membrane, allows a high degree of rotation at C1/C2 (V3). Then, the artery coalesces with the opposing VA intracranially, creating the basilar artery—the primary route of oxygenated blood to the brainstem, cerebellum, and posterior brain (V4) [12].

The VA augments cerebral blood flow in all vertebrates (mammals), including humans [13,14]. The primary role of the VA is to transport blood to the vertebrae of the neck, upper spine, and the region surrounding the exterior of the skull. It also transports blood to two critical parts of the brain: the posterior fossa and occipital lobes [11].

Because of its location, the VA above C2 is considered the most susceptible artery. It is attached to the lamina intracranially, C1 transverse foramen, and groove on the superior face of C1 [15]. The factors contributing to a VA injury are detailed in Figure 1 [15,16].

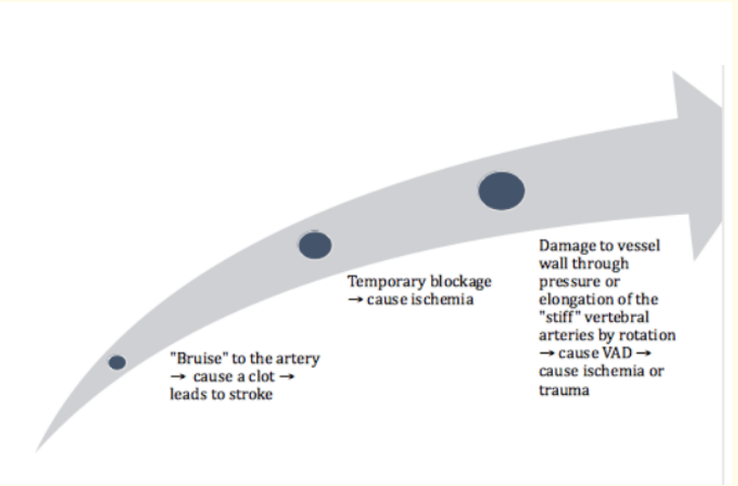


***Figure 1:*** *Causes of vertebral artery (VA) injury [15,16].*

## Vertebral artery dissection (VAD)

Typically, VAD occurs in the extradural portion of the vessel. However, it can emerge as intradural and mixed intradural-extradural dissections. The position of the dissection may direct its presentation and management. Severe migraine and subarachnoid hemorrhage are common in intradural dissection, indicating brainstem or cerebellar ischemia. Most patients with extradural dissection present ischemic brainstem symptoms [17].

The clinical manifestations of VAD include severe neck discomfort, usually in the occipitocervical region, followed by ischemic symptoms after a variable period. Ischemic symptoms may be absent in certain patients. Subarachnoid bleeding is associated with intracranial VAD in >50% of patients. Other symptoms include brain stem thrombus and aortic aneurysm [18]. The typical signs and symptoms of VAD are detailed in Figure 2 [19,20].

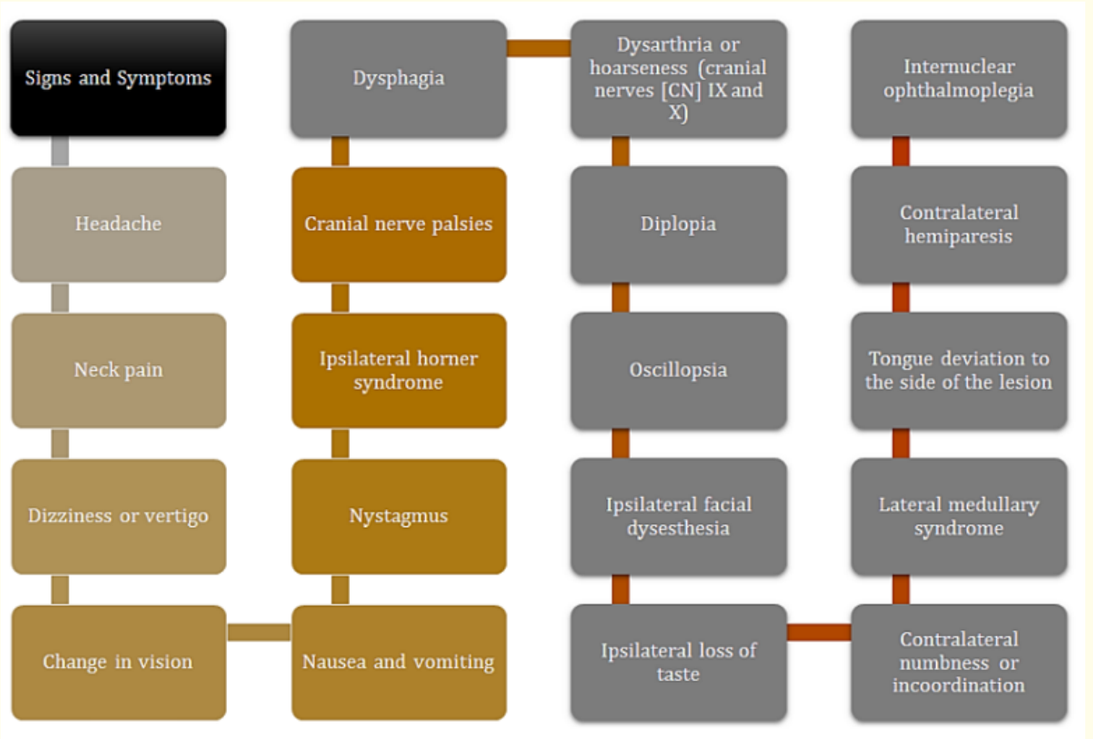


***Figure 2:*** *Signs and symptoms of vertebral artery dissection (VAD) [19,20].*

Signifiant trauma is a major factor in VAD. However, other VAD potential causes are listed in Figure 3 (below).


***Figure 3:*** *Predisposing risk factors for vertebral artery dissection (VAD) [19–22].*

## Epidemiology

The annual incidence of VAD is one-third (2.6 – 3/100,000 individuals) of that of cervical internal carotid artery dissection (CAD) [3,23]. Extracranial VAD is more prevalent, accounting for up to 15% of all documented cases of cervicocerebral dissection, while intracranial VAD is rare, accounting for only around 5% of all recorded cases [3]. In people younger than 45 years, VAD is one of the most common causes of stroke [21]. According to Schievink, bilateral VAD contributes 10% to 25% of all cases of thrombotic stroke in young people [24]. In contrast to gender comparisons, VAD has a 3:1 female-to-male ratio [18,19].

## Diagnosis

Early diagnosis of VAD is vital for improved outcomes, especially when the presentation is asymptomatic or unclear [23]. A multidisciplinary team of a radiologist, an emergency care physician, a registered nurse, a neurosurgeon, and a cardiovascular surgeon most likely sees patients with VADs [21]. The dissection is often diagnosed using a high index of intuition and a variety of imaging modalities [23].

Non-invasive computed tomography angiography or magnetic resonance angiography is the 2011 joint recommendation of the American Stroke Association/ American College of Cardiology Foundation/American Heart Association Class I for an initial diagnosis of suspected VAD [19,21]. Vascular duplex scanning, transcranial Doppler ultrasound, computed tomography (CT), cerebral angiography, and ultrasonographic examination are other diagnostic procedures (Table 1) [7,21,25].

| Diagnosis technique | Description |
|---|---|
| **Computed tomography (CT) scanning** | • A CT scan might reveal ischemia in the posterior fossa or subarachnoid hemorrhage.<br>• It can also detect an obstructed VA or a thrombus in the mural artery. |
| **CT angiography (CTA)** | • CTA is preferable than CT scan because it can detect irregularities in the vascular lumen or thickness of the arterial wall more readily.<br>• The first test of choice is CTA, however a little expertise is needed with the procedure. Changes are visible relatively quickly after ictus, unlike some of the other modalities. |
| **Magnetic resonance techniques (MRI)** | • MRI has surpassed angiography as the gold standard for diagnosing VAD; it is supreme to angiography in the identification of dissections without concomitant luminal anomalies or in cases culminating in nonspecific occlusion.<br>• MRI can also be utilized to track the progress of the dissections. |
| **Vascular duplex scanning** | • Determines aberrant blood circulation in 95% of patients with VAD. Also helps in reveling the signs that are specific to VAD (eg, segmental dilation of the vessel, eccentric channel). |
| **Transcranial Doppler ultrasonography** | • Useful in detecting approximately 75% of the flow-sensitive high-intensity signals (HITS) of irregularities seen in VAD, which are characterized by sporadic scattered microemboli resulting from dissection.<br>• Ultrasound imaging may play a role in the early prognosis of dissection if CT-A or MRA are not attainable. |
| **Cerebral angiogram** | • If the MRI and CT scans fails to demonstrate any abnormality, then a cerebral angiography may be necessary. |
| **Ultrasonographic examination** | • This approach has been reported to be yet another simple and non-invasive means of diagnosing VAD.<br>• It can give anatomic and hemodynamic information regarding the VA intracranial and extracranial segments |

***Table 1:*** *Diagnostic techniques to identify vertebral artery dissection (VAD) [7,21,25].*

It is crucial to distinguish VAD from spinal column fracture, fibromuscular dysplasia, cervical stress, subarachnoid hemorrhage, hypoplasia, aplasia, agenesis of the vessel, severe headache, cerebrovascular disease, ischemia, vasculitis affecting the vertebrobasilar circulation, or vertebrobasilar atherothrombotic disease by using one of these diagnostic techniques [21].

## Management

Early diagnosis and treatment can effectively deter more severe complications of VAD. The management of VAD depends on several factors, including clinical presentation, symptom onset, anatomical observations, number of vessels affected, and contraindications to certain medicines.

The 2014 AHA/ASA guidelines recommend thrombolysis or conservative therapy with antithrombotic medications. Combination therapy of antiplatelets and anticoagulants is associated with an increased risk of hemorrhaging. Thus, its use is contentious [20,23]. Nevertheless, antithrombotic drugs have been used to treat VAD in a few patients. Some of the documented cases are presented in Table 2 [23,26,27,28–32].

| Case | Risk factor | Trigger | Clinical presentation | Imaging | Medical management | Outcome |
|---|---|---|---|---|---|---|
| ***Spontaneous bilateral VADs [23]*** | - | Multiple roller coaster rides | Worsening neck pain and headache for two weeks | Brain MRI: Multiple strokes in the left posterior inferior cerebellar artery (PICA) territory (largest branch of the VA); CT angiography: Concerning for VADs. | Anticoagulation and antiplatelet therapy: | After three months, MR angiography revealed that the dissection had completely resolved. The patient reported no bleeding issues as a result of dual treatment. |
| ***VAD [26]*** | Acute demyelinating Encephalomyelitis (ADEM) | ADEM flare | Bilateral nuchal pain and left side motor deficit since last 2 weeks | Brain 1st MRI (2 weeks before episode): Ischemic lesion in the right parietal lobe without signs of vessel damage; Brain 2nd MRI: Increased lesion in the same area | Anticoagulation: 2 cycles of corticosteroids IV (5 days) | Follow-up, At 11 months: resolution of symptoms, slow remission of the intracranial lesion |
| ***Bilateral VAD [27]*** | Migraines | Strenuous exercise sessions | Numerous instances of self-resolving occipital discomfort with hallucinations or delusions, scintillations, and visual disturbances | MR brain: Unremarkable | Heparin → 10 days; Oral anticoagulation with warfarin for 6 months | At 6 months, the images showed excellent recovery. |
| ***Bilateral VAD: multiple artery affection or early recurrence [28]*** | Migraines, low BMI, and recent infection | - | Overall weakness, neck discomfort, vertigo, and migraine, as well as transient sight abnormalities for more than 10 days. | CT and MRI: Bilateral ischemic lesions in the posterior circulation. | Anticoagulation with LMWH and bridge to warfarin | Symptoms returned two weeks after commencing anticoagulation. |
| ***Extracranial VAD [29]*** | - | - | Neck pain, ataxia, and imbalance | MRI: Bilateral cerebellar infarcts, larger on the right side. | Warfarin for 12 months | Asymptomatic at 12 month |
| ***Traumatic bilateral CAD and VAD [30]*** | - | Motor vehicle accident | Left hemiparesis, dysarthria | CT showed right caudate head and basal ganglia infarction. | Anticoagulation with heparin and warfarin at discharge | Ambulates independently, residual left-sided weakness; maintained on aspirin 81 mg daily after completing 1 year of warfarin therapy |
| ***Postpartum VAD [32]*** | Persistent vaginal bleeding. | Postpartum | One-day history of dizziness, ataxia of gait, vomit, bilious reflux, purging, and prefrontal headache. | MRI revealed a large acute infarct in the left cerebellar hemisphere in the area of the posterior inferior cerebellar artery (PICA), as well as a left VA blockage.CT angiography revealed a left VAD with severe flow restriction that began at the C4 vertebral body level and extended into the intracranial VA, including the left PICA. | Heparin infusion | After a four-day hospital stay, the patient recovered clinically and was released on therapeutic-dose of enoxaparin with hematology/oncology. |

***Table 2:*** *Case studies on treating vertebral artery dissection (VAD) with antithrombotic drugs.*

More potent medications (e.g., intravenous or intra-arterial thrombolytics) are used to manage VAD in particular patients. However, the safety and efficacy of these agents in patients with VAD-induced acute stroke remain unassessed in randomized controlled studies. Thus, surgery or endovascular procedures are recommended in patients with chronic or worsening symptoms despite adequate therapeutic care, those with hemodynamically significant residual stenosis, or those with anticoagulant-related concerns [4].

The prognosis of VAD appears positive because the likelihood of survival with a satisfactory outcome is about 75% or potentially higher (85.7%) if antiplatelet medications are administered [33]. Furthermore, a study of 200 patients with CAD found that the risk of recurrent dissection after therapy was about 1% per year after a mean follow-up of 7.4 years [34].

During the acute phase, the fatality rate of VAD is 10%. Severe cerebral dissection, brain ischemia, or intracranial bleeding are the most common causes of death. Patients who survive the initial crises perform exceptionally well, with long-term repercussions extremely rare [21].

Antithrombotic medications are commonly used to treat VAD but are associated with complications. Also, these medications are not recommended for patients with hemorrhagic myocardial infarction or subarachnoid hemorrhage from intrauterine dissection because the clinical course in these circumstances may deteriorate after anticoagulation. Figure 4 details the other negative impacts of antithrombotic medication use in treating VAD [17,33,35].

## The future of VAD treatment

Differences in the efficacy of currently used antiplatelet and anticoagulant drugs have not been demonstrated, possibly because of the small number of cases studied. However, extensive studies are underway to determine which of these is more beneficial. In addition,

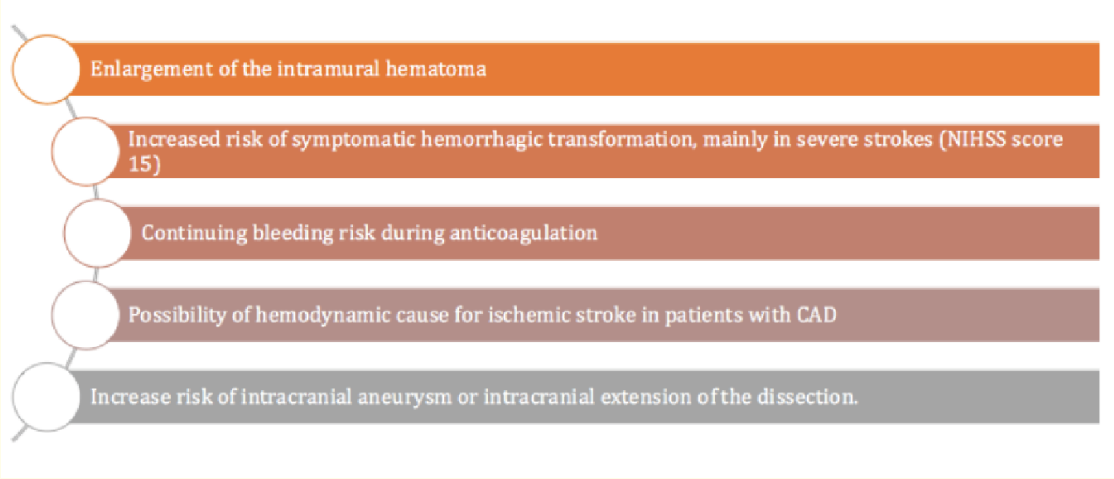


***Figure 4:*** *Adverse effects associated with antithrombotic drugs in vertebral artery dissection (VAD) [17,33,35].*

hypertension is a known risk factor for VAD, and using antihypertensive drugs to manage blood pressure and lower arterial wall pressure may be a vital intervention technique [36].

Statin use in VAD management is also being investigated, and the results are awaited [37]. Also, new treatment approaches, including direct oral anticoagulants and dual antiplatelets for VAD, are being studied [38].

## Discussion (Part 2)

### Major causes of VAD

A history of a mild triggering incident is typically noted in individuals with spontaneous VAD [39]. Trauma, generally moderate or even other rotary events, is the cause of dissection in up to 40% of patients [38]. Chronic cough, emesis, sustained head wobbling, thrill rides, various sports activities (e.g., golf, treadmill running, springboard, and scuba), and surgical and medical procedures (e.g., anesthesia, intravenous catheter placement, and fine-needle aspiration biopsy of the neck) can trigger a VAD [33,40]. Sudden neck movements can cause mechanical stretching of the artery wall, leading to dissection [35, 41].

Hyperhomocysteinemia—an abnormally high level of homocysteine in the blood—is another risk factor for dissection, having homocysteine levels of > 12 mol/L [42].

Connective tissue abnormalities (idiopathic causes), including Ehlers-Danlos syndrome type IV, Marfan syndrome, medial cystic necrosis of intracranial vessels, autosomal dominant polycystic kidney disease, osteogenesis imperfecta, vessel abnormalities, and potential predisposing genetic conditions, are also associated with an increased risk of VAD [35,42].

A spike in blood pressure (hypertensive surge) is probably a risk factor for dissection. VA is considered prone to mechanical damage, considering its known vulnerability to traumatic injury. The endothelial-damaging action of the hypertensive surge may increase the

likelihood of dissection in the naturally fragile VA. When blood flows into this false lumen, it pushes the intimal-medial layer closer to the actual lumen of the artery, wholly or partially blocking blood flow and ultimately leading to cerebral ischemia [43].

## Discussion (Part 3)

### Consequences of VAD

Inflammation occurs in the nape of the neck in 50% of patients, and migraine develops in two-thirds of patients due to VAD. Migraine usually occurs in the occipital area, but the complete hemicrania or frontal region may be affected in rare cases [24,35,44]. Activated nociceptors may induce pain due to dissection of the vascular wall. Bilateral neck discomfort or headaches are possible. Unilateral arm discomfort or paralysis due to cervical-root involvement, commonly at the C5–C6 level, and spinal epidural hematomas are the rare signs of VAD [24,34].

The most common complication of VAD is cerebral ischemia because of VA blockage. The risk of stroke is highest during the first weeks after dissection, although it can occur up to a month later. Ischemic complications are more prevalent in extracranial VAD, with a stroke occurring in 68% of individuals with extracranial VAD compared to 32% with intracranial VAD. Subarachnoid bleeding, on the contrary, occurs virtually exclusively in individuals with intracranial VAD, with a frequency of around 60% in individuals with intracranial VAD [45]. Isolated cervical spinal cord ischemia is a rare but increasing complication of VAD [24]. Spontaneous VAD occurs in the absence of trauma and can be a potentially catastrophic event, causing a delay in diagnosis and leading to poor outcomes [46].

## Discussion (Part 4)

### Treatment of VAD

Individuals with cerebral ischemia due to spontaneous VAD who arrive at the hospital within 3 hours of the onset of the symptoms may be eligible for systemic thrombolytic treatment. Catheter-directed thrombolysis (dissolution of abnormal blood clots) is recommended in patients who come to the hospital up to 4.5 hours after the symptom onset or those who are not eligible for systemic thrombolytic therapy. Individuals not qualified for thrombolytic treatment can receive heparin, aspirin (or other drugs of the same classification), or endovascular or surgical repair, depending on VAD damage [16].

Anticoagulants and antiplatelets are the preferred agents to prevent thromboembolic diseases arising from VAD [19]. In the acute phase of VAD, heparin and oral anticoagulants are usually recommended for approximately 3 months. After 3 months, anticoagulants are typically switched to antiplatelets. Physicians may recommend continuing antiplatelet therapy when arterial abnormalities, such as stenosis or aneurysms, although evidence for its efficacy remains unclear [4].

It remains uncertain if anticoagulants are superior to antiplatelets in preventing early or late recurrent events. A prospective observational analysis indicated that aspirin-treated individuals had a greater incidence (12.4%) of recurring transient ischemic attack and ischemic stroke or death than those treated with an anticoagulant (8.3%). However, the differences were not statistically significant [47].

Chakrapi., *et al*. (2008) described a case of a unilateral internal carotid artery (ICA) and VA dissection treated with extended anticoagulation and dual-antiplatelet therapy. The patient remained asymptomatic at the 6-month follow-up visit and stopped warfarin. The results of the 1-year CT scan revealed 30% residual dissection in the right ICA and 30 – 50% dissection in both VAs. Based on these factors, the patient was prescribed clopidogrel for a year (a total of 18 months from the occurrence) and aspirin permanently [48].

In 2015, the Cervical Artery Dissection in Stroke Study (the first randomized study in the world) compared antiplatelets with anticoagulants in extracranial carotid and VAD treatment in 250 patients for 3 months. According to the findings, there were no changes in endpoints between the antiplatelet and anticoagulant groups. Furthermore, recurrent stroke was uncommon (2%) throughout this period, and both groups had a considerably low rate of adverse effects after dissection [49]. Based on these findings and the results of other randomized controlled trials, the American Heart Association/American Stroke Association recommends antiplatelet or anticoagulant medication for at least 3 - 6 months in patients with dissection-related ischemic stroke (Class IIa recommendation) [50].

Endovascular treatment is explored in patients who experience disease progression despite antithrombotic medication, those with pseudoaneurysms, or those who do not respond to anticoagulation therapy [51]. These treatments are practical, effective, and comfortable, and have explicit radiographic findings [52]. Balloon angioplasty is the currently preferred endovascular therapy, often followed by implanting one or more balloon-expandable or, ideally, self-expanding stents. Coil embolization or the placement of a covered stent may be required in case of associated dissecting aneurysms.

Endovascular treatment for CAD and VAD has excellent short-term clinical and radiological outcomes, with a low, but not inconsequential, complication rate [24,34,35]. A meta-analysis of 39 observational studies involving various treatment methods for VAD in adults was conducted to establish the clinical results of patients receiving endovascular therapy. 75.11% reported excellent results, 10.10% reported good results, and 13.70% reported inferior results. About 10.5% of individuals experienced postoperative problems [53].

Surgical treatment is intended for people with long-term symptoms who have not responded to medications or are not eligible for endovascular treatment. *In situ* interposition grafts and extracranial-intracranial bypasses are the surgical treatments recommended for VAD [24,34]. The position and severity of the damage determine the surgical therapy for VA injuries. Patients with persistent bleeding and hematoma development may require VA ligation or embolization. Arteriovenous fistulas have been recorded and treated surgically using several techniques, including embolization and proximal ligation [54].

## Conclusion

The pathology of VAD pathology remains unclear. However, it has a multifaceted etiology, including genetic and environmental components. VAD is critical in young and middle-aged patients with stroke. At least 40% of all ischemic strokes in the posterior fossa are because of VAD. One of the most prevalent symptoms of VAD is a headache with or without neck discomfort. The primary goals of VAD treatment are to avoid stroke and improve neurological outcomes. Anticoagulation is used to prevent clot formation, propagation, and embolization. Antithrombotic medication therapy is empirical rather than based on evidence. Thus, anticoagulants and antiplatelets must be compared in randomized controlled studies.

## Conflict of Interest Statement

The authors declare that this paper was written without any commercial or financial relationship that could be construed as a potential conflict of interest.

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