Narrative review thesis

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VERTEBROBASILAR INSUFFICIENCY: DIAGNOSIS, TREATMENT, AND PREVENTION

Berdiyeva Kamola Axatovna
Ilmiy rahbar: proff Raxmatullayeva Gulnora Qutbiddinovna

Abstract. *Vertebrobasilar insufficiency is best understood as a clinical syndrome of transient or persistent ischemia in the posterior circulation rather than as a final etiologic diagnosis. This narrative review thesis summarizes a practical evidence-based approach to its recognition, diagnostic confirmation, acute treatment, and prevention. A targeted search of PubMed/MEDLINE and professional-society repositories was performed for English-language guidelines, randomized trials, systematic reviews, meta-analyses, and diagnostic studies published from 1 January 2021 through 13 August 2026. The synthesis shows that dizziness or vertigo alone has low specificity, whereas acute imbalance with central ocular-motor signs, diplopia, dysarthria, dysphagia, limb or truncal ataxia, crossed deficits, visual-field loss, or altered consciousness should trigger an urgent posterior-circulation stroke pathway. Non-contrast computed tomography excludes hemorrhage but is insensitive for small posterior fossa infarction; computed-tomography angiography or magnetic-resonance angiography identifies arterial occlusion, stenosis, or dissection, and diffusion-weighted magnetic resonance imaging defines tissue injury. Early negative diffusion-weighted imaging does not fully exclude ischemia. Treatment is time- and mechanism-dependent: eligible patients require reperfusion assessment, selected basilar-artery occlusion warrants thrombectomy evaluation, and antithrombotic therapy must be matched to non-cardioembolic, cardioembolic, or dissection mechanisms. Long-term prevention integrates blood-pressure and lipid control, smoking cessation, physical activity, diabetes management, rhythm surveillance, and adherence support. Routine intracranial or vertebral stenting is not a first-line preventive strategy. A syndrome-tissue-vessel-mechanism framework reduces diagnostic anchoring and links findings to actionable treatment.*

Keywords: *vertebrobasilar insufficiency; posterior circulation stroke; basilar artery occlusion; acute vestibular syndrome; neuroimaging; reperfusion; secondary prevention.*

INTRODUCTION

The vertebral arteries, basilar artery, and posterior cerebral arteries supply the brainstem, cerebellum, thalami, and occipital lobes. Ischemia within this network can

produce symptoms ranging from transient disequilibrium to devastating tetraparesis, bulbar failure, coma, or locked-in syndrome. Posterior circulation events account for approximately one fifth of ischemic strokes and remain disproportionately vulnerable to delayed recognition because many presentations are non-lateralizing and the conventional National Institutes of Health Stroke Scale (NIHSS) underweights gait, truncal, ocular-motor, and lower cranial nerve dysfunction [1-3].

The term vertebrobasilar insufficiency (VBI) is clinically useful only when it initiates, rather than ends, diagnostic reasoning. It does not specify whether the mechanism is atherosclerotic stenosis, artery-to-artery embolism, cardioembolism, dissection, perforator disease, subclavian steal, systemic hypoperfusion, or rare dynamic mechanical compression. Recording VBI as a definitive explanation for recurrent dizziness without tissue or vascular assessment risks both overdiagnosis and missed stroke [1,4].

The clinical challenge has two time horizons. During the acute episode, clinicians must identify a reperfusion candidate without allowing a low NIHSS score or an initially negative scan to create false reassurance. After stabilization, the mechanism must be determined because the preventive strategy for large-artery atherosclerosis differs from that for atrial fibrillation, cervical artery dissection, or a hemodynamic low-flow state [4-6].

The objective of this thesis is to synthesize current evidence into a clinically usable pathway for the diagnosis, treatment, and prevention of vertebrobasilar ischemia. The central proposition is that each suspected case should be described through four linked domains: syndrome, tissue, vessel, and mechanism. This structure is intended to improve communication, reduce diagnostic anchoring, and make treatment decisions explicitly traceable to evidence.

LITERATURE REVIEW

Terminology, mechanisms, and risk context

Posterior circulation ischemia arises through embolic, thrombotic, hemodynamic, or perforator mechanisms. Atherosclerotic plaque at the vertebral origin, intracranial vertebral segment, or basilar artery may generate local thrombosis, distal emboli, or flow limitation. Cardioembolism should be considered when infarcts involve multiple vascular territories or when atrial fibrillation, recent myocardial infarction, cardiomyopathy, or valvular disease is present. Small-vessel disease often produces compact pontine or thalamic syndromes, but a small lesion does not by itself establish a small-vessel mechanism [1,2].

Vertebral artery dissection is particularly important in younger or middle-aged adults and may follow minor trauma or occur spontaneously. New posterior neck or occipital pain accompanying focal posterior circulation symptoms strengthens

suspicion and justifies vascular imaging of the entire cervical and intracranial course [11]. Subclavian steal may cause arm claudication, inter-arm blood-pressure difference, and retrograde vertebral flow, although anatomic steal is often asymptomatic. Rotational vertebral artery compression is rare and should be reserved for reproducible head-turning symptoms supported by dynamic vascular evidence rather than inferred from common cervical spondylosis [20].

Traditional vascular risks—hypertension, dyslipidemia, diabetes mellitus, smoking, chronic kidney disease, obesity, physical inactivity, and established atherosclerotic disease—raise the prior probability of an ischemic cause. Nevertheless, their absence does not exclude dissection, embolism, or other posterior circulation pathology. Risk factors therefore modify probability but must not replace a focused neurological examination and appropriate imaging.

Clinical recognition and common diagnostic traps

Symptoms frequently include vertigo or dizziness, gait and truncal instability, nausea, dysarthria, dysphagia, diplopia, nystagmus, limb ataxia, unilateral or bilateral weakness or sensory loss, visual-field disturbance, and headache. The localization becomes more credible when symptoms cluster across functions—for example, acute vertigo with inability to stand, direction-changing gaze-evoked nystagmus, diplopia, or dysarthria—rather than when isolated nonspecific light-headedness is the only complaint [1-3].

Brainstem infarction can produce crossed findings: an ipsilateral cranial nerve deficit with contralateral motor or sensory impairment. Cerebellar infarction may initially appear as isolated imbalance but can deteriorate through edema and fourth-ventricle compression. Basilar artery occlusion may fluctuate, progress stepwise, or begin with transient symptoms before severe quadriparesis or impaired consciousness. Such fluctuation is not reassurance; it may indicate an unstable occlusive process [1,18].

In acute vestibular syndrome—continuous dizziness or vertigo with spontaneous nystagmus, nausea or vomiting, head-motion intolerance, and gait unsteadiness—the HINTS examination can help a clinician trained in its use distinguish a central from a peripheral pattern. GRACE-3 emphasizes that HINTS is not a generic test for every dizzy patient and should not be deployed by an untrained examiner or in patients without the appropriate syndrome and nystagmus [5]. Severe truncal ataxia, new hearing loss, central ocular-motor signs, or focal neurological deficits should lower the threshold for stroke imaging.

Two recurrent sources of error are overreliance on a low NIHSS score and premature attribution to a benign vestibular disorder. The posterior NIHSS study demonstrated that adding posterior-circulation deficits improves prognostic

discrimination [21]. A complete examination should therefore document mental status, visual fields, pupils, ocular alignment and movements, nystagmus, facial and bulbar function, limb coordination, sensation, strength, gait, and truncal control whenever safe.

Table 1. Clinical patterns that should increase suspicion of posterior circulation ischemia

Presentation	High-risk features	Main differential	Immediate implication
Acute vestibular syndrome	Severe gait/truncal ataxia, central ocular-motor signs, new hearing loss, focal deficit	Vestibular neuritis; labyrinthitis; toxic/metabolic causes	Stroke-trained examination; urgent vascular/tissue imaging when central features are present
Episodic vertigo or dizziness	Diplopia, dysarthria, weakness, sensory loss, reproducible vascular pattern, recent neck pain	BPPV; orthostatic hypotension; migraine; arrhythmia	Clarify duration and triggers; assess for TIA, dissection, or hemodynamic mechanism
Brainstem syndrome	Crossed cranial-nerve and long-tract signs; bulbar dysfunction; bilateral findings	Demyelination; inflammation; tumor; metabolic encephalopathy	Activate acute stroke pathway; CTA/MRA of head and neck; MRI-DWI when feasible
Cerebellar syndrome	Inability to walk or sit unsupported, dysmetria, headache, vomiting, declining consciousness	Intoxication; infection; mass lesion; peripheral vestibulopathy	Urgent imaging and monitoring for edema, hydrocephalus, and neurosurgical deterioration
Possible basilar occlusion	Fluctuating quadriparesis, dysarthria/dysphagia, abnormal eye movements, reduced consciousness	Seizure; toxic/metabolic coma; neuromuscular disorder	Immediate CTA/MRA and transfer to a thrombectomy-capable pathway if occlusion is present

Source: authors' synthesis from references [1-5,18,21].

Imaging and etiologic investigation

Non-contrast computed tomography (NCCT) is the initial examination in many emergency pathways because it detects intracranial hemorrhage and important mimics. Its sensitivity for small or early posterior fossa infarction is limited by lesion size and beam-hardening artifact. A normal NCCT should therefore not terminate evaluation when the clinical syndrome remains concerning [1,4].

Computed-tomography angiography (CTA) from the aortic arch through the intracranial circulation rapidly identifies basilar or vertebral occlusion, stenosis, dissection-related abnormalities, and relevant collateral anatomy. Magnetic-resonance angiography (MRA) is an alternative when available and may be combined with vessel-wall or fat-suppressed sequences in selected cases. Duplex ultrasound can assess vertebral flow direction and proximal disease but does not adequately exclude distal or intracranial pathology [9]. Digital subtraction angiography is generally reserved for endovascular treatment or when noninvasive imaging is inconclusive and the result will change management.

Diffusion-weighted magnetic resonance imaging (MRI-DWI) is the most sensitive routine method for demonstrating acute ischemic tissue, defining lesion distribution, and helping distinguish embolic, perforator, and border-zone patterns. Baseline DWI may nevertheless be negative, particularly with small brainstem lesions or very early imaging; a large cohort reported DWI-negative imaging in 7.7% of ischemic strokes overall [6]. Persisting or evolving clinical suspicion justifies repeat MRI or alternative imaging rather than diagnostic closure.

CT perfusion may add information when MRI is unavailable or delayed. A 2021 systematic review and meta-analysis reported pooled sensitivity of 72% and specificity of 90% for posterior circulation stroke, compared with substantially lower sensitivity for NCCT [7]. Perfusion maps are not a stand-alone rule-out test; technical coverage, small lesions, and posterior fossa artifacts can reduce reliability. Flow assessment by quantitative MRA may further stratify selected patients with vertebrobasilar stenosis, but anatomic narrowing, infarct pattern, and clinical course must be integrated [19].

Etiologic work-up should include blood pressure in both arms, glucose, complete blood count, renal function, coagulation testing when relevant to reperfusion or anticoagulation, electrocardiography, and cardiac rhythm monitoring. Echocardiography and extended rhythm surveillance are selected according to age, infarct pattern, and suspicion of a cardiac source. Testing for inflammatory, thrombophilic, or genetic disorders should be hypothesis-driven rather than routine. A tissue-negative TIA remains a clinical diagnosis and requires rapid vascular and cardiac evaluation because early stroke risk can be substantial [10].

Table 2. Diagnostic tools: purpose, strengths, and limitations

Tool	Primary purpose	Strength	Important limitation
NCCT	Exclude hemorrhage and major mimics	Fast and widely available	Low sensitivity for small/early posterior fossa infarction
CTA head and neck	Detect occlusion, stenosis, dissection, and collateral anatomy	Rapid vessel-level decision support	Contrast/radiation considerations; stenosis does not prove symptom mechanism
MRI-DWI/FLAIR	Confirm tissue injury and infarct distribution	Best routine tissue sensitivity	Early small brainstem lesions may be DWI-negative; access and motion constraints
CT perfusion	Estimate perfusion abnormality and support selection	Can supplement CT-based pathways	Not a rule-out test; posterior fossa artifacts and variable coverage
Duplex/TCD	Assess flow direction, proximal disease, and hemodynamics	Bedside, repeatable, no radiation	Operator dependence; limited visualization of distal/intracranial anatomy
DSA	Definitive lumen assessment and intervention	Highest spatial/temporal vascular detail	Invasive; reserve for treatment or unresolved management-changing questions
ECG/rhythm monitoring/echo	Identify cardioembolic mechanism	Directly changes antithrombotic prevention	Yield depends on monitoring duration and patient selection

Source: authors' synthesis from references [1,4,6-10,19].

RESEARCH METHODOLOGY

This work used a targeted narrative review design. PubMed/MEDLINE and the repositories of the American Heart Association/American Stroke Association (AHA/ASA), European Stroke Organisation/European Society for Minimally Invasive Neurological Therapy (ESO/ESMINT), European Society for Vascular Surgery

(ESVS), and relevant emergency-medicine guidance were searched for English-language publications dated 1 January 2021 to 13 August 2026.

Search concepts included “vertebrobasilar insufficiency,” “posterior circulation stroke,” “basilar artery occlusion,” “vertebral artery stenosis,” “vertebral artery dissection,” “acute vestibular syndrome,” “HINTS,” “CTA,” “MRI diffusion,” “perfusion,” “thrombectomy,” “dual antiplatelet therapy,” and “secondary prevention.” Priority was given to current clinical guidelines and scientific statements, randomized controlled trials, systematic reviews or meta-analyses, and diagnostic cohort studies. A 2024 systematic review was used for the rare dynamic compression syndrome.

Evidence was extracted into four domains: clinical syndrome, tissue imaging, vascular imaging, and etiologic mechanism. Treatment findings were grouped into acute stabilization and reperfusion, early antithrombotic management, mechanism-specific therapy, rehabilitation, and long-term prevention. Where guidelines differed or trial populations were narrow, recommendations were qualified by patient selection and local stroke-system capacity.

This is not a systematic review or meta-analysis: no registered protocol, exhaustive duplicate screening, or formal risk-of-bias scoring was performed. The results are therefore a structured clinical synthesis, not pooled causal estimates. No new patient data were collected, and institutional ethics approval was not required for the review itself.

ANALYSIS AND RESULTS

A staged diagnostic strategy

The reviewed evidence supports a staged process. First, define the syndrome and time last known well. Any sudden focal posterior circulation deficit, central acute vestibular syndrome, or progressive bulbar and bilateral motor pattern should activate the same time-sensitive response used for other suspected strokes. Second, exclude hemorrhage and identify a treatable large-vessel lesion without waiting for MRI when CTA is immediately available. Third, define tissue injury and its distribution. Fourth, determine the mechanism before assigning a long-term preventive regimen.

The syndrome-tissue-vessel-mechanism formulation is more informative than the isolated label VBI. For example, “acute vestibular syndrome; punctate lateral medullary DWI lesion; ipsilateral vertebral V4 stenosis; probable artery-to-artery embolism” directly guides antiplatelet and risk-factor management. In contrast, “recurrent dizziness due to VBI” does not document lesion, vessel, mechanism, or competing diagnoses and cannot justify invasive treatment.

A negative early study is interpreted in relation to pretest probability. If the neurological examination is normal, symptoms are positional and stereotyped, and a validated peripheral vestibular diagnosis is made, further stroke testing may be

unnecessary. If disabling gait ataxia, cranial nerve abnormalities, central ocular-motor findings, evolving symptoms, or a high-risk vascular history persists, repeat or complementary imaging is appropriate despite an initially negative NCCT or DWI [4-7].

Posterior circulation TIA should be treated as an urgent opportunity for prevention. Symptom resolution does not eliminate risk, and reliance on a score alone can miss high-risk vascular lesions. Rapid CTA or MRA, ECG and rhythm assessment, laboratory testing, and expedited specialist follow-up or observation are selected according to clinical features, imaging, recurrence, and local resources [10].

Acute treatment and early management

Initial management includes airway and swallowing assessment, oxygen only when hypoxemia is present, treatment of severe glucose abnormalities and fever, and avoidance of hypotension. Blood-pressure targets must reflect whether intravenous thrombolysis or thrombectomy is planned and follow the current acute stroke protocol. Because dysphagia and aspiration are frequent in brainstem stroke, oral intake should be withheld until screening is completed. Deteriorating cerebellar infarction requires monitoring for edema, hydrocephalus, and timely neurosurgical evaluation [1,14].

The 2026 AHA/ASA acute ischemic stroke guideline supports intravenous thrombolysis for eligible patients within the approved time window and recognizes alteplase or tenecteplase as options within 4.5 hours under the applicable selection criteria [14]. Posterior circulation location is not a reason to withhold reperfusion. A low NIHSS score should be interpreted alongside disability: inability to stand, severe dysphagia, diplopia, or disabling ataxia may be clinically consequential even when the numeric score is low.

Basilar artery occlusion is a distinct emergency. The ATTENTION trial (treatment within 12 hours) and BAOCHE trial (6 to 24 hours) demonstrated better functional outcomes with endovascular thrombectomy plus medical therapy than with medical therapy alone in selected patients [15,16]. The 2024 ESO/ESMINT guideline integrates these data and emphasizes clinical severity, occlusion location, baseline imaging, time, and treatment context; evidence is weaker for patients with NIHSS below 10, and extensive bilateral or brainstem ischemic change reduces the expected benefit [17]. Transfer to a thrombectomy-capable center should not be delayed when a potentially eligible occlusion is identified.

After hemorrhage is excluded, antiplatelet therapy is standard for most non-cardioembolic TIA or ischemic stroke patients who are not receiving a conflicting antithrombotic regimen. Short-course dual antiplatelet therapy is appropriate only for defined groups such as early minor non-cardioembolic stroke or high-risk TIA and selected symptomatic intracranial atherosclerotic disease; it is not intended as

indefinite treatment [12,13,18]. In INSPIRES, clopidogrel plus aspirin initiated within 72 hours reduced new stroke (7.3% vs 9.2%) but increased moderate-to-severe bleeding (0.9% vs 0.4%), illustrating the need to balance ischemic and hemorrhagic risk [13].

For atrial fibrillation or another established cardioembolic source, oral anticoagulation generally replaces antiplatelet therapy for long-term prevention, with initiation timing individualized to infarct size, hemorrhagic transformation, renal function, and bleeding risk [12]. Cervical artery dissection is managed with an individualized antithrombotic choice, commonly for three to six months, followed by clinical and imaging reassessment [11]. Combining antiplatelet and anticoagulant therapy without a separate indication increases bleeding and should be avoided.

Routine intracranial or vertebral artery stenting is not first-line therapy for stable symptomatic atherosclerotic stenosis. CASSISS found no significant advantage of adding stenting to medical therapy for symptomatic severe intracranial atherosclerotic stenosis [18]. ESVS guidance similarly prioritizes best medical therapy for vertebral disease, reserving revascularization questions for carefully selected patients with recurrent ischemia despite optimized treatment and multidisciplinary review [9]. Rare dynamic vertebral compression requires confirmation of a reproducible mechanical lesion before surgical or endovascular intervention is considered [20].

Prevention and longitudinal care

Prevention begins before discharge. The etiologic label should be explicit, the antithrombotic plan should include duration and review date, and the patient should receive a written list of warning symptoms that require emergency reassessment. Recurrent vertigo, diplopia, dysarthria, weakness, severe imbalance, or altered consciousness after a recent event should not be managed solely by routine outpatient follow-up.

For atherosclerotic vertebrobasilar disease, intensive medical management combines antiplatelet therapy, high-intensity statin treatment when tolerated, blood-pressure control, diabetes management, smoking cessation, physical activity, healthy dietary patterns, and weight management. AHA/ASA prevention guidance commonly targets blood pressure below 130/80 mmHg and low-density lipoprotein cholesterol below 70 mg/dL for many patients with atherosclerotic stroke, while emphasizing individual tolerance and comorbidity [12]. Medication intensification is useful only when paired with follow-up laboratory and safety monitoring.

Cardioembolic prevention requires reliable anticoagulant access and adherence, renal-dose review, and avoidance of unnecessary concurrent antiplatelet therapy. When initial monitoring is unrevealing but an embolic pattern remains unexplained, prolonged rhythm monitoring can detect intermittent atrial fibrillation. Dissection

prevention focuses on the prescribed antithrombotic course, reassessment of vessel healing, and individualized advice regarding high-strain neck activity during the healing phase [11,12].

Lifestyle treatment is part of the neurological prescription. Smoking cessation should include counseling and pharmacologic support when appropriate. A Mediterranean-style or similarly cardioprotective diet, regular aerobic activity adapted to neurological limitations, moderation of alcohol, and evaluation for sleep apnea can reduce the total vascular risk burden. Diabetes goals are individualized; avoiding both chronic hyperglycemia and treatment-related hypoglycemia is essential.

Rehabilitation should begin once medically stable and address gait, balance, limb coordination, swallowing, speech, vision, cognition, fatigue, and return to daily activities. Posterior circulation stroke may leave substantial disability despite a modest NIHSS score; outcome review should therefore include functional mobility and participation, not only limb strength. Falls risk, driving, work, and caregiver education should be revisited at follow-up.

An implementation-oriented follow-up schedule includes an early visit to verify medication access and review recurrent symptoms, subsequent checks of blood pressure and lipids, rhythm-monitoring results when ordered, and repeat vascular imaging when it will change treatment. Adherence barriers—cost, adverse effects, health literacy, and complex dosing—should be documented as clinical risk factors rather than treated as patient failure.

Table 3. Treatment and prevention pathway by clinical state

Clinical state	Immediate action	Disease-modifying strategy	Prevention/follow-up
Suspected acute posterior stroke	Stroke code; last-known-well time; NCCT plus urgent CTA/MRA; swallow and deterioration monitoring	Assess IV thrombolysis eligibility; thrombectomy evaluation if treatable occlusion is present	Determine mechanism before discharge; begin rehabilitation and risk-factor plan
Basilar artery occlusion	Urgent transfer/consultation with thrombectomy-capable center	Endovascular therapy in selected patients plus best medical care [15-17]	Etiology-specific antithrombotic treatment and close neurological follow-up

Clinical state	Immediate action	Disease-modifying strategy	Prevention/follow-up
Non-cardioembolic TIA/minor stroke	Rapid vascular and cardiac assessment; exclude hemorrhage	Antiplatelet therapy; short DAPT only when trial/guideline criteria are met [12,13]	Statin, BP control, smoking cessation, diabetes care; avoid long-term DAPT
Cardioembolic mechanism	Define infarct burden and hemorrhagic risk; rhythm and cardiac assessment	Oral anticoagulation at individualized timing; avoid unnecessary combination therapy	Adherence, renal function, interactions, and rhythm/structural-heart follow-up
Vertebral artery dissection	Image cervical and intracranial vessels; assess for ischemic complication	Individualized antithrombotic therapy, commonly 3-6 months [11]	Repeat clinical/imaging review; graded advice on neck strain during healing
Symptomatic atherosclerotic stenosis	Confirm lesion is causally related to symptoms and infarct pattern	Intensive medical therapy; routine stenting is not first line [9,18]	LDL/BP targets, adherence checks, recurrent-event surveillance; MDT if refractory
Rare dynamic compression	Document reproducible positional symptoms and dynamic vascular compromise	Specialist multidisciplinary decision; decompression or revascularization only if confirmed	Avoid nonspecific diagnosis based on cervical imaging alone; follow symptoms and vessel status

Source: authors' synthesis from references [9-18,20].

Interpretation of the evidence

The strongest recent therapeutic evidence applies to selected basilar artery occlusion and to short-term dual antiplatelet therapy in narrowly defined non-cardioembolic populations. These results cannot be generalized to chronic nonspecific dizziness, all mild posterior strokes, or indefinite combination therapy. Similarly, the absence of benefit from routine intracranial stenting does not eliminate a role for rescue

or highly selected multidisciplinary procedures; it establishes intensive medical therapy as the default starting point [13,15-18].

Diagnostic evidence is complementary rather than competitive. Clinical examination creates pretest probability, CT excludes hemorrhage, angiography defines the vessel, MRI defines tissue, and cardiac or laboratory studies clarify mechanism. The safest strategy is therefore not "CT versus MRI" or "HINTS versus imaging," but a sequenced use of each tool according to syndrome, examiner expertise, urgency, and resource availability.

CONCLUSION AND RECOMMENDATIONS

Vertebrobasilar insufficiency is a working syndrome that demands localization and etiologic clarification. The most defensible pathway recognizes high-risk posterior circulation patterns, performs urgent vessel imaging when occlusion or dissection is possible, uses MRI-DWI to define tissue while respecting early false negatives, and matches reperfusion and antithrombotic therapy to time, disability, vessel status, and mechanism. Prevention is not a single drug: it is an integrated program of mechanism-specific antithrombotic treatment, vascular risk-factor control, rehabilitation, adherence support, and surveillance for recurrence.

- Use the label VBI only as a provisional syndrome; document the clinical syndrome, tissue findings, vessel lesion, and likely mechanism.
- Activate an acute stroke pathway for central acute vestibular syndrome, disabling posterior deficits, progressive bulbar or bilateral motor signs, or suspected basilar artery occlusion, even when NIHSS is low.
- Combine NCCT with timely CTA or MRA when a treatable vascular lesion is possible, and repeat or complement MRI when clinical suspicion remains high after an early negative DWI study.
- Evaluate reperfusion eligibility without delay; refer selected basilar artery occlusion for thrombectomy assessment using current clinical and imaging criteria.
- Choose antiplatelet, short-course dual antiplatelet, or anticoagulant therapy according to the established mechanism and avoid routine long-term combination treatment.
- Prioritize intensive medical prevention and rehabilitation; reserve vertebral or intracranial revascularization for carefully selected refractory cases after multidisciplinary review.
- Schedule measurable follow-up for blood pressure, lipids, diabetes, smoking, rhythm monitoring, medication adherence, recurrent symptoms, and functional recovery.

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