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The management of acute isolated vertigo in the emergency department: a mini review

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Abstract

Acute vertigo is defined as the sensation of movement of oneself or the environment in the absence of actual motion, and it represents a common reason for admission to the emergency department. This symptom is associated with high healthcare resource utilization and prolonged hospital stays. However, the diagnostic yield of brain imaging in the acute phase remains low, given the limited sensitivity of both CT and MRI in detecting central causes of vertigo. In contrast, clinical examination has proven to be highly accurate and efficient when performed by experienced clinicians. Over recent years, several clinical algorithms have been developed to improve the diagnostic accuracy of emergency physicians in evaluating patients with acute vertigo. Among these, the HINTS and STANDING algorithms have undergone external validation in emergency settings, demonstrating good diagnostic performance. The objective of this review is to provide scientific evidence to support clinical decision-making for physicians assessing adult patients presenting with acute vertigo in the emergency department.

Key words: vertigo, dizziness, nystagmus, benign paroxysmal positional vertigo, acute peripheral vestibular dysfunction, ischemic stroke.

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Highlights

- Acute vertigo is a common emergency presentation; clinical assessment and nystagmus evaluation outperform early neuroimaging in diagnostic accuracy;
- Most cases are peripheral (BPPV, acute peripheral vestibular dysfunction), while central causes (stroke, neoplasms) are less frequent but critical to identify;
- Validated bedside algorithms such as HINTS and STANDING reliably differentiate peripheral from central vertigo;
- Management includes canalith repositioning maneuvers for BPPV, symptomatic pharmacotherapy and steroids for APVD, and timely reperfusion for ischemic stroke;
- Structured ED pathways integrating triage, diagnostic evaluation, and disposition optimize patient outcomes and resource utilization.

Abstract

Acute vertigo is defined as the sensation of movement of oneself or the environment in the absence of actual motion, and it represents a common reason for admission to the emergency department. This symptom is associated with high healthcare resource utilization and prolonged hospital stays. However, the diagnostic yield of brain imaging in the acute phase remains low, given the limited sensitivity of both CT and MRI in detecting central causes of vertigo. In contrast, clinical examination has proven to be highly accurate and efficient when performed by experienced clinicians. Over recent years, several clinical algorithms have been developed to improve the diagnostic accuracy of emergency physicians in evaluating patients with acute vertigo. Among these, the HINTS and STANDING algorithms have undergone external validation in emergency settings, demonstrating good diagnostic performance. The objective of this review is to provide scientific evidence to sup-

port clinical decision-making for physicians assessing adult patients presenting with acute vertigo in the emergency department.

Introduction

Acute vertigo is defined as the perception of movement of oneself or the surroundings in the absence of actual motion.[1] It is a frequent cause of Emergency Department (ED) admissions, accounting to 2.1-3.6% of visits in the USA.[2] The utilization of medical resources and the duration of hospital stay for these patients surpass those associated with other similarly prevalent presentations.[2,3] In Italy, the prevalence of ED admissions for vertigo is comparable, with a notable inclination towards requesting brain imaging tests. In fact up to 70% of patients undergo such examinations in Italian EDs.[4-6] However, the accuracy of brain imaging is low, considering the limited sensitivity of both CT and MRI for diagnosing strokes within the initial 12 h.[7] CT exhibits a sensitivity of approximately 10% in detecting posterior circulation ischemic strokes,[8] and the likelihood of a negative MRI is five times higher than that for a stroke occurring in the anterior circulation.[9] Therefore, imaging tests can provide false reassurance in the event of negative results.[10,11] On the other hand, clinical examinations and the accurate assessment of nystagmus have proven to be highly accurate and efficient when performed by experts.[12,13] However, literature data highlight that emergency physicians often do not employ these skills or use them incorrectly.[14] Several clinical algorithms have been introduced in recent years, designed to enhance the diagnostic accuracy of emergency physicians when evaluating this specific pathology.[5,15-18] Both the 'HINTS' and 'STANDING' algorithms have undergone external validation, showing a good diagnostic accuracy.[14,19] Moreover, there have been recent publications of international guidelines dedicated to the management of patients with vertigo in the ED.[20] The objective of this mini review is to furnish scientific evidence supporting the routine clinical assessment of adult patients with acute vertigo, particularly in cases without clear associated neurological signs.

Materials and Methods

The approach adopted to define the management of patients presenting with acute vertigo was based on a consensus document rather than a systematic review. This choice was driven by the limited availability of high-quality scientific evidence, particularly randomized controlled trials, and by the inherent difficulty of translating the complexity of the topic into rigorous evidence-based recommendations. Accordingly, the development of a consensus document was deemed the most appropriate method to reflect the shared expert opinion of the authors, who were selected for their recognized authority in the field.

Results

Frequent causes of vertigo

Isolated vertigo typically is referred to as an inner ear disorder. Nevertheless, it may occasionally indicate an underlying brain pathology, including a stroke, neoplasm, or a demyelinating disease.[21,22] The diagnostic process is frequently challenging, given that patients may use the term "vertigo" to describe different conditions, such as orthostatic hypotension, postural instability, bradycardia, anxiety and panic disorders linked to hyperventilation, hypoglycemia, anemia, and electrolyte abnormalities.[23] Among the peripheral forms, the most prevalent cause is Benign Paroxysmal Positional Vertigo (BPPV),[24] characterized by short-term vertigo induced by head movements and identified through bedside diagnostic maneuvers.[25] The second most common cause of vertigo in the ED population is Acute Peripheral Vestibular Dysfunction (APVD).[26] This term encompasses various conditions including labyrinthitis, vestibular neuritis and vascular vestibulopathy.[27] The prevalence of central diseases varies significantly among studies. Some investigations have identified central causes in 3–6% of cases, with the majority attributed to stroke.[2,28,29] In contrast other authors[30] found that central forms constitute nearly a quarter of patients with vertigo, but they also include transient vascular disorders of the posterior circulation, Vestibular Migraine (VM), multiple sclerosis, tumors of the posterior cranial fossa, and toxic causes.

Clinical approach for differential diagnosis

Concerning acute isolated vertigo, the initial questions should primarily aim to ascertain whether the symptom can be categorized as peripheral vertigo or not. The questions should include the evaluation of triggering factors, duration, prodromes or associated symptoms, ability to maintain an upright position, and cardiovascular risk profile

In recent years, scientific evidence advocates for an approach centered on timing and triggers rather than focusing on the quality of symptoms.[20] This approach allows for the identification and differentiation of three potential vestibular syndromes: i) Acute Vertiginous Syndrome (AVrS), and ii) Episodic Vertiginous Syndrome (EVrS), that is further categorized into Spontaneous EVrS [s-EVrS] ot Triggered EVrS [t-EVrS]

Acute vertiginous syndrome

Acute vertiginous syndrome is characterised by a sudden onset of vertigo lasting more than 24 h and it is associated by autonomic symptoms such as nausea and vomiting, along with postural instability in the absence of auditory symptoms. It may arise from either a sudden dysfunction of a peripheral vestibular receptor or a dysfunction of the central vestibular structures within the central nervous system. The most frequent peripheral cause is APVD, generally attributed to Viral Neuritis (VN).[32,33] The clinical features of this condition result from an isolated lesion of the peripheral vestibular receptor. However, AVrS can be the initial sign of an ischemic stroke involving the brainstem or cerebellum, Menière's Disease (MD) or Vestibular Migraine (VM). The coexistence of sudden hearing loss is suggestive for an ischemic syndrome of the Anterior Inferior Cerebellar Artery (AICA) because it gives rise to the internal auditory artery with its cochlear branch.

Episodic vertiginous syndrome

Episodic vertiginous syndrome represents transient and recurrent episodes of vertigo that last from seconds to minutes or hours. Two distinct types are acknowledged: those without triggering factors, termed spontaneous, and those with triggering factors, referred to as provoked.[20]

Spontaneous episodic vertiginous syndrome

The duration of spontaneous episodic vertigo is from minutes to a few hours. Upon admission to the ED, patients with these symptoms are often asymptomatic, thus the diagnosis relies on the medical history. s-EVrS is most associated with benign conditions such as VM, MD, reflex syncope, and panic attacks. Less common

but potentially dangerous causes include cerebrovascular diseases (vertebrobasilar TIA) and non-vestibular conditions (pseudo-vertigo), such as cardio-respiratory diseases, endocrinopathies, and intoxications.[20]

Triggered episodic vertiginous syndrome

Episodes of vertigo can be triggered by specific actions or events, with the most common triggers being changes in head or body position. Less common triggers include intense sounds or the Valsalva maneuver. The duration of each episode of dizziness varies, lasting from a few seconds to minutes. The most frequent causes of t-EVrS include BPPV and orthostatic hypotension. Among dangerous causes, it is crucial to consider forms of Central Paroxysmal Positional Vertigo (CPPV), which may arise from expansive lesions in the posterior cranial fossa. This condition is typically associated with other neurological signs and symptoms.[20]

Neurological examination

In patients presenting with an acute onset of vertigo syndrome, a comprehensive neurological examination is essential. Utilizing the following checklist can help identify: i) language disorders; ii) ocular motility disorders; iii) disorders of the bulbar cranial nerves; iv) Bernard-Horner syndrome; v) visual field defects; vi) motor deficits; vii) sensory deficits; viii) cerebellar deficits; ix) plantar cutaneous reflex in extension. The presence of at least one of these signs should be considered indicative of a central origin of vertigo.[6]

One of the main steps is the evaluation of balance and gait. A simple method to use in everyday clinical practice is defined by Carmona *et al*.,[34] in particular Grade 0 and 1 showed a high negative predictive value to exclude stroke (> 90%) (Table 1). Concerning vascular etiology neurological signs and symptoms vary based on the specific vascular district involved.

The evaluation of nystagmus

The observation and analysis of nystagmus are fundamental in the evaluation of patients with acute vertigo. Observing nystagmus necessitates both fixation and non-fixation conditions, as fixation tends to mask nystagmus originating peripherally. When assessing for nystagmus, it is essential to conduct examinations in the primary position of gaze, both with and without fixation. This should be done with the patient looking straight ahead with the head still, in both supine and sitting positions. The examiner should carefully observe the following aspects: i) determine whether nystagmus is present; ii) identify the plane on which the nystagmus beats; iii) determine the direction of the nystagmus; iv) observe the temporal trend; v) evaluate how the nystagmus responds to changes in gaze position; vi) change the patient's head position and observe how the nystagmus responds.[6]

Clinical tests

Head impulse test

In a normal scenario, when a patient focuses on a target and his head is swiftly rotated in one direction, the eyes show a compensatory movement in the opposite direction to maintain fixation on the target. This reaction is based on the Vestibulo-Oculomotor Reflex (VOR), relying on the intact functionality of the semicircular canals. In the case of damage of the lateral semicircular canal on the side toward which the head is rotated, the eyes will exhibit a diminished or absent counter-rotation when the head moves toward the affected ear. Consequently, the patient loses sight of the target, followed by a rapid "saccadic" movement to regain focus on the fixed object. This corrective movement is directed toward the healthy ear. The positivity of HIT toward one side, as opposed to the other, serves as a highly indicative sign of labyrinthine damage and peripheral origin of vertigo. The side of positivity corresponds to the direction in which the patient's head is rotated (Figure 1).[6]

Cover test

In certain cases of vertigo a vertical misalignment of the eyes may be present, with one eye positioned higher than the other. The cover test involves covering one eye at a time and observing the movement of the uncovered eye (Figure 1). If, upon covering the lower eye, the higher eye moves downward, and vice versa, the test is considered positive. This finding is highly indicative of central pathology. If a realignment of the eyes is not observed, the test is considered negative and does not provide conclusive evidence regarding the location of the underlying damage.[15]

Positional manoeuvres

The main clinical maneuvers performed at the patient's bedside and fundamental for the identification of BPPV are represented by the Dix-Hallipike and Pagnini-McClure maneuvers.

In the Dix-Hallpike maneuvers, the patient begins in a seated position with legs on the table. The examiner gradually rotates the patient's head approximately 45° to one side and quickly transitions him to a supine position, with the head hyperextended out of the table. The rapid phase of the nystagmus is directed towards the right shoulder (counterclockwise-up) for right-sided stimulation and towards the left shoulder (clockwise-up) for left-sided stimulation. A latency period is often observed. The nystagmus is paroxysmal, intensifying rapidly and then diminishing.

Regarding the Pagnini-McClure maneuver, the patient is asked to remain in a sitting position or supine for at least 5 min. When the patient turns their head toward the affected ear or lies on the same side, it results in 'geotropic' nystagmus, characterized by the rapid phase beating towards the affected ear and, consequently, towards the ground. The transition to the opposite side induces a 'geotropic' nystagmus with a rapid phase consistently directed towards the ground. Thus for example, a right nystagmus when the

Table 1. Assessment of gait unsteadiness (adapted from Carmona *et al.* 2016).[34]

Severity of gait unsteadiness	Definition	Positive predictive value of ataxia grade for stroke [128]
Grade 0	Normal	0% [n=0/5] had stroke
Grade 1	Mild to moderate imbalance with walking independently	7% [n=3/42] had stroke
Grade 2	Severe imbalance when standing or cannot walk without support	28% [n=11/39] had stroke
Grade 3	Falling at upright posture/inability to stand unaided	100% [n=28/28] had stroke

patient lies on the right side and a left nystagmus when lying on the left side. A more pronounced nystagmus is observed when the patient lies on the affected side [6].

Diagnostic algorithms: HINTS, STANDING, and Titrate

Numerous clinical tests have been evaluated to distinguish acute peripheral vertigo from acute central one. However, none of them has proven capable of an accurate differential diagnosis. For this reason, several diagnostic algorithms composed of multiple tests have been proposed. The first bedside diagnostic algorithm proposed is HINTS (Head Impulse Test, Nystagmus, Test of Skew): an otoneurological examination comprising three bedside tests, exclusively used in patients with AVrS. In the validation study, recruited patients had at least one risk factor for stroke. After applying the algorithm, all patients underwent brain MRI and those with suspected peripheral pathology underwent caloric tests. The authors define HINTS as "benign," suggesting a peripheral lesion, if the nystagmus is unidirectional, the HIT is positive and there is no skew deviation.

HINTS is deemed "dangerous" indicating a central form, if at least one of the following is present: i) negative HIT; ii) pure vertical or torsional spontaneous nystagmus or gaze-evoked nystagmus; iii) positive cover test

Subsequently, the authors introduced a fourth step, the bedside assessment of hearing loss, hence the term HINTS "plus".[17] Its presence may suggest a vascular origin due to the involvement of the AICA. HINTS achieves sensitivity values of 99% and specificity of 97% for the diagnosis of central form of vertigo, values superior to DWI-MRI performed in the first 24–48 h. Another proposed and validated diagnostic algorithm is STANDING (SponTAneous Nystagmus, Direction, Head Impulse Test, StandiNG; Figure 2). Developed through collaboration between vestibologists and emergency physicians, it requires a brief training period under the supervision of a physician experienced in vertigo evaluation.[4,5] This structured, four-step bedside algorithm

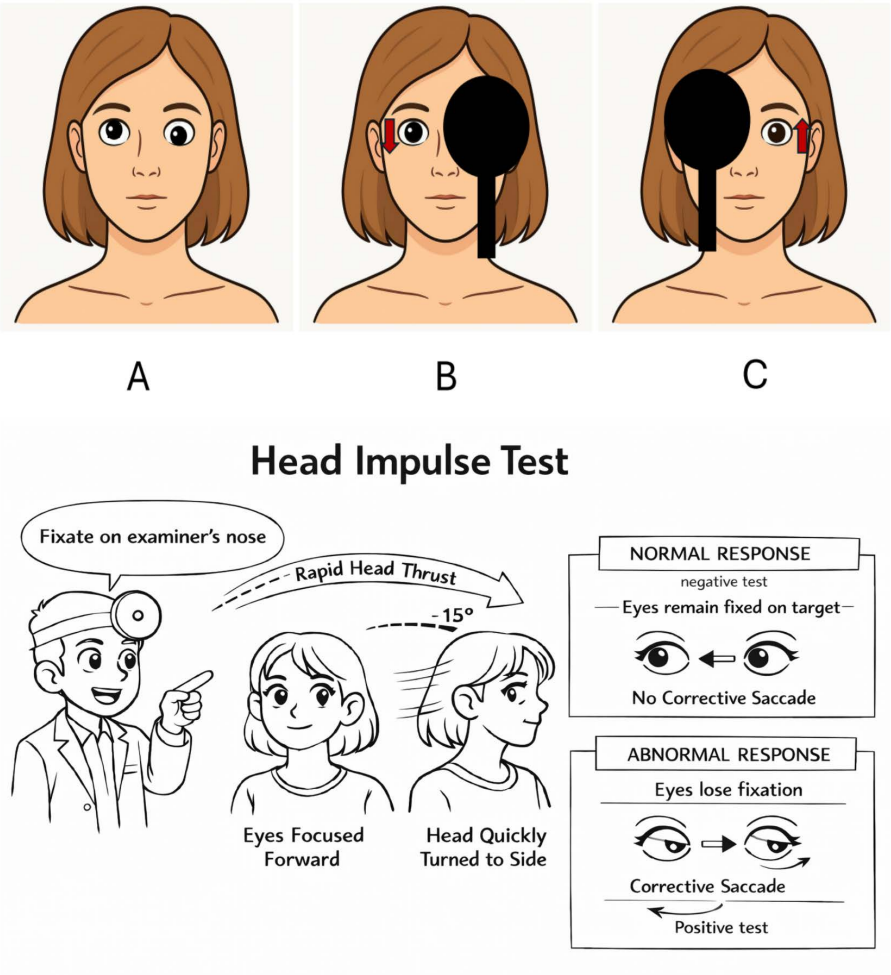

Figure 1. Cover test and Head Impulse Test. The dizzy patient (images on the top) sits in front of the examiner and sometimes a vertical misalignment is evident (**A**). If when covering the lower eye the higher eye moves downward and when covering the higher eye, the lower eye moves upward, the test is positive (**B-C**). If no realignment of the eyes is observed, the test is considered negative. The Head Impulse Test (images on the bottom) evaluates the vestibulo-ocular reflex by observing eye movements during rapid, passive head rotations. A normal response keeps gaze stable, while corrective saccades indicate vestibular dysfunction.

combines clinical signs with specific maneuvers performed directly at the patient's bedside.

STANDING has demonstrated high reliability and accuracy in detecting central vestibulopathy among unselected patients presenting to the emergency department with acute vertigo. The agreement between assessments performed by emergency physicians using STANDING and those conducted by vestibular specialists (considered the reference standard) was excellent, with a Cohen's K of 0.86 (and a concordance rate of 95.6%). The algorithm has also been externally validated in two studies published by emergency physicians in Paris[19,35] and in a multicenter study conducted in Tuscany.[35]

Both STANDING and HINTS provide accurate yet simplified methods for evaluating nystagmus. While the HINTS algorithm is best applied to patients with AVrS and spontaneous nystagmus, STANDING is suitable for assessing all cases of isolated vertigo, including positional forms. Notably, STANDING incorporates the HINTS components (except for the cover test), alongside the evaluation of positional nystagmus, upright posture, and gait. This allows for an initial differential diagnosis even in patients presenting with vertigo with or without spontaneous or positional nystagmus.

It is evident that both algorithms emphasize nystagmus evaluation within the diagnostic process. However, this should always be integrated with a targeted anamnesis and physical examination of other organ systems, particularly the neurological and cardiovascular systems. The TI.TR.A.T.E. (TIming, TRiggers, And Targeted Examination) represents a newer diagnostic framework designed to determine the probable etiology of acute vertigo by categorizing patients into four groups, combining anamnestic and objective clinical findings.[16] The TI.TR.A.T.E. has not been validated till now.

Treatment

Benign paroxysmal positional vertigo

The treatment for BPPV is physical due to its mechanical nature. The objective is to liberate the semicircular canal from otoconial debris, repositioning it into the utricle to facilitate reabsorption.

BPPV therapy of posterior semicircular canal

The Semont maneuver[19] and the Epley repositioning maneuver[38] are the most commonly employed techniques for treating this

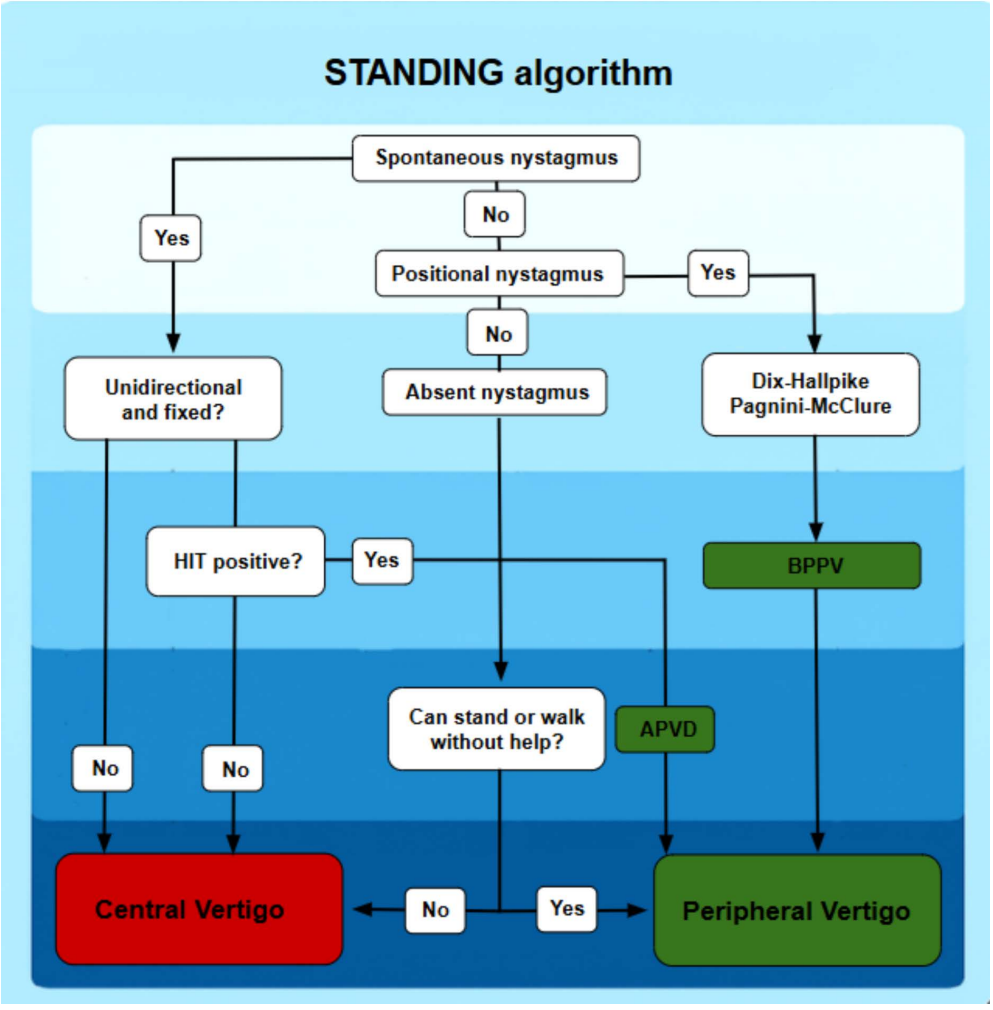

Figure 2. STANDING algorithm. HIT, Head Impulse Test; APVD, Acute Peripheral Vestibular Dysfunction; BPPV, Benign Paroxysmal Peripheral Vertigo.

form of BPPV. Literature suggests that both liberating maneuvers are equally effective, resolving positional symptoms in one or two sessions.[39] The choice between the Semont and Epley techniques depends on the examiner's preference and the characteristics of the nystagmus, particularly its intensity. If the nystagmus is not notably intense, the repositioning maneuver (Epley) may be more effective, leveraging the gravity of the debris, as opposed to the liberating maneuver (Semont), which relies on the inertia of the debris.

BPPV therapy of lateral semicircular canal

In the geotropic variant of BPPV, two frequently employed techniques are the Gufoni maneuver[40] and the Vannucchi forced liberating position.[41] The Gufoni maneuver aims to dislodge otoconial debris from the lateral semicircular canal by leveraging a rapid deceleration. Initiated with the patient in a seated position, the maneuver swiftly transitions the patient to a lateral position on the side corresponding to the healthy side. Vannucchi's forced liberating position necessitates the patient to maintain a lateral decubitus posture on the unaffected ear side for an extended period of 12 h. In this position, gravitational forces facilitate the movement of otoconial debris out of the affected canal towards the utricle. Due to its prolonged duration, this therapeutic approach is suitable for home treatment. When executed precisely and consistently, this maneuver boasts a symptom and sign resolution rate exceeding 90% within 24 h.[41] A "shortened" version lasting one hour has been recently proposed.

Drug therapy

Drug therapy plays a limited role in the management of BPPV; nevertheless, there are specific scenarios where pharmacological interventions can be beneficial. In patients exhibiting pronounced neurovegetative symptoms, the administration of antiemetic drugs may prove advantageous. Another circumstance warranting pharmacological consideration arises when a patient experiences post-treatment instability in the subsequent days. This phenomenon is relatively common and the use of antihistamines or anticholinergics, such as dimenhydrinate or cinnarizine, also in combinations, can be highly beneficial. It is recommended to sustain this pharmacotherapy for a few days to address and manage the observed instability associated with physical therapy.

Acute peripheral vestibular dysfunction

The lack of clear understanding of the etiopathogenesis of APVD poses challenges in prescribing pharmacological interventions. In practice, a substantial proportion of patients demonstrate gradual amelioration in vertigo within days following the acute episode. However, 20 to 50% of individuals may exhibit persistent and consequential symptoms, including static and dynamic instability and difficulty in maintaining steady visual fixation on an object.[42] The acute management of APVD involves the utilization of antiemetic and vestibule-suppressive medications to mitigate intense neurovegetative symptoms and reduce the severity of rotational vertigo. Vestibulo-suppressive drugs encompass anticholinergics, antihistamines and benzodiazepines. Antiemetic drugs are also valuable for alleviating neurovegetative symptoms, particularly when administered intravenously in the presence of pronounced emetic manifestations. Due to the presumed viral etiology of APVD, a brief corticosteroid regimen has been proposed,[43] with the potential to enhance clinical outcomes in affected individuals.[44] While a recent meta-analysis concludes that the available evidence is insufficient,[45] a retrospective study underscored that individuals treated with methylprednisolone (1 mg/kg for 5 days, followed by an additional 5 days at 0.5 mg/kg) exhibited superior outcomes in terms of vestibular signs and symptom recovery compared to those utilizing non-steroidal treatments. Despite these conflicting findings, it has been suggested that initiating steroid therapy within 24 h of APVD onset yields more favorable outcomes regarding the recovery of vestibular function in comparison to treatment initiated between 24 and 72 h post-onset.[46]

Ischemic stroke

For individuals presenting with recent-onset isolated vertigo, a key clinical challenge lies in determining whether the symptoms may be attributable to an acute, potentially treatable cerebral ischemic event. A review of randomized controlled trials evaluating fibrinolytic therapy for acute ischemic stroke highlights the efficacy of Recombinant Tissue Plasminogen Activator (rtPA) when administered systemically within 4.5 hours of symptom onset, provided that no contraindications to fibrinolysis exist. Evidence from meta-analyses confirms that the therapeutic benefit increases with earlier administration, reinforcing the importance of initiating treatment within the recommended time window.

Furthermore, multiple randomized clinical trials have demonstrated that endovascular revascularization – comprising mechanical thrombectomy and/or thromboaspiration within 6 hours of symptom onset in patients with confirmed large vessel occlusion – significantly improves functional outcomes. In clinical practice, patients presenting within 4.5 hours of onset should promptly receive rtPA. When large vessel occlusion is documented after systemic thrombolysis, mechanical thrombectomy is indicated. For those arriving between 4.5 and 6 hours after symptom onset, thrombectomy should be considered if imaging confirms large vessel occlusion.

Advances in neuroimaging have further extended the therapeutic window for revascularization, allowing treatment in selected patients whose symptoms persist beyond 6 hours. These sophisticated imaging modalities enable identification of a favourable "mismatch" profile—defined by a substantial area of salvageable ischemic penumbra relative to a smaller core of irreversible infarction. CT perfusion and MRI with diffusion–perfusion sequences are the primary techniques used to detect this pattern. On the basis of such imaging criteria, patients may be considered for systemic rtPA administration between 4.5 and 9 hours from symptom onset, or when the exact time of onset is unknown, such as in wake-up strokes.[47]

Several studies have reported favourable outcomes from reperfusion therapies using endovascular approaches, extending the potential treatment window up to 24 hours after symptom onset.[48] Regarding posterior circulation strokes, a meta-analysis of randomized controlled trials including patients with basilar artery occlusion has demonstrated the superiority of mechanical thrombectomy over best medical management. This superiority encompasses reductions in both disability and mortality at 90 days, albeit with a higher incidence of symptomatic intracerebral hemorrhage.[49,50] Nevertheless, some uncertainty remains concerning the optimal implementation of this therapy in real-world clinical practice.

Suggested pathway of patient with acute vertigo

This chapter outlines an organizational model for managing acute vertigo in the ED, structured around three main phases (Figure 3).

Triage and early assessment

The primary objective is the rapid differentiation between potentially life-threatening and benign causes. We recommend using standardized queries during pre-hospital or triage assessment to collect key data on prodromes, triggers, symptom duration, and cardiovascular risk factors. Specifically, assessing standing ability using the Carmona scale[34] serves as a simple and effective screening tool. Patients unable to maintain a standing position or walk unassisted (Carmona class 2–3) warrant immediate medical evaluation for suspected acute cerebrovascular disease.

Diagnostic evaluation

The implementation of standardized algorithms (e.g., STANDING) is suggested to distinguish peripheral from central vertigo. Patients with a clinical profile clearly indicating a peripheral cause (e.g., BPPV) should be directed to an outpatient specialist fast-track (ENT/Vestibology), ideally within 24–48 hours. This pathway avoids unnecessary neuroimaging and prolonged ED stays, allowing specialists to confirm the diagnosis and perform repositioning maneuvers if indicated.[36] Conversely, if central pathology (e.g., stroke) is suspected, the stroke pathway must be activated immediately, involving the stroke team/neurologist and radiologist for urgent neuroimaging (CT/MRI).

Emergency department outcome (discharge, observation, or admission)

Final disposition relies on clinical complexity and the urgency of diagnostic tests following the ED physician's evaluation, occasionally requiring multidisciplinary collaboration (vestibologists, neurologists, radiologists). Patients requiring non-urgent workups may be discharged with a scheduled outpatient follow-up (ENT/vestibologist). For diagnostic uncertainties or further testing, the use of Short Stay Observation Units is crucial to resolve cases within 24-48 hours, thereby reserving hospital admission strictly for selected cases.

Discussion

The collaborative work among the various professional figures involved in the management of "vertigo" has highlighted the importance of open and constructive communication between spe-

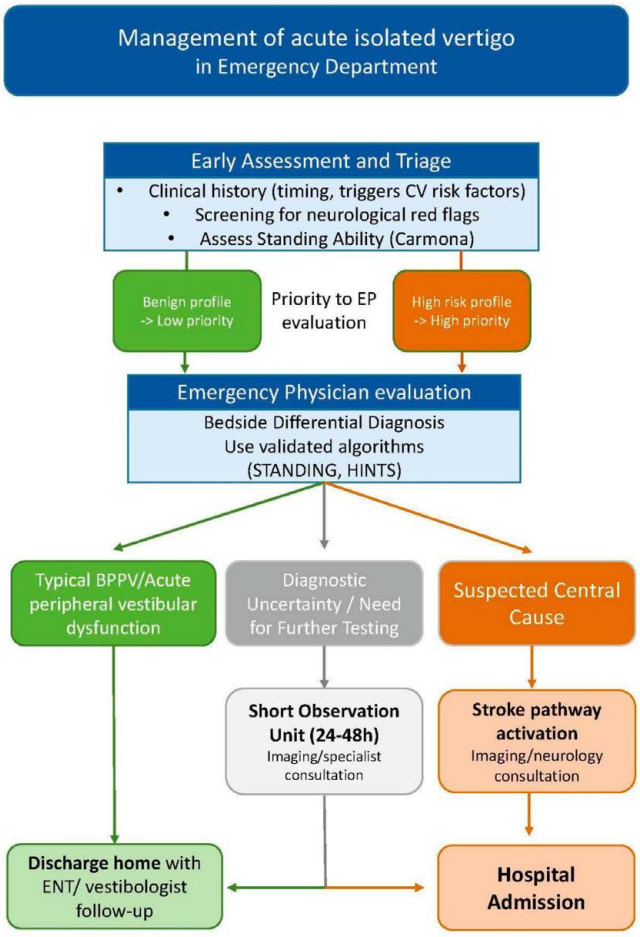

Figure 3. Management algorithm for acute isolated vertigo in the Emergency Department, based on early risk stratification and bedside evaluation using validated clinical tools. Depending on diagnostic findings, patients are discharged with specialist follow-up, observed for further testing, or admitted through stroke pathway activation.

cialists. Vertigo, being an inherently multidisciplinary symptom, necessarily requires observation, evaluation, and interpretation from multiple perspectives.

First and foremost, it is essential to establish a shared terminology for describing symptoms and, even more critically, for identifying and characterizing nystagmus. Whether it is spontaneous or positional, its direction, duration and reproducibility are aspects often taken for granted by some specialists but not yet well understood by most physicians working in emergency departments or in community healthcare settings. Moreover, reporting the presence of nystagmus in a vertiginous patient without specifying how it was observed and what its characteristics are does not aid diagnosis; on the contrary, it generates confusion and may lead the diagnostic process toward lengthy, costly, unnecessary, and sometimes even hazardous pathways.

Much work remains to be done to disseminate this culture and to implement the clinical and diagnostic protocols recommended in this document effectively. We wish to emphasize once again that without a concrete effort toward widespread dissemination and comprehensive training—through accredited scientific societies as well as institutional, academic, and national health service programs—the recommendations contained herein will remain goals to be achieved rather than established standards of good clinical practice.

Conclusions

Acute vertigo represents a frequent and challenging presentation in the emergency department, with significant implications for patient safety and healthcare resource utilization. Validated clinical algorithms such as STANDING provide reliable tools to distinguish peripheral from central causes of vertigo and may reduce unnecessary imaging and hospital admissions. Their systematic implementation in emergency practice has the potential to improve diagnostic efficiency, optimize resource use, and enhance patient outcomes. Further research is warranted to refine these approaches and to facilitate their broader integration into emergency care pathways.

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Online supplementary materials
Bullet points

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