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Cerebellar Infarctions Mimicking Acute Peripheral Vertigo: How to Avoid Misdiagnosis?

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Abstract

Objective. To determine the prevalence of cases of missed cerebellar stroke mimicking acute peripheral vertigo (APV), the so-called pseudo-APV, and to identify the clinical indicators useful for differentiating APV from cerebellar infarction that presents as isolated vertigo.

Study Design. Case series with chart review.

Setting. Tertiary referral center.

Subjects and Methods. We conducted a retrospective chart review of cases of missed cerebellar infarction over the past 5 years. All patients had first undergone an otoneurological evaluation and computed tomography brain scan in the emergency department before a complete bedside examination was performed in our otoneurological unit.

Results. We identified 11 patients with pseudo-APV (2.8% of all the cases presenting to our unit complaining of acute vertigo). Spontaneous nystagmus (of central type in 2 cases) was recorded in all patients. The Head Impulse Test was clearly negative in 9 cases. The duration of vertigo lasted more than 72 hours in 7 patients. In 4 patients, delayed neurological signs followed acute vertigo 2 to 3 days after the onset. Magnetic resonance imaging showed 8 cases of infarction in the posterior-inferior cerebellar artery territory; in 1 patient, an involvement of the anterior-inferior cerebellar artery territory was recorded; 2 patients showed a hemispheric ischemic cerebellar involvement.

Conclusions. Pseudo-APV is not an uncommon diagnosis in otoneurological practice. The presence of moderate-severe imbalance and the persistence of vertigo for more than 72 h from the onset, together with the results of bedside examination tests (spontaneous nystagmus and Head Impulse Test), are useful indicators for recognizing a cerebellar ischemic origin in cases of acute vertigo.

Keywords
vertigo, acute vestibular syndrome, cerebellar infarction, differential diagnosis, pseudo–acute peripheral vestibulopathy, nystagmus, bedside examination

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Acute peripheral vertigo (APV), the third most common cause of peripheral vestibular involvement,1 represents 2.5% of the cases in a typical emergency department.2 APV is a clinical disorder caused by acute unilateral damage of the peripheral vestibular structures, which is characterized by long-lasting (more than 24 hours) rotatory vertigo, spontaneous nystagmus, postural instability, and neurovegetative symptoms without signs of cochlear and brain stem involvement.3 The ethiopathogenesis of APV still remains a matter of debate. Vestibular neuritis is considered the most frequent cause of APV.1,3 An ischemic origin of APV has been suggested4,5 on the basis of the frequent association of APV with blood hyperviscosity,6 alterations of the hemostatic system,7 or dysfunction or microvascular endothelium.8 While the most common manifestations of cerebellar infarction are vertigo and/or dizziness,9,10 in these cases other cerebellar symptoms or signs usually accompany these symptoms. Nevertheless, focal cerebellar stroke, especially in the posterior-inferior cerebellar artery (PICA) area of vascularization, may mimic APV, with acute vertigo being the only presenting symptom,10,11 the so-called pseudo-APV. Early detection of a pseudo-APV has a dual diagnostic and prognostic relevance: first, the evolution toward a large cerebellar infarction represents a dangerous clinical condition because of brain stem compression and increased intracranial pressure, and second, a large percentage of small cerebellar stroke is usually caused by
an often-misunderstood cardiogenic embolism, the recognition and treatment of which can prevent further life-threatening brain stem and/or cerebellar stroke.

Despite a recent large population-based study showing that isolated dizziness, vertigo, or imbalance strongly predict a noncerebrovascular cause, small observational studies estimate that almost 25% of acute vestibular syndromes could be due to posterior fossa infarctions. Many further difficulties are encountered in the attempt to evaluate the frequency of misdiagnosis of pseudo-APV, the consequences of which could lead to adverse outcomes in 40% of cases.

The fact that brain magnetic resonance images (MRIs) are not always readily available and the possibility of a false-negative result make bedside predictors extremely useful in the early detection of pseudo-APV. A key point to note is that the Head Impulse Test (HIT) is negative in cerebellar stroke presenting with isolated vertigo.

We reviewed a series of patients referred to the ear, nose, and throat (ENT) department with a diagnosis of APV (vestibular neuritis) to identify the clinical features and the diagnostic investigations useful for avoiding pitfalls in the diagnosis of pseudo-APV.

Patients and Methods

We conducted a retrospective chart review including all patients evaluated over the past 5 years in the Otoneurological Unit of Pisa University Hospital (tertiary referral center) with a diagnosis of isolated acute spontaneous vertigo. In the acute care setting, patients suffering from vertigo first undergo a bedside clinical evaluation of the vestibular signs performed by an emergency department (ED) physician and a computed tomography (CT) brain scan. The next step is to evaluate the presence of neurological signs in order to rule out a central disorder. If the clinical presentation does not show signs of central involvement, an APV is suspected, and the patient is referred to the Otoneurological Unit, where a complete bedside examination, pure-tone audiometry, and caloric responses are performed. Bedside examinations consist of a search for spontaneous, gaze-evoked, and positional nystagmus (tested with and without Frenzel’s glasses), a head-shaking test (HST), and a HIT. Imbalance was evaluated as follows: normal (the patient is able to stand on tandem Romberg for 3 seconds with eyes open), mild (the patient is unable to stand on tandem Romberg at least for 3 seconds with eyes open), moderate (unable to stand on Romberg for at least 3 seconds with eyes open), and severe (the patient is unable to stand up without help).

We identified 11 patients who had clinical symptoms mimicking APV with a subsequent diagnosis of cerebellar stroke; 9 cases were directly admitted to our unit from the ED, while 2 subjects were evaluated after a previous hospitalization in a secondary care center (after 5 and 23 days, respectively, from the onset of acute vertigo).

Approval of the study was granted by the local Ethics Committee (Comitato Etico dell’Azienda Ospedaliera-Universitaria Pisana).

Results

The clinical features, diagnostic tests, and results of brain imaging are summarized in Table 1.

In the past 5 years (2007-2011), 392 patients suffering from APV were referred to the Otoneurological Unit of our ENT department. Eleven patients (2.8%), 6 women and 5 men, with a age at presentation ranging from 47 to 80 years (mean, 62.36 years), were subsequently diagnosed as pseudo-APV. In all the cases, the clinical features presented as a sudden onset of rotational vertigo associated with neurovegetative symptoms (nausea and vomiting). Most of the patients (10 of 11) presented 1 or more cardiovascular risk factors, such as hypertension (8 cases), obesity (3 cases), type 2 diabetes (1 case), atrial fibrillation (1 case), previous heart attack (1 case), and abnormal blood lipid levels (3 cases).

Three patients complained of headache. Two patients described a unilateral hearing loss: the onset was reported as sudden in 1 case (patient 3), while the other (patient 7) had a previous diagnosis of Ménière’s disease and the hearing loss was considered as part of the acute stage of the disease. The neurological examination and CT scan performed at the time of the presentation of the acute vertigo was negative in all the cases.

With regard to the 9 patients referred to us from the ED, the otoneurological examination revealed a spontaneous nystagmus in all cases. In 7 cases, the spontaneous nystagmus (II in 5 patients, III in 2 patients) had the typical features of a peripheral lesion (horizontal unidirectional, enhanced after HST). A central involvement was suspected in 2 patients: in 1 case (patient 4), a direction-changing gaze-evoked nystagmus was recorded, and in the other (patient 9), the unidirectional horizontal nystagmus in the primary gaze became rather vertical downbeating when the patient was looking up. A HIT was performed in all 9 patients: in 8 cases, it was clearly normal (no observed saccade), while in 1 patient (patient 7), it proved to be not definitely negative. Imbalance was severe in 6 cases and moderate in 3. The duration of acute vertigo associated with a severe-moderate level of imbalance lasted for more than 72 hours in 6 patients.

Two other patients (patients 10 and 11) were previously evaluated in facilities other than ours and at the time of presentation of APV had spontaneous horizontal unidirectional nystagmus. The HIT was studied only in patient 10 and resulted negative. This latter patient was referred to our center after 5 days from the acute presentation because of the persistence of the clinical symptoms (gait ataxia and vertigo) that showed no improvement despite a steroid and vasoactive treatment. Patient 11 came to our observation after 23 days from acute onset because of the persistence of moderate imbalance despite the disappearance of the spontaneous nystagmus. In this case, a normal response to the caloric test associated with the anamnestic recording of slight dysarthria during the acute presentation (this symptom was not registered during the previous hospitalization) indicated a neuroimaging study of the brain.
The persistence of severe vertigo and/or imbalance after 72 hours from the presentation and the presence of a moderate-severe imbalance furnished the main indication for proceeding with a brain MRI; the indication for MRI was corroborated in 4 cases because of the onset of delayed (18 hours–3 days from appearance of acute vertigo) focal neurological signs (3 patients showed limb ataxia, and 1 patient had limb ataxia and double vision). The presence of a normal HIT (associated in 2 cases with a spontaneous nystagmus of central type) represented a further indication for an MRI investigation of the brain.

The results of the MRI (available within 1-6 days in 10 of 11 patients; only in patient 2 was the MRI performed after 35 days) showed in all cases a cerebellar infarction. Two patients (patients 7 and 9) reported an extensive hemispheric cerebellar ischemic area. Patient 3 showed a well-defined involvement of the territory supplied by the anterior inferior cerebellar artery. In the remaining patients, the MRI showed a small area of cerebellar infarction (Table 1). All of these patients were therefore referred to the neurological unit.

Further investigations demonstrated in 1 case (patient 5) a vertebral artery dissection, and in 2 cases (patients 6 and 11), cerebellar infarction arose from emboli originating from the heart (patent foramen ovale).

**Discussion**

Vertigo and imbalance are the most common symptoms in patients with isolated cerebellar infarctions, and they appear without any other signs of cerebellar dysfunction in approximately 38% of patients. Several recent papers clearly point out how closely a cerebellar stroke may mimic an APV. Usually when the clinical pattern of a cerebellar stroke mimics APV, the ischemic area is located in the territory supplied by the medial branch of PICA. This territory includes key structures (nodulus and uvula) strictly connected with the ipsilateral vestibular nuclei and receiving direct projections from the labyrinth.

The differentiation between APV and pseudo-APV is very challenging, and several ways to tell the difference clinically have been proposed, principally depending on a careful history and physical bedside examination. Known vascular risk factors have to be evaluated to identify those patients at high risk of vascular origin of their vertigo. In our series, all of the patients except 2 showed vascular risk factors. In our study, the mean age at presentation was 63.
years, and it is noteworthy that the younger (47 years old) of the 2 patients with no cardiovascular risk factors developed a pseudo-APV as a consequence of a vertebral artery dissection. This condition is considered the leading identifiable cause of cerebellar stroke in young adults\(^{17,28}\) and can occasionally present with a pseudo-APV.

Some hallmark examination signs are described to assess the risk of stroke in acute vertigo presentation. First, the characteristics of spontaneous nystagmus: in our study, 9 of the 11 patients presented a spontaneous unidirectional nystagmus, which resembled damage to the labyrinth, confirming that this kind of presentation is rather common in isolated cerebellar stroke.\(^{21,24}\) In 2 cases, the presence of spontaneous nystagmus of the central type clearly indicated possible central nervous system involvement, inducing us to perform an immediate neuroimaging study.

Second, several papers have affirmed that HIT is perhaps the best indicator for a central lesion with a pseudo-APV presentation.\(^{11,14}\) The HIT was reported as normal (although with some uncertainty in patient 7) in all of our patients (except in 1 case in which the test was not performed) even in the presence of spontaneous nystagmus. Our results seem to confirm that a negativity of HIT in patients with spontaneous unidirectional nystagmus could suggest central lesions. Nevertheless, it is well documented that the sensitivity of the HIT depends on the degree of canal paresis, as well as on the phase of the disease,\(^{29}\) being about 100% only in the case of complete vestibular loss and in the acute phase of AVS, and more than 50% of canal paresis is needed for the HIT to be positive.\(^{30}\) Furthermore, small fast corrective saccades could occur during the head rotation that are impossible to detect with the naked eye (covert saccades).\(^{31}\) A correct interpretation of the HIT requires a high degree of skill and experience, not always available in frontline clinicians, and our study design does not allow us to really assess the value of HIT for detecting a central nervous system involvement. More information regarding the real impact of HIT in detecting pseudo-APV could be obtained by the evaluation of specificity and sensitivity of this test in a group of nonstroke patients. Unfortunately, we are not able to obtain these data in our 381 patients with APV because our study was not planned for this kind of purpose, and we consider this limitation as a minor weakness of the article. It is well established that HIT could be negative up to 18.5% of patients during the acute phase of APV.\(^{29}\) For all of these reasons, a negative HIT alone should not be considered the single predictor of central lesions. Nevertheless, we gave more importance to some characterizing features of the clinical course of the disease: the intensity of imbalance and the duration of the acute symptomology.

Although some authors consider the severity of imbalance a less reliable finding,\(^{11}\) usually a patient with a cerebellar infarct cannot maintain an upright position without help, even with the eyes open; on the contrary, patients with vestibular neuritis can usually stand without support. Most of our patients fell down on standing, showing a moderate or severe imbalance that is out of proportion to vertigo. In patient 2, the indication to perform an MRI of the brain was due to not only a normal HIT but also the observation of the persistence of a severe imbalance that did not improve despite the disappearance of spontaneous nystagmus (Figure 1). Furthermore, patient 11, referred to our clinic after 23 days after acute onset of vertigo, showed a persistence of moderate/severe imbalance, despite the complete disappearance of spontaneous nystagmus, which led to suspicion of a central lesion; this suspicion was enhanced by the detection, after a more detailed history, of a sensation of dysarthria during the acute phase of the disease. The MRI confirmed the presence of a cerebellar lacune (Figure 2),

![Figure 1. Magnetic resonance imaging scan showing a triangle-shaped ischemic area involving the left posterior-inferior (patient 2: female, 68 years old).](image1)

![Figure 2. Magnetic resonance imaging scan showing a lacunar infarction of the left cerebellar deep hemispheric white matter (patient 11: male, 56 years old).](image2)
and further investigations showed an unrecognized patent foramen ovale.

In our series, 8 patients showed a long duration of vertigo and dizziness for more than 3 days with poor or no improvement of acute symptomatology. The long duration of vertigo with no improvement of the symptomatology in patient 7 was considered a recommendation to perform a radiological investigation. This patient had suffered from Ménière’s disease and had been successfully treated with intratympanic gentamicin. The patient developed an acute attack of vertigo associated with an increase of tinnitus and a worsening of a left sensorineural hearing loss. The vertigo showed no improvement after 24 hours, and the patient was consequently admitted to our hospital, where the bedside examination showed a III\(^{\circ}\) left spontaneous unidirectional nystagmus associated with a not clearly negative HIT, probably due to the interference of the previous vestibular lesion. The CT showed a stroke extensively involving the left cerebellar hemisphere. On the same day as the CT (3 days after the acute presentation), the patient developed a marked limb ataxia (Figure 3).

In our opinion, in accordance with previous reports,\(^\text{13,24,32}\) the persistence of acute vertigo with poor or no improvement after 2 to 3 days from the onset represents a very important clinical indicator that suggests performing an MRI even in the absence of cerebellar signs.

Indeed, the sensitivity of the latter clinical feature (7 of 11 patients had long-lasting vertigo, more than 72 hours) seems to be less than a negativity of HIT (9 of 11 patients), while the presence of a moderate or severe imbalance, observed in all of the patients, seems to be the best predictor of pseudo-APV.

A brain MRI should also be considered in the case of an acute audiovestibular loss; when this occurs in association with other neurological symptoms or signs, an ischemic stroke in the territory of the anterior inferior cerebellar artery (AICA) should be suspected.\(^\text{33,34}\) In our series, only 2 of 11 patients showed hearing loss associated with acute vertigo and spontaneous nystagmus. One of these patients (patient 3) resembled the above-mentioned characteristics of AICA stroke preceded by audiovestibular loss.

This study has several limitations. First, we described a nonconsecutive, retrospective, and nonsystematic collection of cases diagnosed primarily as peripheral disease and subsequently correctly understood as cerebellar infarcts. Second, the investigators who analyzed the clinical records were not blind to the study design, being in most of the cases the same clinicians who performed the clinical and instrumental examinations. Finally, the clinical and instrumental examination was not homogeneous: some oculomotor signs (eg, skew deviation) were performed only in the more recent cases, and this is why they are not reported in our study. For all of these reasons, we are not in a position to assess the real prevalence of a cerebellar stroke in a population affected by APV.

**Conclusion**

Even if the percentage of cases with cerebellar infarction simulating APV is not clearly defined (about 3% in our experience), a misdiagnosis may be very common. An underestimation of this value could be very conceivable because it can be argued that a very small cerebellar stroke in mPICA territory closely mimicking a peripheral vestibular syndrome could present a good prognosis remaining undiagnosed.

We believe that our study could be useful to focus the attention of frontline physicians, neurologists, and otolaryngologists on keeping in mind the differential diagnosis of a posterior circulation stroke in patients with acute spontaneous peripheral-type audiovestibular symptoms, particularly in subjects presenting vascular risk factors. To avoid a missed diagnosis of cerebellar infarct, all physicians who come into contact with this category of patients should have some familiarity with ocular testing and the clinical features of APV, the correct interpretation of which could avoid an excessive number of unnecessary brain MRIs. Although the small number of cases does not allow us to reach definitive conclusions, it is important to underline that moderate or severe imbalance was evident in all of the pseudo-APV patients, indicating that this aspect is perhaps the most important parameter for detecting a cerebellar involvement.

In the past 10 years, the execution of CTs and MRIs has increased more than 1.5-fold without a corresponding growth of central lesion diagnoses;\(^\text{12}\) optimization of the resources is now fundamental to reduce health care costs, especially in a time of economic crisis. Our report aims to...
identify clinical features and bedside oculomotor signs that may be useful for better assessing peripheral or central origins of the disease. In our report, not all of the patients were studied with a complete oculomotor test battery, but we think that a careful evaluation of spontaneous nystagmus and HIT along with watchful monitoring of the symptomatology, with particular attention to the severity of imbalance and the duration of acute vertigo, could be distinctive hallmarks that may indicate a central lesion.

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Author Contributions
Augusto P. Casani, study concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript, and study supervision; Iacopo Dallan, study design, manuscript drafting and revision, data collection; Niccolò Cerchial, acquisition of data, drafting of the manuscript, administrative support, and technical and material support; Riccardo Lenzi, acquisition of data, manuscript drafting, and revision; Mirco Cosottini, study concept and design, critical revision of the manuscript, and technical support; Stefano Sellari-Franceschini, analysis and interpretation of data and study supervision.

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