

Misdiagnosis of Acute Peripheral Vestibulopathy in Central Nervous Ischemic Infarction

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Introduction: Vertigo is a very common symptom at otorhinolaryngology (ENT), neurological, and emergency units, but often, it is difficult to distinguish between vertigo of peripheral and central origin.

Patients and Methods: We conducted a retrospective analysis of a hospital database, including all patients admitted to the ENT University Hospital Graz after neurological examination, with a diagnosis of peripheral vestibular vertigo and subsequent diagnosis of central nervous infarction as the actual cause for the vertigo. Twelve patients were included in this study.

Results: All patients with acute spinning vertigo after a thorough neurological examination and with uneventful computed tomographic scans were referred to our ENT department. Nine

of them presented with horizontal nystagmus. Only 1 woman experienced additional hearing loss. The mean diagnostic delay to the definite diagnosis of a central infarction through magnetic resonance imaging was 4 days (SD, 2.3 d).

Conclusion: A careful otologic and neurological examination, including the head impulse test and caloric testing, is mandatory. Because ischemic events cannot be diagnosed in computed tomographic scans at an early stage, we strongly recommend to perform cranial magnetic resonance imaging within 48 hours from admission if vertigo has not improved under conservative treatment. **Key Words:** Central infarction—Cranial imaging—Nystagmus—Vertigo.

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Vertigo is a very common symptom at otorhinolaryngology (ENT), neurological, and emergency units, but often, it is difficult to distinguish between vertigo of peripheral and central origin. There are certain additional symptoms, such as headache, nystagmus, cranial nerve palsy, focal neurological deficits, or vegetative symptoms, which could lead to the right diagnosis. Nevertheless, in a cerebellar or brainstem ischemia, vertigo can be the only symptom. In some cases, the primary cranial imaging (mostly computed tomographic scans) does not show any abnormalities, and patients with cerebral ischemia with a diagnosis of vestibular neuritis are referred to the ENT department. We reviewed all patients with central ischemia initially presenting with vertigo as their solitary symptom who were referred to our department with a diagnosis of vestibular neuritis within the last 7 years.

We wanted to emphasize on the importance of a careful neurological investigation in patients who present with acute-onset vertigo and the necessity of repeating

cranial imaging if vertigo does not improve under standard therapy.

PATIENTS AND METHODS

We conducted a retrospective analysis of the hospital database including all patients admitted to the ENT University Hospital Graz (tertiary referral center) between 2004 and 2011 who had a diagnosis of vertigo and subsequent central infarction, identified with the following codes from the *International Classification of Diseases, 10th Revision*: R42, H81.3, H81.4, H81.8, I65.0, M53.0, I67.8 in combination with I63.0, I63.1, I63.2, I63.3, I63.4, I63.5, I63.6, I63.8, I63.9 in 1 patient. Before admission to the ENT department, all patients had undergone neurological examination, and computed tomography (CT) of the skull had been performed at the neurological emergency unit. The neurological examination included cranial nerve function, sensory and motor function, tendon and Babinsky reflexes, Laseque sign, and coordination and gait testing (Romberg test, finger to nose, heel to shin, rapid alternating movements, and, if possible, walking with open and closed eyes). Conditions of all patients were preliminarily diagnosed by a neurologist as peripheral vestibular vertigo with subsequent diagnosis of a central nervous infarction as the actual cause for the vertigo.

Twelve patients met the criteria, and the following parameters were analyzed from patients' charts: age, sex, presenting

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symptoms, neurological status, primary and secondary imaging, vestibulometry, and delay to definite diagnosis.

Microsoft Excel 2007 (Microsoft, Seattle, WA, USA) and SPSS v18 (SPSS, Inc., Chicago, IL, USA) were applied for statistical analysis; values are presented as means, SD, medians, and range. The study was approved by the institutional review board of the Medical University of Graz.

RESULTS

Twelve patients (7 men and 5 women) were included in the study. The mean age at presentation was 59 years (median, 65.5 yr; range, 25–76 yr; SD, 17.69 yr). There was no statistically significant difference in age between women and men ($p = 0.123$, Mann-Whitney U test). All patients complained about rotatory vertigo with nausea and vomiting. Two patients had additional symptoms: one had bilateral tinnitus and the other experienced right-sided hearing loss, simultaneously to vertigo onset. All patients were sent to the ENT department with a diagnosis of peripheral vestibular vertigo after previous neurological examination at the neurological department including complete neurological status and CT of the skull. Neurological status as well as radiological reports of the CT did not show any signs of central nervous infarction. Vestibulometry, including video-oculography and electronystagmography, revealed horizontal nystagmus with opened and closed eyes in 9 patients. No other pathologic disease could be found in vestibulometry. Three patients did not show any nystagmus (Table 1).

We administered pentoxifyllin and betahistin dihydrochloride in all cases until a definite diagnosis of central

infarction was obtained. In addition, low-dose cortisol was given to the patient with vertigo and hearing loss. Within the hospital stay (mean, 4.8 d; range, 1–9 d; SD, 2.55 d), 5 of the patients did not show any symptom improvement, 4 patients developed ataxic gait, 2 patients showed left-sided paresthesia, and 1 patient developed double vision. Consequently, magnetic resonance imaging (MRI) was performed with a mean diagnostic delay of 4 days (range, 0–7 d; SD, 2.3 d). MRI revealed cerebellar infarction in 9 patients (4 left hemisphere, 4 right hemisphere, 1 vermis cerebelli); of these, 3 patients had an additional infarction of the medulla oblongata. Two patients had a solitary infarction of the medulla oblongata, one caused by a dissection of the vertebral artery. One patient had a thrombosis of the sigmoid and transverse sinus (Table 1). Mastoiditis and other infectious causes were excluded by laboratory (C-reactive protein level and white blood cell count) and clinical examination as well as reevaluation of cranial imaging.

In Patient 6 (Table 1), the correct diagnosis was found within 9 hours after referral to our clinic because he developed paresthesia of the left side, and consequently, an MRI was performed on the same day.

On diagnosis, all patients were referred to the stroke unit of the neurology department.

CONCLUSION

Acute onset of vertigo is a very common entity seen in emergency departments. To exclude a life-threatening cause of this symptom, it is mandatory to consider central affections. In most cases, acute vertigo is due to peripheral vestibulopathy, but a central ischemic event always has to be taken into account.

Gauss et al. (1) gave a very good review of clinical symptoms that can lead to the right diagnosis. Acute spinning vertigo with vegetative symptoms and hearing loss or tinnitus is typical for a peripheral vestibular disease, whereas vertigo without nausea is more likely to be caused by a central affection. Also, impaired cranial nerve function and other focal signs, such as Horner syndrome, diplopia, ataxia, or dysesthesia, are typical for central vertigo. Deviation nystagmus or dissociated or vertical nystagmus is usually of central origin. This is why the authors insist on a complete and careful neurological investigation, even when cranial imaging is uneventful.

According to Seemungal (2), acute, isolated vertigo has 3 major causes, namely, vestibular neuritis (the most common), followed by cerebellar stroke and migrainous vertigo. In combination with hearing loss and tinnitus, Ménière's disease is the major cause of vertigo. He also points out 3 important tests for differential diagnosis between central and peripheral vertigo: First is the head impulse test, which is usually intact in ischemic cerebellar strokes. The second is the bedside iced water caloric testing. In a peripheral lesion, injection of iced water on the side with lesion causes no nystagmus or has no effects on any spontaneous nystagmus. Third is that the ability to

TABLE 1. *Epidemiology, nystagmus, diagnostic delay, and MRI findings*

ID	Sex	Age (yr)	HS	Nystagmus	Delay	Localization of infarction according to MRI
1	M	71	6	SN I left	1	Right CH
2	F	67	7	SN I left	5	Left CH, left dorsal MO, left FL, right frontotemporoparietal lobe
3	M	76	5	SN I right	7	Right lateral MO
4	M	69	9	SN I right	5	Right CH
5	M	63	2	SN III right	1	Left CH, left dorsal MO
6	M	64	1	None	0	Left CH, left lateral MO, anterior pons
7	F	46	5	SN II right	5	Right CH
8	M	57	2	None	4	Left CH
9	F	25	7	None	7	Thrombosis sigmoid and transverse sinus
10	F	27	2	SN III right	4	Left lateral medulla oblongata (dissection A. vertebralis)
11	F	73	7	SN III left	6	Right CH
12	M	75	5	SN III left	4	Vermis cerebelli

HS indicates hospital stay at the ENT-Department in days before transfer to the stroke unit; SN, spontaneous horizontal nystagmus, classification: e.g., right: I: nystagmus only beating at right gaze, II: beating at right and straight forward gaze, III: nystagmus present even in left gaze; CH, cerebellar hemisphere; MO, medulla oblongata; FL, frontal lobe.

suppress spontaneous nystagmus in the light suggests intact central mechanisms. Newman-Toker et al. (3) also described the head impulse test as a useful tool for differentiating acute cerebellar strokes from vestibular neuritis. In cerebellar strokes, its result is usually negative (91%), whereas all patients with vestibular neuritis showed a positive head impulse test result.

In a study consisting of patients with acute vestibular syndrome, Thabet (4) found 9 of 30 patients experiencing a central disease, of which 4 turned out to be an ischemic event.

Few authors have described patients with central infarctions and isolated vertigo who were misdiagnosed as having peripheral vertigo due to the lack of other additional symptoms. In some cases, the infarction was localized in the cerebellum (5–9), whereas others showed ischemia of the brainstem (10–13). Kim et al. (14) described the case of a patient with signs of an acute peripheral vestibular syndrome (sudden onset of vertigo, nausea, and vomiting and spontaneous right-beating horizontal nystagmus) that turned out to be an infarction of the hippocampus and basal ganglia, whereas Brandt et al. (15) described almost the same symptoms in a patient with ischemic lesion of the posterior insula.

In our study, all patients presented with spinning vertigo, nausea, and vomiting. Neurological status as well as computed tomographic scans did not show any signs of central nervous infarction. Nine patients showed a horizontal spontaneous nystagmus. MRI revealed cerebellar infarction in 9 patients, of whom 3 had an additional infarction of the medulla oblongata. Two patients had a solitary infarction of the medulla oblongata, and 1 patient had a thrombosis of the sigmoid and transverse sinus (Table 1).

Urban et al. (5) put emphasis on cerebellar nodulus infarction by describing 2 cases of positioning vertigo because of nodulus infarction, which is unusual in events in the central nervous system.

In our study, we could not experience a correlation between the side of the lesion (medulla oblongata and cerebellar hemisphere) and the beating of the nystagmus, although Kim et al. (16) found the nystagmus almost always ipsilesional in the medullary infarction.

As far as ischemia of the anterior inferior cerebellar artery is concerned, vertigo, ipsilesional nystagmus, and hearing loss are the predominant symptoms, and their clinical signs are similar to those of peripheral vestibulopathy. These symptoms can be a prodrome even weeks before an actual ischemic stroke in the cerebellum (1,17–22).

It is very unusual that, in our patients, only 1 woman experienced a sensorineural hearing loss. The other patients (11/12) did not complain about hearing loss, and all audiograms showed symmetric hearing curves.

Eighty-two patients with anterior inferior cerebellar artery territory infarcts were investigated in a study by Lee et al. (23). In 98% of patients, acute spontaneous prolonged vertigo was the main symptom. They also showed that a selective loss of vestibular function is very rare (5%). Sixty percent of the patients had a combined loss of auditory and vestibular function.

Mean age at presentation was 59 years, although 2 women were relatively young (25 and 27 yr). In young and middle-aged patients, spontaneous cervical artery dissection is the major cause of cerebral infarction (24), which was diagnosed in 1 woman (dissection of the left cervical artery) (10), although the second young woman experienced a thrombosis of the sigmoid and transverse sinus. In literature, several cases of vertebral artery dissection presenting with vertigo as the predominant symptoms have been described lately (6,7,10,25).

As far as cranial imaging is concerned, Gauss et al. (1) suggested that, in immediate situations, CT is the method of choice to exclude intracranial hemorrhage, but if symptoms are persisting after 24 hours, an additional MRI should be performed. Nevertheless, MRI is better than CT for the detection of acute ischemia and it can also detect acute and chronic hemorrhage. Therefore, MRI is the imaging of choice if the slightest suspicion of an acute stroke exists (26). Bonkowsky et al. (6) recommend MRI if vertigo does not improve after 48 hours.

Most authors do not report about the diagnostic delay from the onset of the first symptoms to the definite diagnosis through MRI. In our study, the mean diagnostic delay was 4 days, and in 1 patient, it took even 7 days to obtain a definite diagnosis. In our opinion, this is far too long, and therefore, we recommend to perform an MRI if vertigo does not improve within 48 hours of therapy, even if there are no additional neurological symptoms.

Our study clearly demonstrates that it can be difficult to distinguish between a peripheral or central origin of vertigo. Therefore, a careful otologic and neurological examination, including the head impulse test and caloric testing, is mandatory.

Despite the absence of typical focal neurological signs, spinning vertigo with vegetative symptoms can be caused by an ischemic event in the medulla oblongata or cerebellum. This is why we strongly recommend to perform a cranial MRI if vertigo has not improved after 48 hours of conservative treatment. This seems to be the right way to decrease the diagnostic delay of a potential life-threatening central ischemic event.

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