Background: Tumarkin falls are sudden drop-attack falls that occur in a subset of patients with Meniere syndrome (endolymphatic hydrops), an inner ear disorder characterized by vertigo spells and hearing loss.

Objective: To describe the clinical features and quantitative audiovestibular testing results in a case series of patients with Tumarkin falls, episodic vertigo, and normal hearing.

Setting: University referral center for disorders of balance and hearing.

Methods: Case series (unselected) of all patients with Tumarkin falls and a normal audiogram at least 1 year after onset of vestibular symptoms (n=6) from a retrospective analysis of the records of all patients with Tumarkin falls presenting to Neurotology Clinic at UCLA Medical Center, Los Angeles, Calif, from October 1, 1975, to February 1, 2001 (N=55). Quantitative audiologic and vestibular function testing, neurologic history, and examination were performed.

Results: Five of 6 patients had unilateral caloric paresis, and 1 had bilateral vestibulopathy. Five of 6 had a personal and/or family history of migraine headaches meeting International Headache Society criteria. All patients had a subjective sensation of feeling pushed by an external force, and half of the patients had a subjective tilt of the environment concurrent with the fall.

Conclusions: The incidence of migraine is high in this subgroup of patients with Tumarkin falls and normal hearing. The clinical description of the falls is similar to those associated with Meniere syndrome. Further studies are needed to understand the etiology of Tumarkin falls in these patients with normal hearing.

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SUDDEN DROP-ATTACK falls without loss of consciousness have been noted in a small percentage of patients with Meniere disease (endolymphatic hydrops).1-4 Meniere disease is an inner ear disorder characterized by fluctuating hearing loss, tinnitus, and episodic vertigo. Tumarkin1 described patients with Meniere disease who have sudden falls that occur without warning, without loss of consciousness, and without any concomitant neurologic symptoms or sequelae. The patients typically report the sensation of being pushed, and they fall in the same direction with repeated falls.3,4 In some cases, patients report a sudden tilt of the environment simultaneous with the fall.3,4 Because surgical ablation of the vestibular periphery cures Tumarkin falls, these falls must originate from the vestibular periphery.2,4

The clinical features of Tumarkin falls are consistent with mechanical stimulation to the otoliths, causing a burst of neural impulses from the otolithic organs to the vestibulospinal pathways, triggering the fall. A well-documented case of a patient with an otolithic Tullio phenomenon provides clues to the mechanism of otolithic involvement in Tumarkin falls.5 In this patient, a loud sound to the affected ear triggered a fall to the right and backward, a counterclockwise tilt of the visual scene, and a documented ocular tilt reaction, with characteristics consistent with ipsilateral otolithic stimulation. A short-latency vestibulospinal reflex was recorded in the tibialis anterior and gastrocnemius muscles that may mediate the postural imbalance.5 There are similarities between the postural imbalance in this case report of otolithic Tullio phenomenon and Tumarkin falls in Meniere disease. In both cases, the direction of the fall is stereotyped, and the fall is often associated with a room tilt illusion.3,4 However, in Tumarkin falls, there are no precipitating factors and no aura, and the falls are more abrupt and violent.

There are no previous reports, to our knowledge, of Tumarkin falls in associa-
tion with vestibulopathies other than Meniere syndrome and delayed endolymphatic hydrops, a variant of Meniere syndrome. We present 6 case reports of patients with sudden falls with characteristics of Tumarkin falls, recurrent spontaneous vertigo spells, and normal hearing. These patients clearly do not meet the diagnostic criteria for Meniere syndrome as defined in 1995 by the American Academy of Otolaryngology–Head and Neck Foundation.6

METHODS

From October 1, 1975, to February 1, 2001, 55 patients were seen at the Neurotology Clinic at UCLA Medical Center, Los Angeles, Calif, who met the clinical criteria for Tumarkin falls. The criteria for inclusion as Tumarkin falls were the report of a sudden fall that occurred without warning and without loss of consciousness, with no associated neurologic symptoms, normal results of a neurologic examination, and the clinical assessment of one of us (R.W.B.). Each patient had completed a standard dizziness questionnaire and underwent neurotologic evaluation by one of us (R.W.B.). All patients were interviewed regarding migraine symptoms by means of standard International Headache Society (IHS) criteria.7 Patients had undergone quantitative electronystagmographic testing with bithermal caloric testing and audioligic testing. All 55 medical charts of these patients were analyzed retrospectively, and of these, 47 patients had otologic diagnosis with vestibulopathies other than Meniere syndrome and delayed endolymphatic hydrops, a variant of Meniere syndrome. We present 6 case reports of patients with sudden falls with characteristics of Tumarkin falls, recurrent spontaneous vertigo spells, and normal hearing. These patients clearly do not meet the diagnostic criteria for Meniere syndrome as defined in 1995 by the American Academy of Otolaryngology–Head and Neck Foundation.6

RESULTS

There were 6 patients: 4 women and 2 men (Table 1). All patients had a history of episodic spontaneous vertigo, recurrent Tumarkin falls, and no otologic symptoms of tinnitus, aural fullness, or fluctuations in hearing. The average age at onset of vertigo spells was 40.6 years. The interval between the onset of vertigo spells and that of the Tumarkin falls ranged from at onset to 12 years, with an average of 4.5 years. Three of 6 patients had Tumarkin falls as the initial presentation of vestibular problems. All patients reported the subjective sensation of being pushed by an external force. Three of 6 reported a sudden tilt of the environment concurrent with the fall. In patients 1 and 5, the room tilt was 90° toward the ipsilateral damaged ear, and in case 6 the room tilt was 90° vertically. No patients had auditory symptoms of tinnitus, ear fullness, or fluctuating hearing temporally related to the vertigo or falls, or in isolation.

TABLE 1

<table>
<thead>
<tr>
<th>Case</th>
<th>AGE</th>
<th>SEX</th>
<th>FAMILY HISTORY</th>
<th>MIGRAINE HISTORY</th>
<th>VESTIBULAR HISTORY</th>
<th>WORKUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40</td>
<td>M</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>F</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>M</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Normal</td>
</tr>
<tr>
<td>4</td>
<td>60</td>
<td>M</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>5</td>
<td>30</td>
<td>F</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Normal</td>
</tr>
<tr>
<td>6</td>
<td>37</td>
<td>M</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Normal</td>
</tr>
</tbody>
</table>

CLINICAL HISTORY

In five (83.3%) of 6 patients, there was a personal and/or family history of migraine meeting IHS criteria (Table 2). Three of 6 met the IHS criteria for migraine headaches, and 1 met IHS criteria for acephalgic migraine with visual aura. Four of 6 patients had a family history of migraine headaches. In the patient without a personal or family history of migraine (patient 1), there was a history of headaches concurrent with vertigo, but the head-

MIGRAINE HISTORY

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QUANTITATIVE AUDIOVESTIBULAR TESTING RESULTS

All patients had an audiogram at least 2.5 years after onset of vertigo spells. The range in interval between onset of vertigo spells and audiologic testing was 2½ to 14 years (Table 1). In all cases, serial audiograms disclosed normal hearing. In patients 1 through 5, quantitative vestibular testing (electronystagmography) showed a unilateral caloric paresis. Patient 6 had a profound bilateral peripheral vestibulopathy, manifested by an absent response at 0.05 Hz and a symmetrically decreased gain and increased phase lead at 0.2, 0.4, 0.8, and 1.25 Hz on sinusoidal rotational testing.

MIGRAINE HISTORY

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ache quality did not meet IHS criteria for migraine. The family history in his case was unknown. Five of 6 patients did not have a temporal association of vertigo spells or Tumarkin falls with headaches or aura. Four of the 6 (patients 2, 4, 5, and 6) had vertigo spells, as well as migraine headaches, triggered by specific precipitants, eg, sleep irregularities, hormonal changes, or specific foods.

All 6 of our patients with Tumarkin falls and normal hearing had a history of episodic spells of vertigo ranging from several seconds to several days. The small number of patients may reflect a true low incidence or the previous lack of recognition of the association of Tumarkin falls with migraine. Although selection bias can be an inherent confounder of any retrospective study, we tried to minimize that risk by the inclusion of all patients diagnosed as having Tumarkin falls and the unselected inclusion of all of these patients who had normal hearing.

The interval between vestibular symptoms and presentation to the Neurotology Clinic at UCLA Medical Center ranged from 2½ to 14 years. By definition for inclusion in this study, during that period there were no otologic symptoms of fluctuating hearing, tinnitus, or aural fullness, and there was documentation of normal hearing on serial audiograms. The diagnostic criteria for definite Meniere disease, as proposed by the American Academy of Otolaryngology–Head and Neck Foundation, include (1) 2 or more episodes of vertigo of at least 20 minutes to 24 hours in duration; (2) audiometrically documented hearing loss; (3) tinnitus or aural fullness; and (4) exclusion of other causes. Most patients who go on to develop Meniere disease will have audiologic symptoms of tinnitus, aural fullness, or fluctuating hearing within 1 year of onset of symptoms. The 6 patients described in this study, therefore, do not meet the diagnosis of Meniere disease, and they are unlikely to later develop Meniere disease.

The clinical description of the falls in these patients is identical to that described by patients with Meniere syndrome–associated Tumarkin falls. In a study of 12 patients with Meniere syndrome presenting with Tumarkin falls, Baloh et al noted that all patients had a subjective sensation of being pushed, and 3 of the 12 had a room tilt illusion. In a study of 7 patients with Meniere syndrome presenting with Tumarkin falls at age greater than 40 years, Baloh et al noted that all patients had a subjective sensation of being pushed, and 3 of the 12 had a room tilt illusion. In a study of 7 patients with Meniere syndrome presenting with Tumarkin falls at age greater than 40 years, Baloh et al noted that all patients had a subjective sensation of being pushed, and 3 of the 12 had a room tilt illusion.

Table 1. Clinical Features and Audiovestibular Test Results

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Rate of Falls</th>
<th>Description of Falls</th>
<th>Interval Between Vertigo Spells and Falls</th>
<th>Description of Vertigo</th>
<th>ENG</th>
<th>Interval: Onset of Vertigo and Audiometry, y</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/55 5 in 14 y</td>
<td>Push to L, room tilt 90° to R</td>
<td>Initial</td>
<td>Vertigo spells lasting few minutes, no triggers</td>
<td>33% R VP</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>2/F/46 &gt;15 in 3 y</td>
<td>Push to R</td>
<td>Initial</td>
<td>Vertigo, nausea lasting days; triggers: fatigue, stress</td>
<td>35% L VP</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>3/F/43 6 in 3 y</td>
<td>Push to L, knee injury</td>
<td>Initial</td>
<td>Intermittent dysEquilibrium lasting minutes, no triggers</td>
<td>43% R VP</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>4/F/53 2 in 1 y</td>
<td>Push to R</td>
<td>10 y</td>
<td>Vertigo, nausea lasting 1-5 min; triggers: menses</td>
<td>59% L VP</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>5/F/55 25 in 4 mo, several 3 y ago</td>
<td>Push to R, room tilt 90° to L</td>
<td>5 y</td>
<td>Vertigo, nausea, vomiting lasting from minutes to days; triggers: fatigue, stress</td>
<td>100% L VP</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>6/M/37 Several in past month</td>
<td>Falls back, room tilt 90° vertical</td>
<td>12 y</td>
<td>Vertigo, nausea lasting few seconds to 30 min; triggers: miss meal, lack of sleep</td>
<td>Bilateral peripheral vestibular dysfunction†</td>
<td>12</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: Audio, average pure-tone audiometry; ENG, electroneystagmogram; L, left; R, right; VP, caloric vestibular paresis.

Table 2. Summary of Personal and Family History of Migraine

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age at Onset of HAs</th>
<th>HA History</th>
<th>Meets IHS Criteria for Migraine</th>
<th>Family History</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40s</td>
<td>HA pressure with vertigo</td>
<td>No</td>
<td>Unknown</td>
</tr>
<tr>
<td>2</td>
<td>Teens</td>
<td>Occasional HAs, history of severe motion sickness, visual stimuli trigger HA</td>
<td>No</td>
<td>Father and sister: migraine</td>
</tr>
<tr>
<td>3</td>
<td>Teens</td>
<td>Bifrontal, nausea, sleeps off HA, awakens her</td>
<td>Yes</td>
<td>Father, 19-y-old daughter: migraine</td>
</tr>
<tr>
<td>4</td>
<td>Teens</td>
<td>Severe, pulsating, sick HAs in teens, occasionally as adult</td>
<td>Yes</td>
<td>Mother, sister, 20-y-old daughter: migraine</td>
</tr>
<tr>
<td>5</td>
<td>30s*</td>
<td>Migraine visual aura lasting 15 min few times per year, no HA</td>
<td>Yes (acephalgic migraine)</td>
<td>Mother: migraine</td>
</tr>
<tr>
<td>6</td>
<td>Teens</td>
<td>Sick HA, with photophobia and phonophobia in teens, now rare</td>
<td>Yes</td>
<td>Mother: vertigo spells</td>
</tr>
</tbody>
</table>

Abbreviations: HA, headache; IHS, International Headache Society.

*Onset of visual aura.
than 65 years, Ishiyama et al12 reported a similar clinical presentation of Tumarkin falls. The factor that distinguishes the patients in the present study is the absence of hearing abnormality; in all previous studies, patients with Tumarkin falls had audiometrically documented hearing loss.

In this study, 3 of 6 patients reported a subjective room tilt in association with the fall. The direction of the room tilt was stereotyped for each individual patient and was in the direction opposite to the direction of the fall. The subjective room tilt has been associated with an ocular tilt reaction that can occur with otolithic stimulation2 and has been noted in some patients with Meniere syndrome-associated Tumarkin falls.3,4 There was not a temporal relationship between the vertigo spells and the Tumarkin falls; however, patients 1, 3, and 4 reported that the Tumarkin falls occurred in the setting of a recent flurry of episodic vertigo spells, both indicative of ongoing peripheral vestibular abnormality.

In these cases of Tumarkin falls without associated auditory symptoms or signs, there was an association with migraine in 5 of 6 cases (Table 2). An association between Meniere disease and migraine had been proposed by Mene`re in 1861.9 The prevalence of migraine in patients with Meniere disease is about twice as high as that in the general population.10 The prevalence of migraine in the present case series was almost 3-fold higher than the highest estimates of the prevalence of migraine (5.3%-19% in males; 11%-28% in females).11 However, since migraine is so common, the combination of migraine, vestibular loss, and Tumarkin falls in these patients may have been a chance occurrence. In two thirds of our patients, the episodes of vertigo had features in common with migraine, including precipitation by alcohol, lack of sleep, emotional stress, and hormonal changes of menses. These triggers have been noted in Meniere disease as well. Thus, it is possible that the disorder in these patients represents an atypical variant of Meniere disease. Electrocochleography will be useful as an adjunctive measure to look for evidence of endolymphatic hydrops despite normal hearing.

Neurotologic symptoms are common with migraine; episodic vertigo occurs in about one fourth of unselected patients with migraine.12 However, the definition of migraine-equivalent or migraine-associated vertigo remains unclear when there is not a clear temporal association between the headache or migraine aura and the vestibular symptoms, and most often the vertigo attacks occur during the headache-free interval.13

The association of audiovestibular disorders with migraine extends back to the 19th century.14 Migraine has been reported to be associated with benign paroxysmal vertigo of childhood15 and benign recurrent vertigo in adults, ie, episodic vertigo without hearing abnormality.12,13,16-17 Rassekh and Harker11 reported that 81% of patients with “vestibular Meniere’s,” recurrent attacks of vertigo without associated auditory symptoms, had migraine.

There is evidence that migraine is associated with peripheral vestibular end-organ damage. Cutrer and Baloh13 reported that 20.9% of patients meeting the criteria for migraine-associated dizziness had unilateral caloric paresis. Cass et al18 reported that 18% of patients with migraine-associated dizziness had unilateral caloric paresis. Ishiyama et al13 presented an association between benign paroxysmal positional vertigo (BPPV) and migraine as further evidence of peripheral vestibular damage associated with migraine. Patients with idiopathic BPPV were 3 times more likely to have migraine than patients with BPPV secondary to a known cause, and almost half of the patients with the onset of BPPV before 50 years of age met the diagnostic criteria for migraine.19 Childhood BPPV has also been linked to migraine.20 Benign paroxysmal positional vertigo is a well-documented sequela to ischemic damage of the inner ear, presumable due to release of otocoria from the macular membrane.21 Migraine has been proposed to cause sudden sensorineural hearing loss22,23 and monocular blindness,24 possibly via vasospasm of the small arteries of the ear and eye. Thus, it may be possible that migraine causes damage to the peripheral vestibular system via vasospasm of the small arteries of the inner ear.

The occurrence of Tumarkin falls in this subgroup of patients with vestibulopathy and vertigo hints that migraine-associated vertigo may be a phenomenon of the peripheral vestibular system in at least this subset of patients; however, further investigation in a larger study is required. In the population of patients presented in this study, all patients with Tumarkin falls without hearing abnormality had objective evidence and symptoms of peripheral vestibulopathy, ie, recurrent spontaneous vertigo spells and caloric paresis. In contrast, Dieterich and Brandt22 reported a low incidence of caloric paresis (8%) and a high incidence of electronystagmographic findings indicative of central abnormalities, in a retrospective study of 90 patients with migraine-associated vertigo. Thus, migraine-associated vertigo probably has heterogeneous causes. In the present study, given that the results of neurologic examination were normal and there was no evidence of central abnormalities on the electronystagmographic recording, a central cause for the sudden falls is unlikely. If the mechanism of the Tumarkin falls in our patients is the same as that of Tumarkin falls in Meniere syndrome, these falls originate from the vestibular periphery. Although we must use caution in establishing an etiologic link, we propose that the mechanism of migraine-associated vertigo and Tumarkin falls in these patients is a peripheral vestibular phenomenon. Calcium channel blockers, β-blockers, selective serotonin reuptake inhibitors, and acetazolamide have been used for the prevention of migraine-associated vertigo spells.25 Whether these agents are efficacious for the prevention of Tumarkin falls in these patients is unknown. We propose that for intractable Tumarkin falls with evidence for asymmetric vestibulopathy on testing, selective vestibular ablative procedures should be considered, ie, vestibular nerve section with hearing preservation.

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Author contributions: Study concept and design (Dr G. Ishiyama); acquisition of data (Dr G. Ishiyama); analysis and interpretation of data (Dr G. Ishiyama, A. Ishiyama, and Baloh); drafting of the manuscript (Dr G. Ishiyama); critical revision of the manuscript for important
intellectual content (Drs G. Ishiyama, A. Ishiyama, and Baloh); statistical expertise (Drs G. Ishiyama and Baloh); obtained funding (Dr A. Ishiyama); study supervision (Drs A. Ishiyama and Baloh).

Corresponding author and reprints: Robert W. Baloh, MD, Department of Neurology, Box 951769, University of California, Los Angeles, UCLA School of Medicine, Los Angeles, CA 90095 (e-mail: rwbaloh@ucla.edu).

REFERENCES