swiss medical weekly

Original article | Published 20 February 2025 | doi:https://doi.org/10.57187/s.4147
Cite this as: Swiss Med Wkly. 2025;155:4147

Treating Menière's disease with rimegepant

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Summary

A recent hypothesis states that Menière's disease is caused by inappropriate expression, i.e. enhanced release of the neurotransmitter calcitonin gene-related peptide. Here, we tested this hypothesis by administering rimegepant, a new calcitonin gene-related peptide antagonist approved for the acute treatment of migraine and for the prevention of episodic migraine, to six patients with both Menière's disease and migraine. Two patients received the first dose of 75 mg rimegepant to treat an acute attack of Menière's disease. One of these two plus the remaining four patients were treated with 75 mg rimegepant every other day for secondary prevention. One patient developed an allergic reaction after the first administration and was excluded from further treatment. In the two patients treated during acute Menière's disease, symptoms were relieved and resolved about 30 min earlier than migraine symptoms. While all five patients had reduced migraine, all completely resolved Menière's symptoms on preventive therapy with rimegepant for up to eight months. These results support the idea that calcitonin gene-related peptide is linked to the pathogenesis of Menière's disease and suggest that inhibition of calcitonin gene-related peptide signalling may represent a promising therapeutic option for Menière's disease patients.

Introduction

Menière's disease is an inner-ear disorder characterised by attacks of vertigo lasting 20 minutes to 12 hours, accompanied by fluctuating low- and mid-frequency hearing loss, increased tinnitus and/or ear pressure. It has an estimated prevalence ranging from 3.5/100,000 adults in Japan [1] to 513/100,000 adults in southern Finland [2] or between 0.04% and 0.51%, respectively. A recent study in California [3] reported 190/100,000 or 0.19%. The prevalence of Menière's disease appears to be lower in Asian countries, although there are few epidemiological studies. Kim et al. reported that the prevalence of Menière's disease in Korea increased from 0.04% in 2013 to 0.15% in 2017 [4].

Different prevalences have also been reported for migraine. According to Burch et al. [5], the global prevalence of migraine is 15%, but with variations from 9% in the Western Pacific (China), 12% in the USA, 25–33% in Southeast Asia to 35% in the European Union and Nepal. In a very recent article on the prevalence of migraine in Asia, it was estimated to be 13.8% in Asian countries.

Menière's disease and migraine are cross-correlated, with a 10% prevalence of migraine in Korean patients with Menière's disease compared with only 3.5% in a matched control group [4]. The 3.5% migraine figure is much lower than the prevalences mentioned above. Thus, the risk of migraine would be 2.9 times higher in patients with Menière's disease than in subjects without Menière's disease. Ghavami et al. [6] reported that 51% of Menière's disease patients also suffer from migraine. Thus, it is generally accepted that there is a very high correlation between Menière's disease and migraine.

There also exists a vestibular migraine, first listed in the 3rd edition of the International Classification of Headache Disorders (ICHD-III) in 2018 [7]. As mentioned in the previous publication on calcitonin gene-related peptide as the cause of Menière's disease, according to Tabet and Saliba [8] "there are no known definitive diagnostic tests that can reliably distinguish the two conditions". Nevertheless, we describe the typical combination of clinical Menière's disease symptoms in patients with migraine as two diseases.

Although its first description dates back 164 years [9], the aetiology and pathophysiology of Menière's disease is still poorly understood and no evidence-based effective oral or intravenous treatment is available. Transtympanic or intratympanic injections of steroids were thought to be promising according to a 2011 Cochrane review [10] that included only one study, but the most recent Cochrane meta-analysis [11] of 10 included trials, all using dexamethasone, concluded that intratympanic corticosteroids may make little or no difference in the number of people reporting improvement in their vertigo at 6 to 12 months or more of follow-up.

Another therapy, mostly third-line, is endolymphatic sac surgery. However, there is considerable controversy about its efficacy and whether the sac should be decompressed, opened or shunted [12].

Only destructive procedures such as vestibular neurotomy, neurectomy, labyrinthectomy or transtympanic injections of aminoglycosides, most commonly gentamicin, have a significant effect on reducing the frequency of vertigo in Menière's disease [13]. However, these procedures carry a significant risk of hearing loss.

Since all or almost all patients with Menière's disease show endolymphatic hydrops [14], it is often assumed that

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Menière's disease is caused by endolymphatic hydrops. However, guinea pigs with endolymphatic hydrops, induced by resection of the endolymphatic sac, do not develop Menière attacks [15]. The only symptom which seemed to be closely correlated with endolymphatic hydrops was low-frequency hearing loss [16], but this correlation has also been questioned [17].

Various changes occur in the inner ears of Menière's disease patients, including signs of inflammation [18] and decreased blood supply [19], and various causes, such as viral [20] or autoimmune inflammation [18] or allergies [21, 22], have been suspected of causing Menière attacks. Hence, Menière's disease has recently been called Menière's syndrome, because it is suspected that its symptoms may be due to disparate causes and aetiologies. In 1992, Cutrer and Baloh first suspected a common involvement of neuropeptides like "substance P, neurokinin A and calcitonin gene-related peptide**"** in Menière's disease and migraine [23]. They also speculated that "calcitonin gene-related peptide and possibly other neuropeptides released from trigeminal afferents and vestibular efferents may increase excitability of the inner ear vestibular receptors".

In 2021, calcitonin gene-related peptide was suspected of being the main cause of Menière's disease [24], because it is one of the main transmitters in cochlear and vestibular efferents, thus explaining simultaneous cochlear and vestibular symptoms. Calcitonin gene-related peptide is a potent vasodilator and endolymphatic hydrops may be induced by dilation of capillaries in the stria vascularis, which may induce endolymphatic hydrops like oedema induced in other parts of the body. Calcitonin gene-related peptide is also known to induce neurogenic inflammation [25, 26], explaining the inflammatory signs described in the inner ears of Menière's disease patients. Later, Menière's disease as well as isolated cochlear or vestibular symptoms were suspected of being caused by inner ear migraine [27]. But Frank et al. did not focus on calcitonin gene-related peptide. Since we are still unable to distinguish Menière's disease from vestibular migraine as argued in the hypothesis that calcitonin gene-related peptide causes Menière's disease [24], which is supported by other authors [6, 27, 28], we also see Menière's disease as a special form of inner ear migraine. In their recent review, Baron and Steenerson [29] argue that "given the frequent overlap of these two conditions, and the difficulty in treating Menière's disease, a patient with Menière's disease who also has a history of migrainous headaches could reasonably be trialled with targeted calcitonin gene-related peptide therapy".

The above suggests that calcitonin gene-related peptide may be causally involved in Menière's disease and provides a rationale for treating Menière's disease with calcitonin gene-related peptide antagonists, which have been available since 2018. However, the first calcitonin gene-related peptide antagonists (erenumab, fremanezumab, galcanezumab, eptinezumab) are large monoclonal antibodies that cross the blood-brain barrier only by 0.1–0.3% owing to their molecular weight of 180–200 kDa [30]. Nevertheless, they work for the prevention of migraine because the ganglion of the trigeminal nerve is situated outside the blood-brain barrier [31]. Unfortunately, it has not yet been investigated whether they may cross the blood-labyrinth barrier. Interestingly, severe disturbance of the blood-labyrinth barrier in the cochlea has been reported in older patients with endolymphatic hydrops [32] and a breakdown of the blood-labyrinth barrier has recently been described in Menière's disease [33].

More recently, small molecule calcitonin gene-related peptide antagonists, so-called gepants, have become available. Despite their small size of only 0.5–0.6 kDa [29], these substances cross the blood-brain barrier to a limited extent of about 1–3%. The spinal fluid concentrations of telcegepant and olcegepant were only about 1.3% of plasma concentration [34], but Hostetler et al. determined the in vivo cerebrospinal fluid / plasma ratio to be between 2% and 3% and showed the highest level of binding in the cerebellum, brainstem and meninges [35]. The 1–3% of plasma level in the CSF measured for gepants is still small, but at least an order of magnitude higher than the 0.1–0.3% reported for galcanezumab [30]. The distribution volume in the inner ear is unknown. But even if the gepant molecules do not cross the normal blood-labyrinth barrier, they may still reach the inner ears in Menière's disease patients because of the breakdown of the blood-labyrinth barrier in Menière's disease [33]. Alternatively, they could also reach the inner ear through the cochlear and vestibular efferents, which arise from the superior olive in the brainstem, since calcitonin gene-related peptide releasing neurons have been described in peri-olivary locations [36]. We recommend the very recent review about calcitonin gene-related peptide distribution and its effects on cochlear and vestibular systems by Baron and Steenerson [29]. Nevertheless, we assumed that gepants could reach the inner ear either way and hypothesise that they may prevent Menière attacks.

Our first goal was to determine in a small pilot study whether treating patients with Menière's disease and migraine with gepants would support the hypotheses that

1. Menière's disease is caused by calcitonin gene-related peptide, and that
2. gepants can reach the inner ear and prevent Menière attacks.

Positive results would support our second goal of organising a large prospective randomised placebo-controlled clinical trial to test whether this drug will be the first evidence-based effective oral treatment for Menière's disease.

Methods

We describe a series of six Menière's disease patients who were treated with rimegepant, a new medication for treating migraine. Treatment was in accordance with the Declaration of Helsinki as revised in 2023. An Institutional Review Board approval was not required according to Kantonale Ethik Kommission Zurich (BASEC_Req-2024.00515).

Patients with definite Menière's disease according to the 2015 Bárány criteria [37] were instructed to fill in a diary listing all migraine and Menière's disease symptoms and their duration. In addition to an intensive history of all symptoms of migraine and Menière's disease, audiograms, bilateral bithermal vestibular testing (calorics), video head impulse testing (vHIT) as well as cervical and ocular vestibular evoked myogenic potentials (cVEMP, oVEMP),

subjective visual vertical (SVV) and fundus photography were performed. All anonymised test results will be sent to interested readers on request.

In addition, patients were specifically interviewed about migraine symptoms such as headache severity, duration, and location, as well as associated symptoms such as increased sensitivity to noise and light, and aura symptoms. If the patient met the ICHD-III diagnostic criteria, a diagnosis of migraine was made [7]. If both diagnoses were confirmed, he/she was asked to participate in this pilot study. With simultaneous appearance of symptoms of Menière's disease and migraine in 50% or more the patients also fulfilled the Bárány criteria for vestibular migraine and discrimination between both diseases was not possible. All patients were also asked about previous treatments/medications to prevent migraine and/or Menière attacks and their effects.

We first wanted to treat patients with both Menière's disease and migraine to avoid off-label therapy. We enrolled six patients with the following history of Menière's disease: at least 3 Menière attacks per month in the last 6 months and rotatory vertigo of at least 3 hours in most Menière attacks during the last 6 months. As mentioned above, differentiation between Menière's disease and vestibular migraine was not possible due to the high overlap of symptoms in both diseases. Even the very recent and thorough review by Baron and Steenerson does not provide a specific test that clearly discriminates between the two diseases.

When we saw the very impressive effect in the first four patients, we decided to also try the same medication in one patient with Menière's disease only. Two patients were instructed to start their preventive medication at the beginning of their next Menière's attack to see if there was also an acute effect on the duration of Menière's symptoms.

Results

Six patients agreed to take part in this pilot study. Five had migraine and Menière's disease and one (#6) had had migraine between the ages of 20 and 60 years, approximately, and Menière's disease since her 74th year of life. Table 1 shows their characteristics.

As expected, in all five patients treated with rimegepant (Vydura®) every other day, the frequency of migraine attacks was significantly reduced after the first dose of rimegepant. In addition, all five patients have been free of Menière attacks since their first dose of rimegepant. After five months, one of them (patient #2) was unable to continue the rimegepant regimen due to a supply shortage. During the 3-week period without rimegepant, she had 8 migraine attacks and 6 Menière attacks; after the supply was restored, she had no further migraine attacks or Menière attacks. One of the five patients was free of Menière attacks for more than 8 months – however it is very likely that this would also have been the case for patient #2 if her supply had not been interrupted. Patient #3 has been free of Menière attacks for 7 months; patient #5 for more than 6 months; and patient #6 for almost 5 months. Table 2 shows the duration of freedom from Menière attacks and other descriptive data.

One patient (#4 in table 1) developed a severe skin rash on the whole body as well as pain in the right upper abdomen and diarrhoea. The medication was immediately stopped.

Table 1:
Characteristics of patients included in the study.

Pat. #	Sex	Age at inclusion in study	Age at onset / end of migraine	Age at onset of Menière's disease
1	Female	76 yr	67 yr	65 yr
2	Female	57 yr	25 yr	51 yr
3	Female	45 yr	17 yr	39 yr
4	Male	43 yr	16 yr	34 yr
5	Male	62 yr	41 yr	41 yr
6	Female	82 yr	20/60 yr	74 yr

Table 2:
List of patients, their estimated frequencies of Menière and migraine attacks and their reduced frequencies after starting rimegepant up to the time of the last data collection.

Pat. #	Estimated frequency of Menière attacks (6 months ahead)	Estimated frequency of migraine attacks (6 months ahead)	First rimegepant use (date)	Duration of rimegepant use (months)	Number of Menière attacks while using rimegepant	Number of migraine attacks while using rimegepant	Previous treatments without success
1	10–12	6–12	2023-11-08	>8	0	3	Betahistine, sumatriptan, intratympanic steroids
2	12–14	12–22	2023-11-12 (interrupted from 2024-04-12 to 2024-05-03)*	>8	0	3	Betahistine, sumatriptan, intratympanic steroids, topiramat, Mg^{++}, erenumab, metoprolol
3	7–9	8–9	2023-12-15	>7	0	2	Betahistine, intratympanic steroids, metoprolol, erenumab
4	12–16	12–16	2024-01-05	1 tablet once	12–16, **no rimegepant**	12–16, **no rimegepant**	Betahistine, Mg^{++}, riboflavin, intratympanic steroids, erenumab, fremanezumab
5	8–10	10–20	2024-02-02	>6	0	0	Betahistine, Mg^{++}, riboflavin, intratympanic steroids, sumatriptan
6	12–15	No migraine since 22 years	2024-03-12	>4	0	0	Betahistine, intratympanic steroids, mirtazapine

Mg^{++}: Magnesium.

*The interruption of rimegepant use in patient #2 was due to a supply shortage.

Patient #4 took the first dose of rimegepant at the start of a Menière attack with simultaneous migraine. Although he showed the described allergic symptoms, his Menière symptoms – usually lasting for 6–12 hours – started to improve after about one hour and had completely resolved at 1.5 hours. Interestingly, his migraine symptoms also improved, but about half an hour later than his Menière symptoms.

Patient #5 also took the first dose 14 minutes after the start of Menière symptoms as well as migrainous symptoms (see table 2). While his attacks of spinning vertigo usually lasted 3–6 hours with increased tinnitus and hearing loss usually lasting much longer than vertigo, he used sumatriptan spray before to reduce his symptoms. This reduced the duration of symptoms, especially of vertigo, to 30–60 minutes. All Menière symptoms started to improve 37 minutes after he had taken rimegepant and had completely resolved at 1 hour.

In both patients, migrainous headache with increased light and noise sensitivity lasted longer than the Menière's disease symptoms. Patient #5 became tired and slept for two hours. After this nap, his migrainous symptoms (headache as well as increased sensitivity to noise and light) were also completely resolved.

Discussion

We describe a small case series of six patients with Menière's disease and migraine who were treated with rimegepant. Only patient #6 had Menière's disease only but had had migraine until about 14 years before the first Menière symptoms appeared. Only in this patient can a vestibular migraine be excluded.

We saw an impressive positive effect of rimegepant on acute Menière attacks in two patients, who took the first dose shortly after the combined onset of migraine and Menière's disease symptoms and whose Menière's disease symptoms improved even earlier than their migraine symptoms.

We also saw a promising preventive effect in all six patients of our small first case series. Five of these patients used it as a preventive treatment for both migraine and Menière's disease and patient #6 used it for prevention of Menière's disease only.

The effect in migraine has already been described [39, 40] but we describe very promising effects of rimegepant in both acute Menière attacks and the prevention of Menière's disease. It seems especially remarkable that migraine symptoms were significantly reduced, but Menière's disease symptoms were completely abolished in all five patients during the preventive period, which is ongoing. And even when treating an acute attack, symptoms improved better and faster than migraine symptoms.

This study has several limitations. First, the study sample is quite small. We know that such a small case series constitutes weak evidence and does not prove the effect statistically. Nevertheless, this study was not designed to provide statistical evidence, but as a pilot study to determine whether a larger randomised controlled trial is warranted for this new medication. Therefore, one author (SH) tried to start prevention during an acute attack in two patients with prolonged vertigo to see if an acute effect could be

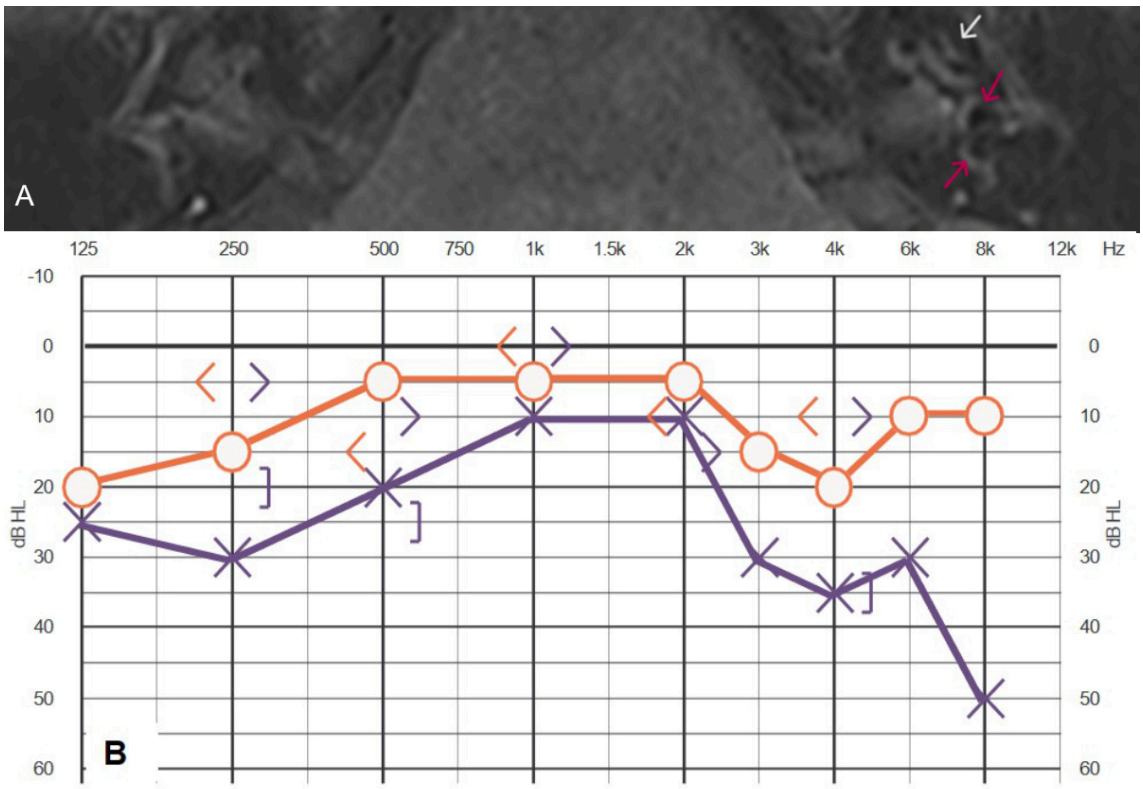
Table 3:
Report of patient #4 (according to table 1) with effects of rimegepant on symptoms during a Menière/migraine attack.

Patient #4	**Date of Menière/migraine attack: 2024-01-05**					
Symptom	**Strength (0–10)**	**Affected ear (right/ left/ both)**	**Time when symptoms started**	**Time when rimegepant taken**	**Time when symptoms started reducing**	**Time when symptoms resolved**
Tinnitus	9–10	Right	15:45	**15:50**	16:40	18:30
Pressure feeling	7	Right	15:50		16:40	17:30
Hearing loss	9–10	Right	15:50		16:40	17:30
Rotatory vertigo	8		15:50		16:40	17:30
Headache	8		15:50		17:30	18:00
Noise sensitivity	8	Both	15:50		17:30	18:00
Light sensitivity	8		15:50		17:30	18:00

Table 4:
Report of patient #5 (according to table 1) with effects of rimegepant on symptoms during a Menière/migraine attack.

Patient #5	**Date of Menière/migraine attack: 2024-02-02**					
Symptom	**Strength (0–10)**	**Affected ear (right/left/ both)**	**Time when symptoms started**	**Time when rimegepant taken**	**Time when symptoms started reducing**	**Time when symptoms resolved**
Tinnitus	7	Left	9:39	**9:53**	10:30	11:00
Pressure feeling	5	Pulse for about 1s	9:39			9:39
Hearing loss	?	Left	9:39		10:30	11:00
Rotatory vertigo	5		9:39		10:30	11:00
Headache	4		9:39		Sleep at 13:30	15:40
Noise sensitivity		Both	9:39			15:30
Light sensitivity	4		9:39			15:30

Figure 1: MRI and audiogram of patient #4 (table 1 and 2). (A) MRI 3D inversion recovery sequence showing cochlear (white arrow) and vestibular (red arrows) endolymphatic hydrops grade 1 in the left ear, according to Baráth et al. [38]. (B) Audiogram 5 years after the start of Menière's disease showing low- and high-frequency hearing loss called "peak.audigram".



observed and then included in a larger study. However, all participants in the study were well characterised both clinically and with audiovestibular function tests. Unfortunately, clear discrimination between Menière's disease and vestibular migraine is not possible, if symptoms of both diseases occur simultaneously. Despite these limitations, we believe that this small case series may be of interest to clinicians who are managing this disabling condition and have no evidence-based non-destructive medical therapy available.

Therefore, we decided to publish these very promising first results in a very small group of patients which support the previous hypothesis that Menière's disease is mainly caused by calcitonin gene-related peptide [24], at least in patients with Menière's disease and migraine and possibly also in patients with Menière's disease only. The efficacy in only one patient with Menière's disease only is even less evident than in the four with Menière's disease and migraine, but it shows at least a possible effect. Since other authors also argue for an inner ear migraine [6, 27, 41, 42], we agree with this concept and we believe that the effect of the therapy shown in this pilot study provides strong support for further evaluating it in a large randomised controlled trial.

With regard to one patient from our small case series, we also suggest that rimegepant may have a significant preventive effect not only in patients with Menière's disease and migraine, but also in patients with Menière's disease only. This will be an important question in a planned randomised controlled trial.

As described in the Introduction, there are no evidence-based therapies described for the treatment of an acute attack of Menière's disease, and all preventive treatments for Menière's disease – except vestibular destructive procedures – are controversial, so there are no generally accepted medications for the treatment of Menière's disease.

Conclusion

Despite the abovementioned limitations of this very small case series and according to our hypothesis, we would suggest treating patients with Menière's disease and migraine as well as those with isolated Menière's disease (without migraine) with rimegepant or other gepants, if they are available. We suggest that rimegepant is a potentially strong medication for prevention of Menière's disease as well as for treating acute Menière attacks.

A larger, double-blind, randomised, placebo-controlled trial is warranted and is about to be initiated to provide scientific evidence of this impressive effect of rimegepant. We will also evaluate the effect of rimegepant on hearing tests, vestibular function tests and endolymphatic hydrops. We hope that we will soon have scientific proof that the hypothesis that calcitonin gene-related peptide causes Menière's disease is correct and that this disabling disease can be effectively treated, significantly improving the quality of life of Menière's disease patients. We are pleasantly surprised that rimegepant appears to be even better at preventing Menière's disease than migraine, for which it was originally developed. And the faster and better effect in acute attacks of Meniere's disease is also very interesting and promising.

Acknowledgments
We thank the pharmacy "City Apotheke zur Sihlporte" for providing the drug at cost (i.e. without a profit margin). A philanthropic donor, who wishes to remain anonymous (but was disclosed to the editorial board) paid the reduced price for the three Swiss patients included.

Potential competing interests
All authors have completed and submitted the International Committee of Medical Journal Editors form for disclosure of potential conflicts of interest. No potential conflict of interest related to the content of this manuscript was disclosed.

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