

Clinical Focus

Management of Recurrent Vestibular Neuritis in a Patient Treated for Rheumatoid Arthritis

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Purpose: This clinical report is presented to describe how results of vestibular function testing were considered along with other medical history to develop a management plan that was ultimately successful.

Method: The patient underwent audio-vestibular assessment including comprehensive audiogram, videonystagmography, cervical vestibular evoked myogenic potential, and postural stability testing.

Results: Results from initial testing were most consistent with uncompensated peripheral vestibular dysfunction affecting the right superior vestibular nerve. These results, considered along with history and symptoms, supported vestibular neuritis. After a second vertigo event, we became concerned about the potential temporal association between the patient's rheumatoid arthritis treatment and symptom onset. It is established that treatment for rheumatoid arthritis can exacerbate latent viral issues, but

this has not specifically been reported for vestibular neuritis. There are reports in the literature in which patients successfully used viral suppressant medication to decrease viral activity while they were able to continue benefiting from immunosuppressive therapy. We hypothesized that, if the current patient's vestibular neuritis events were related to her treatment for rheumatoid arthritis, she may also benefit from use of viral suppressant medication while continuing her otherwise successful immunosuppressive intervention.

Conclusions: Patients treated with biologic disease-modifying antirheumatic drugs are more susceptible to viral issues, and this may include vestibular neuritis. For the current case, identifying this possibility and recommending viral suppressant medication allowed her to continue with successful treatment of rheumatoid arthritis while avoiding additional vertigo events.

One of the more common causes of peripheral vestibular dysfunction is vestibular neuritis. Brandt et al. (2010) reported that vestibular neuritis was the third most common diagnosis for patients with vertigo presenting to their specialty clinic, and this accounted for 7% of their diagnoses. Strupp, Dieterich, and Brandt (2013) found that vestibular neuritis was diagnosed in 8.3% of their 17,718 patients with vertigo, making it their sixth most common diagnosis.

Vestibular neuritis (also termed vestibular neuronitis) is believed to be caused by viral inflammation of the vestibular nerve(s). Herpes simplex virus 1 (HSV-1) is a likely candidate and is often found in autopsied human vestibular

ganglia (Arbusow et al., 1999). It is believed that this virus is present in a latent state in many individuals (Furuta et al., 1993). Symptoms arise when the virus becomes active and replication leads to edema within the nerve. Depending on constraints imposed by the size of the surrounding bony channel, cell and axon damage results (Fetter & Dichgans, 1996). This damage decreases information reaching vestibular nuclei from the peripheral vestibular structures innervated by the affected nerve. A sudden decrease in neural spikes from those affected structures causes an asymmetry at vestibular nuclei, which then leads to the acute symptoms.

The symptom most often associated with vestibular neuritis is intense rotary vertigo with a duration of hours to days. This is present during the acute event. There are usually accompanying symptoms of nausea, emesis, and imbalance. Hearing loss is uncommon as the viral inflammation is isolated to the vestibular nerves and typically the superior vestibular nerve (Fetter & Dichgans, 1996). After the acute event, many patients report symptoms consistent with uncompensated peripheral vestibulopathy (Brandt, Strupp, Arbusow, & Dieringer, 1997). These patients may

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report dizziness with rapid head/body movement, unsteadiness, and, occasionally, oscillopsia.

Vestibular neuritis events often follow some other type of health issue like an upper respiratory infection (Clemis & Becker, 1973). The presence of the other health issues may negatively impact the person's immune system. This is believed to cause replication of the virus, which starts the process of inflammation that ultimately leads to the acute symptoms. This is of potential interest in the current case because patients with autoimmune problems like rheumatoid arthritis are sometimes treated with medications that attempt to disrupt autoimmune activity. These medications include disease-modifying antirheumatic drugs (DMARDs), which can be nonbiologic or biologic. Nonbiologic DMARDs are synthetic and considered "non-targeted" in that the medication, in rheumatoid arthritis, affects the overall processes related to the autoimmune response. Biologic DMARDs are engineered to function as natural proteins in the immune system and disrupt "targeted" steps in the autoimmune response like the binding of tumor necrosis factor-alpha. A detailed review can be found in Burke and White (2014). Changes in the immune system associated with use of medications, including classes of DMARDs, are known to lead to an increased risk for bacterial and viral issues (van Dartel et al., 2013).

Specifically, Tran, Ducancellea, Maison, and Lunel-Fabiania (2017) have shown that immunomodulating drug therapy (including DMARDs) is associated with an increased risk of herpes zoster. There is an increased risk of herpes zoster associated with aging in all individuals. The risk increases from 3/1,000 in patients under 30 years old to 8/1,000 in patients over 60 years old, at which point intervention is recommended. In their analysis, Tran et al. determined that the risk for herpes zoster in patients undergoing immunomodulating drug therapy is at least as high as that seen in the overall population older than 60 years old. The authors recommended prevention with antiviral medication therapy.

Jansen, Vos, and Löwenberg (2016) published a case report on HSV-1 causing necrotizing tonsillitis in a patient treated with a combination of infliximab (Remicade), which is a biologic DMARD, and mercaptopurine for ulcerative colitis. There was no response with antibiotic therapy, so a vaccine was used and is reported to have promptly improved the patient's status. Checchin, Buda, Sgarabotto, Sturmiolo, and Inca (2009) describe a case in which a patient experienced recurrent HSV-1 related to treatment of Crohn's disease with azathioprine, a DMARD. The patient's treatment plan changed to include infliximab. Because that medication has a high incidence of virus reactivation, the authors prescribed oral valacyclovir prophylactically. This allowed the patient to continue with beneficial therapy using the biologic DMARD (infliximab) while controlling potential HSV-1 outbreaks with the viral suppressant medication.

In the current case, we describe a patient who experienced symptoms and presented with results consistent with vestibular neuritis. The vestibular neuritis events appeared to follow treatment with infliximab for her rheumatoid

arthritis. The patient was placed on viral suppressant medication valacyclovir (Valtrex) and was able to continue treatment for rheumatoid arthritis without experiencing further symptoms of vestibular neuritis.

Method

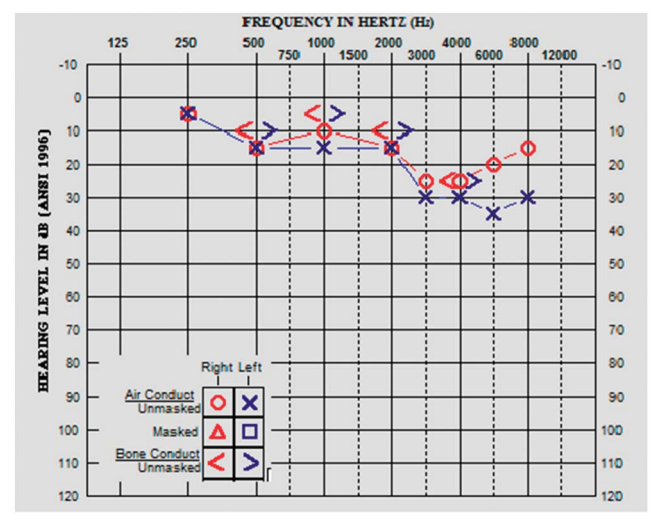
Case Description

A 60-year-old woman presented with history of acute-onset vertigo, emesis, and imbalance for several hours. She reported visiting a local urgent care center where blood was drawn and laboratory tests were completed. The laboratory studies were reported to have been unremarkable. In addition, this patient underwent computerized tomography scan of the head, which was also reported to have been unremarkable. She was placed on meclizine, a commonly prescribed vestibular suppressant, and discharged. There was gradual improvement in status, but the patient remained too uncomfortable to drive a car or ride her horses. She reported exacerbation of symptoms with rapid head/body movement, blurred vision, and unsteadiness. Symptoms were better each morning but worsened throughout the day. The patient was concerned that she may be unable to return to work. By the time of our appointment, she had stopped meclizine. Her other medications included infliximab, methotrexate, and celecoxib (Celebrex). As previously mentioned, infliximab is a biologic DMARD, whereas methotrexate is a nonbiologic DMARD sometimes used in combination with biologic DMARDs. Celebrex is a nonsteroidal anti-inflammatory drug.

Initial Examination and Evaluation

The vertigo event occurred 12 days before our evaluation. Audiogram indicated a mild sensorineural hearing loss of 3000–8000 Hz for the left ear (see Figure 1). Remaining

Figure 1. Pure-tone threshold results obtained at initial appointment are provided (American National Standards Institute [ANSI], 1996).



pure-tone thresholds were normal, and pure-tone averages were in agreement with speech recognition thresholds. Speech discrimination scores were excellent. Tympanometry results indicated normal middle ear pressure and compliance, bilaterally. Ipsilateral and contralateral acoustic reflexes were present at normal sensation levels with stimulation to either ear. No reflex decay was recorded. Initial Dizziness Handicap Inventory (DHI) score was 84, which indicated a severe impact on health-related quality of life. The DHI is a widely used measure of self-perceived impact of dizziness on quality of life (Jacobson & Newman, 1990). Postural stability testing using the Gans Sensory Organization Performance (SOP; The American Institute of Balance, 2012–2018) test revealed a vestibular dysfunction pattern. This is shown in Figure 2. The Gans SOP is a test of static postural stability that incorporates the well-known Romberg, sharpened Romberg, and Fukuda stepping tests. The Romberg tasks are completed in conditions with eyes open and eyes closed. The duration for each task is 20–30 s. These are also completed on a firm surface and a dynamic surface similar to the Clinical Test for Sensory Interaction on Balance (Shumway-Cook & Horak, 1986). Patients with uncompensated peripheral vestibular dysfunction will have difficulty on tasks with no visual cues and limited somatosensory cues. The Fukuda Stepping Test consists of having a patient march in place for 50 steps with eyes closed. Patients with uncompensated peripheral vestibular dysfunction will often turn toward the lesion side and sometimes away from the lesion side. In the current case, the patient had a sway with sharpened Romberg on a firm surface with vision denied and a fall with Romberg on a dynamic surface with vision denied as well as a right turn on the Fukuda Stepping Test. The patient had left-beating spontaneous nystagmus using video-oculography recording with vision denied (12°/s). This enhanced with headshake (18°/s) and suppressed with fixation. Caloric testing revealed a 24% weakness on the right, with a 58% directional preponderance to the left. We note that a weakness of 23% is considered clinically significant in our facility, whereas other facilities use 25%. Cervical vestibular evoked myogenic potential (cVEMP) testing was completed. Single-channel recordings were obtained with a noninverting electrode on the belly of the ipsilateral sternocleidomastoid muscle and an inverting electrode on the belly of the contralateral sternocleidomastoid muscle. Ground was placed on the forehead. Stimulus was a 500-Hz tone burst presented at 95 dB nHL. The patient was positioned supine. She lifted her

head and rotated away from the stimulated ear to contract the sternocleidomastoid during recording. Traces were replicated, and cVEMP responses were normal with stimulation to either ear.

These results indicated reduced vestibular reactivity for the right lateral semicircular canal, which is innervated by the right superior vestibular nerve. cVEMP responses were normal bilaterally suggesting intact saccule and, importantly in this case, inferior vestibular nerve function on each side. The presence of spontaneous and enhancing nystagmus with fast phase toward the left ear was in agreement with Ewald’s law (excitation on the left greater than the right) suggesting dysfunction on the right side, which was the caloric finding. These results, along with postural stability measures, suggested that the patient was not yet compensated to the unilateral peripheral vestibulopathy.

History and symptoms were most consistent with vestibular neuritis given the presence of vertigo, the duration of the event, and the lack of auditory symptoms. Our results suggested that the right superior vestibular nerve was affected. This was supported by the caloric weakness on that side in the presence of bilaterally normal cVEMP responses. Evidence supporting an uncompensated state indicated the need for vestibular rehabilitation therapy (VRT).

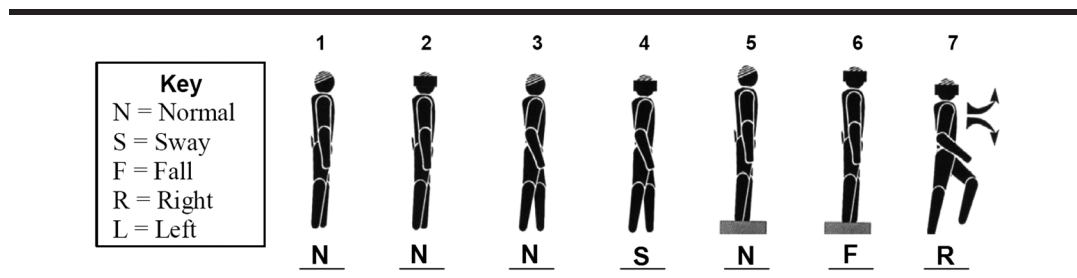
Initial Intervention

Results were discussed with the patient along with a brief explanation of central compensation to peripheral vestibular dysfunction. It was explained that the patient’s decision to stop using vestibular suppressant medication (meclizine) should help the process of central compensation. We recommended gradual resumption of normal activities as the patient continued to experience improvement. We also opted to instruct the patient in a self-directed program of VRT with an emphasis on gaze stabilization, habituation of the dizziness with head movement, and also gait with lateral head turns. The patient was instructed to complete these exercises at least twice each day for 4–6 weeks. A 3-week follow-up appointment was scheduled.

Initial Outcomes

The patient returned after 3 weeks and reported “much improved!” DHI was 4 indicating no impact on health-related quality of life. She had returned to driving,

Figure 2. Initial postural stability results obtained using the Gans Sensory Organization Performance test.



working, and even riding horses. She was continuing with her exercises, but only the gait with head turn exercises provoked symptoms. Gans SOP continued to indicate a fall with Romberg on a dynamic surface with vision denied (see Figure 3). Fukuda and all other Romberg tests were now normal. There was no longer any spontaneous nystagmus, and the left-beating headshake nystagmus was improved (4°/s). This patient was encouraged to continue the VRT and other recommendations for another 1–3 weeks. Otherwise, she was to call as needed.

Second Event Examination and Evaluation

The patient experienced a second vertigo event 14 days after the follow-up appointment. She described the second event as having less intense vertigo and a shorter duration. DHI was 56, which was not as high as the initial evaluation but was still in the severe range. Gans SOP continued to indicate a vestibular dysfunction pattern similar to the follow-up appointment. The only abnormality was a fall with Romberg on a dynamic surface with vision denied. Again, the patient had left-beating spontaneous nystagmus, which enhanced with headshake. No other testing was completed given the similarity in presentation to the initial event and in an effort to keep health care costs reasonable.

It was felt that this patient had experienced another peripheral vestibular event with similar results. On initial evaluation, it was felt that the patient experienced vestibular neuritis. However, recurrence rates for vestibular neuritis only range from about 1.9%–10.7% (Huppert, Strupp, Theil, Glaser, & Brandt, 2006; Kim et al., 2011), so we felt that it was important to consider other possibilities.

This patient did have a diagnosis of rheumatoid arthritis, a chronic condition with autoimmune activity affecting the lining of joints in the body (Li et al., 2017). Rheumatoid arthritis is a health problem implicated in autoimmune inner ear disease (AIED; Barna & Hughes, 1988), so this needed to be considered in the current case. AIED is characterized primarily by bilateral, asymmetric, and progressive sensorineural hearing loss (Bovo, Ciorba, & Martini, 2009). There can be effects on the vestibular system, but this is less common. Accepted treatment of AIED includes steroids and methotrexate, and there is one report of successful treatment with infliximab (Heywood, Hadavi, Donnelly, & Patel, 2013; Jancatova, Zelenik, Kominek, & Matousek, 2015).

Our patient was already using two of the three treatments some have reported as effective for AIED during the course of her rheumatoid arthritis management. Our case had very little hearing loss, and her predominant symptoms were related to vertigo, so AIED, like recurrent vestibular neuritis, remained in consideration, but probability for either seemed small.

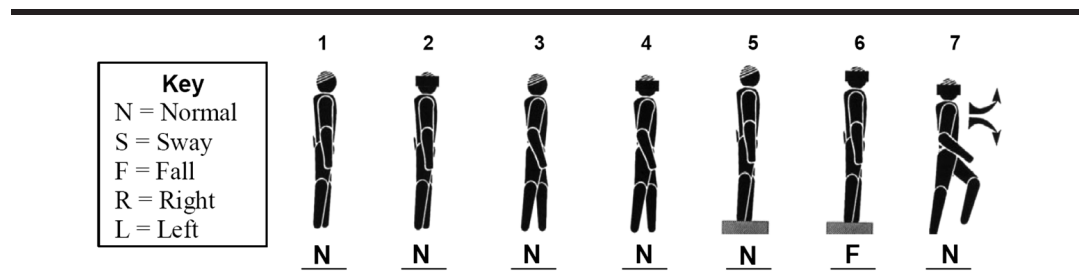
Treatment of the patient's rheumatoid arthritis was further considered. Her infliximab was delivered by infusion therapy. She received an infusion on October 14 and experienced her initial vertigo event on November 7. Her next infusion was on December 15, and the second vertigo event followed on December 20. Infliximab was introduced in 1998 and is classified as a biologic DMARD. This medication specifically blocks tumor necrosis factor-alpha. Patients usually receive this medication every 4–8 weeks, and Singh et al. (2009) reported in a Cochrane review that 43% of patients experienced a decrease in signs of their rheumatoid arthritis when using infliximab compared with 21% of patients who received a placebo. Because this medication affects the immune system, it has been reported that patients on infliximab have an increased rate of infection and virus reactivation (Murdaca et al., 2015; Segan et al., 2015). This includes reactivation of HSV-1, a virus often implicated in vestibular neuritis.

Second Event Intervention

Given the excellent response to VRT, we wanted to recommend the patient to follow our initial plan of resumption of normal activities and self-directed VRT. However, patients with recurrent vertigo events are not ideal candidates for VRT as that intervention does not stabilize the vertigo events and the compensation process must restart with any subsequent events.

We were concerned that the patient could be experiencing recurrent vestibular neuritis with viral reactivation related to use of the biologic DMARD, infliximab. The biologic DMARD was reported to have been quite effective in the management of her rheumatoid arthritis symptoms, so that needed to be continued. We considered recommending use of viral suppressant medication, valacyclovir. There is no evidence that valacyclovir decreases the duration of vestibular neuritis events (Strupp et al., 2004); however, there is one report that valacyclovir used prophylactically may help

Figure 3. Gans Sensory Organization Performance test results obtained at follow-up appointment after 3 weeks of vestibular rehabilitation therapy.



decrease frequency of attacks (Amber, Castaño, & Angeli, 2012). There is also evidence that viral suppressant medication can be helpful in decreasing virus reactivation, including HSV-1, in patients using immunomodulating medications.

Second Event Outcome

This patient did add valacyclovir daily to her medication regimen and has used this since. She reported significant improvement similar to outcomes with our initial management recommendations but was not reevaluated in our office. Infliximab infusion therapy has been received every 8 weeks for 21 months, and there have been no other vertigo events. The patient has discussed continued use of valacyclovir with both her primary care physician and her rheumatologist. Both agree that this can be continued indefinitely.

Discussion

Vestibular neuritis is a common cause of vertigo and peripheral vestibular dysfunction (Brandt et al., 2010; Strupp et al., 2013). There is an association with viral activity, and HSV-1 is found in vestibular ganglia of autopsied patients (Arbusow et al., 1999; Furuta et al., 1993). It is believed that rapid replication of the virus causes edema of the vestibular nerve. If the bony channel of the nerve cannot accommodate the swelling, there is damage and a sudden decrease in neural information from that portion of the vestibular structures. This causes vertigo and other symptoms associated with peripheral vestibular dysfunction like imbalance, nausea, and so forth. It is rare for vestibular neuritis to present as a recurrent issue, but this has been estimated to occur in 10.7% of patients (Kim et al., 2011).

There is some support that patients may be more likely to experience vestibular neuritis after other illnesses, and this may be related to immune system compromise (Clemis & Becker, 1973). Patients with autoimmune issues like ulcerative colitis, Crohn's disease, and rheumatoid arthritis are often managed with immunomodulating medications. There is an increased risk for bacterial and viral issues in these patients, which is believed to be related to the medication intervention (Murdaca et al., 2015; van Dartel et al., 2013). At least a few authors have reported that prophylactic use of viral suppressant medications like valacyclovir is helpful in keeping viral activity in check while allowing the patient to continue with immunomodulating medication therapy (Checchin et al., 2009; Jansen et al., 2016; Tran et al., 2017). To our knowledge, this has never been reported for recurrent vestibular neuritis.

The current case is a patient with history, symptoms, and vestibular test results most consistent with vestibular neuritis affecting the right superior vestibular nerve. Results of functional measures indicated that the patient was uncompensated to this vestibulopathy. Recommendations included gradual resumption of normal activities along with a self-directed program of VRT. The patient experienced significant improvement after only 3 weeks. However, this

patient then experienced a second vertigo event. Because recurrent vestibular neuritis is uncommon, we considered alternatives like AIED, which can be observed in patients with rheumatoid arthritis. Ultimately, we felt that the vertigo events were less likely to be related to AIED because of only a mild sensorineural hearing loss. In addition, some of her medications (infliximab and methotrexate) for rheumatoid arthritis have been reported to be helpful in patients with AIED (Heywood et al., 2013; Jancatova et al., 2015).

It was noticed that both the initial and second vertigo events followed infusion treatment with infliximab. This biologic DMARD is associated with increased viral activity and infection (Murdaca et al., 2015; Segan et al., 2015). In fact, van Dartel et al. (2013) reported that the risk of serious infections in patients with rheumatoid arthritis treated with infliximab was higher than for another DMARD, etanercept. This is consistent with Singh et al. (2009) who reported more withdrawals from trials related to adverse effects for patients treated with infliximab compared with etanercept. The evidence in the literature at least supported the possibility that our patient may have been susceptible to viral reactivation related to her treatment with infliximab.

In consideration of this possibility, we discussed the use of viral suppressant medication to help avoid additional vestibular neuritis events. This was interesting to the patient and her physicians because infliximab was proving to be effective for her rheumatoid arthritis, but she was hesitant to continue with this therapy given any chance of additional disruptive vertigo events. The patient was placed on prophylactic valacyclovir, and this allowed her to continue with infliximab. She has had no other vertigo events in 21 months. During recent discussions with her primary physician and her rheumatologist, both advised her to continue with this regimen indefinitely.

Conclusions

Vestibular neuritis is a common cause of peripheral vestibular dysfunction and is associated with viral inflammation such as reactivation of HSV-1. VRT is effective in promulgation of central compensation but is ideally useful in stabilized, uncompensated patients, not patients with recurrent events. There is evidence that patients using biologic DMARD medications for treatment of autoimmune diseases like rheumatoid arthritis are at an increased risk for virus reactivation given a compromised immune state. Patients using biologic DMARDs could be at an increased risk for recurrent vestibular neuritis, which is usually considered rare. We present a case in which viral suppressant medication valacyclovir allowed a patient with possible recurrent vestibular neuritis to remain on her effective rheumatoid arthritis treatment while reducing the risk of recurrent vestibular neuritis and the need for additional VRT.

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