Journal of Bodywork & Movement Therapies 24 (2020) 59–62

Contents lists available at ScienceDirect

Journal of Bodywork & Movement Therapies

journal homepage: www.elsevier.com/jbmt

Fascia Science and Clinical Applications

Vestibular failure managed with osteopathic manipulative treatment: A report of two cases

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ARTICLE INFO

Article history:
Received 2 February 2019
Received in revised form
29 January 2020
Accepted 17 February 2020

Keywords:
Fascia
Vestibulo-ocular reflex
Vertigo
Dizziness
Proprioception
Osteopathic medicine

ABSTRACT

Introduction: Vestibular failure or hypofunction can be generated by pathologies such as vestibular neuritis (VN), causing the onset of rotatory vertigo and the vestibulo-ocular reflex (VOR) hyporeaction. VN is a post-viral inflammation-producing vestibular nerve-axon impairment, which reaches compensation in 70% of cases. Here, we present two cases of vestibular failure that did not respond to pharmacological therapy, but did show modulated vestibular response after an osteopathic manipulative treatment. Dizziness handicap inventory (DHI) was used to assess disability, while VOR was examined by means of video head impulse test (v-HIT). Case 1 showed bilateral VOR areflexia with severe related disability due to chronic vertigo, while case 2 showed sub-acute VN complicated by intense vomiting. After treatment, both cases had a complete remission of symptoms, with a reduction in DHI score of 60 and 70 points respectively, as well as a normalization of the v-HIT exam.
Conclusion: OMT might work to modulate VOR, through osteopathic manipulation of the fascial-system and interaction with proprioceptive inputs. Further clinical trials should be performed to investigate the OMT clinical efficacy in uncompensated vestibular neuritis.

1. Introduction

Vertigo and dizziness are common symptoms. Dizziness, intended as general term that includes vertigo, has a lifetime prevalence of 15–35% in the general population, with rotational vertigo occurring in 80% of these cases (Neuhauser, 2016). Vestibular neuritis (VN) has an incidence of 3.5–24 cases per 100 000 people per year, with a peak distribution in patients 30–50 years old (Neuhauser, 2016). In 70% of cases there is a spontaneous remission of symptoms within three months, while in the other 30% of cases vestibular compensation is not reached (Halmagyi et al., 2010). VN is characterized by a sudden unilateral vestibular failure which causes severe rotational vertigo that can be sometimes associated with vegetative symptoms, aural fullness or tinnitus (Gacek, 2008). Acute medical treatment seeks to limit autonomic symptoms with levosulpiride, thiethylperazine or metoclopramide. As second line treatment, diazepam or betahistine should be dispensed in addition to vestibular rehabilitation. Manual and manipulative therapies are also recommended in cases of proprioceptive cervical vertigo (Li and Peng, 2015).

Here, we report two cases of acute and chronic un-compensated vestibular neuritis, both refractory to pharmacological therapy, that were managed with osteopathic manipulative treatment (OMT) in addition to standard medical care.

2. Case report

2.1. Case 1

A 55-year-old man presented to our outpatient clinic in July 2014 with a recurrent rotatory vertigo lasting 2 years. He had a past medical history of VN, presenting in 2001 as a single durable vertigo episode that was treated with thiethylperazine for seven days. In 2012 a new episode of paroxysmal rotatory vertigo associated with nausea, vomiting and malaise was diagnosed as benign paroxysmal positional vertigo (BPPV) and treated with thiethylperazine for five days, and the Epley manoeuvre was performed by the ENT specialist. This vertigo episode was the beginning of the onset of chronic oscillopsia, which was managed with betahistine 4 mg/day and vestibular rehabilitation performed by a

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https://doi.org/10.1016/j.jbmt.2020.02.018
1360-8592/

physiotherapist in 10 sessions of postural and neck exercises. In July 2014, a disrupting intense horizontal rotatory vertigo manifested.

At baseline, during neurological examination, the patient was able to stand (but was unstable with Romberg test). Muscle strength in the upper and lower limbs was conserved (5/5), and no cerebellum-related signs were found. An MRI scan, previously ordered by the general medicine specialist, did not show expansive lesions in the brain, brainstem or cerebellum. Vestibular bedside examination revealed a bilateral primary-position nystagmus elicited by Frenzel's goggles. The vHIT presented a VOR areflexia with no test/retest modifications and the DHI outlined severe disability (T0: DHI = 64 pt., Rgain = −0.08, Lgain = −0.04, gain asymmetry = n/a) (see Fig. 1) (Colnaghi et al., 2017; Halmagyi et al., 2017).

Despite the fact that the patient was still following the betahistine regimen, he still complained of bilateral vestibular failure and vertigo. Hence, the ENT specialist requested an osteopathic consultation, in an attempt to assess and improve the function of the somatosensory system through the manipulation of the fascia. The patient was assessed and treated by the osteopath on the same day.

Upon functional examination, upper cervical tract instability was excluded through the alar stress test and active and passive cervical motion tests. The patient's somatic dysfunctional pathway was managed using the inter-scapular and sub-nuchal myo-fascial unwinding technique, sacroiliac balanced ligamentous tension technique, C0–C1, T3-T4 high velocity thrusts and the palatine-fascia release. After the OMT, on the same day the patient was re-assessed with v-HIT revealing a surprising VOR-gain reweight (T1: Rgain = 1.24, Lgain = 1.07, gain asymmetry = 14%).

Ten days after the OMT, the DHI was re-administered resulting in a score reduction of 60 points with a related significant disability improvement. At follow-up of 45 days after the first OMT, the patient presented a stabile bilateral VOR response and normal abilities in daily life activities (T3: DHI = 4 pt., Rgain = 1.03, Lgain = 0.93, gain asymmetry = 10%) (see Table 1). Hence, no further formal follow-ups were collected owing to the clinical stability of the patient.

2.2. Case 2

A 61-year-old female counsellor presented in September 2017 to the outpatient clinic guided by her husband, complaining of a disabling left rotatory vertigo associated with movement fear. The patient's history revealed a misdiagnosed rotatory vertigo attack occurring in 2013, which was managed with betahistine. Moreover, she reported two whiplashes which occurred in 1986 and 2004 and two abdominal-pelvic surgeries (appendicectomy in the 1979, hysterectomy in the 2001). During August 2017, the patient had a vertigo attack accompanied by intense vomiting, which was treated with metoclopramide and betahistine 8 mg/day. Although the intense vomiting crisis remitted, after one month the patient continued to experience a constant rotatory vertigo, concomitantly with incessant nausea and tinnitus.

During the first evaluation, the ENT specialist performed a neurological physical exam, excluding major cerebellum and brainstem signs and severe ataxia. Upon vestibular bedside examination, the patient reported a high-frequency tinnitus, and presented a left-beating, horizontal, rotatory spontaneous nystagmus. The functionality of the vestibular system was assessed with vHIT

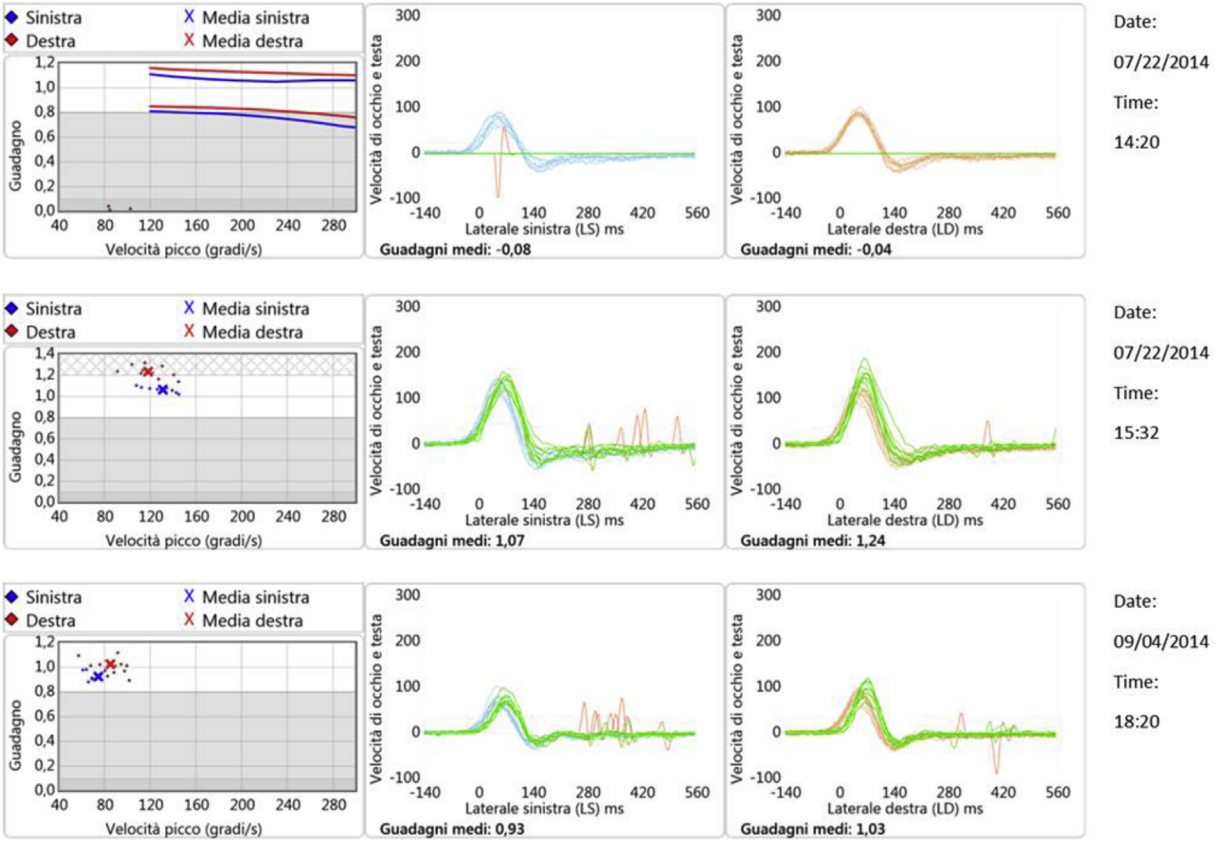

Fig. 1. The three panels show Case 1's v-HIT outcomes at T0, T1 and T2. An absence of a VOR response can be observed in the first chart, followed by a gain reweight in the two subsequent charts.

Table 1
Table 1 collects the main outcomes per the observation times of Case 1. VOR activity was registered immediately after OMT, which was followed by a DHI improvement.

Time points	Date Time	DHI Pt.	Right Gain nu	Left Gain nu	Gain Asymmetry %
T0	07/22/2014 14:20	64	−0.04	−0.08	–
T1	07/22/2014 15:32	–	1.24	1.07	14
T2	08/01/2014 15:00	4	–	–	–
T3	09/04/2014 18:20	4	1.03	0.93	10

and DHI, which denoted a right VOR-gain deficit associated with severe disability (T0: DHI = 72 pt., Rgain = 0.55, Lgain = 0.67, gain asymmetry = 18%) (see Fig. 2).

Later, the patient was referred to the osteopath for a clinical examination of the myo-fascial system and musculoskeletal apparatus. According to the cervical spine active and passive motion tests, upper cervical tract instability was excluded. Somatic dysfunctions were identified in the pelvic girdle and cranio-cervical region. The first OMT was performed on the abdominal-pelvic scar and the sacroiliac fascial complex, combining the use of direct and indirect techniques (Tozzi, 2012).

VOR functionality was assessed immediately following the first OMT, showing a VOR-gain asymmetry reduction of 15% from baseline. Two other OMTs were provided in the subsequent month, intervening on the peri-orbital fascia, palatine fascia, scapular region and T4-T5 with high velocity thrust manipulation. No VOR-gain asymmetry variations were revealed during the second vHIT assessment performed after the second OMT (T2, Rgain = 0.67, Lgain = 0.69, gain asymmetry = 3%). At the end of osteopathic therapy, one month after the first intervention, v-HIT and DHI were re-assessed and displayed a substantially stable clinical scenario (T3, DHI = 4 pt., Rgain = 0.75, Lgain = 0.82, gain asymmetry = 9%). Remarkably, the patient had vertigo and nausea remission soon after the first OMT. The VOR reweight started after the first manual

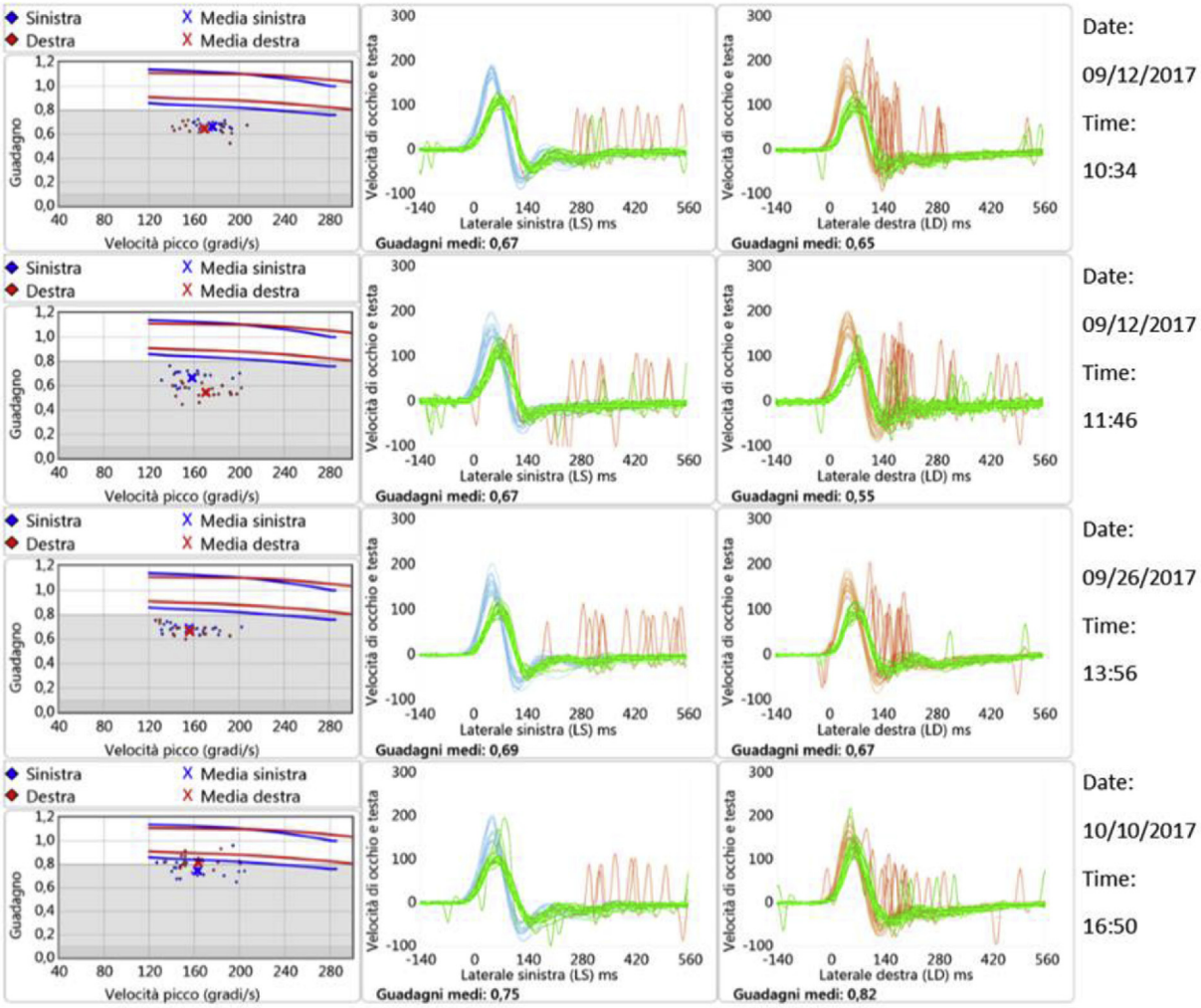

Fig. 2. The first panel refers Case 2 at, T0 where a right VOR deficit can be observed. The second panel highlights the T1 VOR response immediately after the first OMT, showing an up-regulation of right gain. The T2 and T3 v-HITs are shown in the remaining panels, displaying a constant VOR modulation improvement.

Table 2
Table 2 summarizes Case 2's main clinical outcomes. After the OMT intervention (T1) improvement can be seen in VOR reweight, concomitantly with daily life activity.

Time points	Date Time	DHI Pt.	Right Gain nu	Left Gain nu	Gain Asymmetry %
T0	09/12/2017 10:34	72	0.55	0.67	18
T1	09/12/2017 11:46	–	0.65	0.67	3
T2	09/26/2017 13:56	–	0.67	0.69	3
T3	10/10/2017 15:24	4	0.75	0.82	9
T4	01/10/2018 16:00	2	–	–	–

intervention, and it was accompanied by symptoms reduction and disability improvement (see Table 2). At a follow-up of 3 months after the last visit, the patient had a reduction in DHI of 70 points, with no further vertigo episodes.

3. Discussion

To the best of our knowledge, these two VN patients are the first cases in literature documenting the OMT-induced effects resulting in VOR gain reweight followed by a vertigo cessation. These two different cases both presented with vestibular hypofunction and limited response to pharmacological therapy. After OMT, both patients' DHI scores decreased and VOR gain impairment significantly improved.

The vestibular system has a multisensory bilateral-integrated organization, and the precision of VOR can be attributed to the constant modulation of the vestibular, proprioceptive, visual, and inter-vestibular cues computed at the level of the vestibular nuclei (Dieterich and Brandt, 2008; Cullen and McCrea, 1993). VOR is fundamental to maximize gaze stability during active and passive motions. An experiment on rhesus monkeys described the capacity of vestibular nuclei neurons to become more sensitive to proprioceptive stimuli after vestibular loss, whereas they were previously activated mainly by the vestibular afferences (Sadeghi et al., 2011). The fascial system contributes to proprioceptive sensibility as a body-wide proprioceptive organ via its innervation and connection to the musculoskeletal system (Benjamin, 2009). In the fascial context, somatic dysfunctions can be caused by either chronic or acute traumas, which can lead to altered tissue texture, asymmetry, reduced range of motion, and tissue sensitization (Fryer, 2016).

Interestingly, both patients responded with VOR-gain remodulation immediately after the osteopathic manipulations (see Figs. 1 and 2), leading to vertigo cessation which was maintained in the subsequent months of follow-up. Nonetheless, it is improbable that OMT can induce changes in the vestibular organs or inner ear. Based on the clinical evolution of those two cases, the correlation of the fascial system with central vestibular modulation and the efficacy of OMT in the treatment of vertigo should be further investigated.

4. Conclusions

This clinical experience in two patients with VN highlights the possible influence of fascial dysfunctions on vestibular compensation and VOR modulation delay.

Declaration of competing interest

The authors have no relevant financial conflict of interest.

Acknowledgments

The authors thank the participants of the present study.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jbmt.2020.02.018.

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