The omohyoid and sternocleidomastoid muscles entrapment of the internal jugular vein: Which role in Ménière disease patients? Treatment perspective description

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Abstract

The objectives were to analyze the Internal Jugular vein entrapment caused by muscles compression and the possible role and correlation in Ménière disease. We describe the eco-color Doppler evaluation of a sternocleidomastoid and omohyoid muscles compression of internal jugular vein in a Ménière patient, responsible of an anomalous venous cerebral and ear outflow. The proposed treatment was a three months muscular decontraction physiotherapy program. The physiotherapy session allowed a complete muscles relaxation with an improvement of Internal Jugular vein caliber associated to a normalized cerebral and inner ear venous outflow and a progressive attenuation of Ménière symptoms during the treatment and its disappearance at the end of the physiotherapy program. The comfort of the patient was confirmed during one-year follow-up. Muscles entrapment of Internal Jugular veins may be correlated with an anomalous cerebral and inner ear venous outflow, promoting the Ménière disease symptoms. Physiotherapy treatment may represent an intriguing option alternatively of muscle surgical. The present case seems to indicate a possible first line treatment by physiotherapy, reserving surgical resection to not responders. Further studies with a wider sample of patients are warranted.

Introduction

Zamboni and his team have first investigated the role of venous system in the cerebral hemodynamic mechanisms as well as brain and ear physiology, describing its characteristics through the anatomical and functional point of view, indicating the differences in terms of elasticity and compliance1 and underlining the comparable importance in relation to the arterial system that, until then, had attracted almost completely the attentions and finally defining the chronic cerebro-spinal venous insufficiency (CCSVI) as an altered head outflow due to intraluminal defects2 and generating an increased interest in the last years about the possible involvement as common ethiopategenetic mechanism in different pathologies as multiple sclerosis (MS), Parkinson or Alzheimer disease, Ménière syndrome (MéS) and migraine.3

Which role in Mèniére disease of the internal jugular vein:domastoid muscles entrapment

The omohyoid and sternocleidomastoid muscles entrapment of Internal Jugular vein (IJV) may be correlated with an anomalous venous cerebral and ear outflow and a progressive attenuation of Ménière symptoms during the treatment and its disappearance at the end of the physiotherapy program. The comfort of the patient was confirmed during one-year follow-up. Muscles entrapment of Internal Jugular veins may be correlated with an anomalous cerebral and inner ear venous outflow, promoting the Ménière disease symptoms. Physiotherapy treatment may represent an intriguing option alternatively of muscle surgical. The present case seems to indicate a possible first line treatment by physiotherapy, reserving surgical resection to not responders. Further studies with a wider sample of patients are warranted.

Case Report

A 54-years-old woman with MéS disease related to fastidious and continuous bilateral tinnitus associated to auricular fullness (mainly in the left ear) and weekly episodes of dizziness related sometimes to autonomic phenomena as nausea and vomiting was admitted to our Audiology Department and was subjected to audiometric examination (normal) as well as cerebral computed tomography and magnetic resonance imaging scans (negative). Ethics board approval was obtained for this study. We decided to perform a cerebrospinal outflow Eco-color Doppler (ECD) ultrasound evaluation of the vertebral vein (VV) and IJV, revealing a partial muscular compression of right IJV and a complete muscular compression of left IJV in J2 and J3 segments (Figure 1A and B) responsible of an anomalous venous flow, in almost complete resolution during Valsalva maneuver, revealing the real caliber of IJV and the absence of vascular anomalies with a normal outflow, denoting a type 2 of CCSVI.

Therefore we directed the patient to a physiotherapy treatment for muscular compression mainly focused to SCM and OM (first used for this CCSVI type), with the goal of a muscle complete relaxation and a restoration of physiological IJV caliber and venous outflow. After one month treatment ECD analysis revealed a partial muscle decompression and partial caliber improvement of IJV (Figure 1C and D) while after a complete three months cycle (Table 1) of physiotherapy treatment, we repeated ECD analysis observing an almost complete basal reduction of muscle compression and a normalized outflow through IJVs (Figure 1E and F) with an enhance of comfort for the patient due to tinnitus progressive attenuation and auricular fullness disappearance. During one-year follow-up, the patient maintained the improved quality of life, without no more episodes of dizziness.

Discussion

Ménière disease was described for the first time in 1861 and defined as an idiopathic syndrome of endolymphatic hydrops,4 but still today the mechanism of this pathology is not yet fully understood and several heterogeneous factors are related as possible responsibles: inflammation or autoimmune disease, genetic predisposition,
hypoproteinaemia, viral infection, dietary deficiencies.7

In the last years, the presence of cerebral venous system abnormalities encountered by ECD, according to the CCSVI criteria and originally exclusively related as a contributing factor to MS,2 with a significant impact on brain pathophysiology, especially on intracranial fluid balance and cerebrospinal fluid (CSF),3 has been recently correlated with MëS, since these cerebrovascular alterations could also affect inner ear districts, which also drain into the IJV system. An anomalous venous outflow through the IJV and VV may be responsible of a progressive reduction or altered venous drainage into the venous cerebrospinal system of the anterior and posterior vestibular veins and/or of the cochlear veins with the result of an excess of endolymphatic volume secondary to stria vascularis cellular damage loosing the normal homeostasis. The elevation of hydrostatic pressure, due to venous outflow obstruction, the chronic venous stasis and the damage of stria vascularis increase the fluid from the capillaries into the endolymphatic space in a mechanism involved in MëS.9

Recently, Bavera et al. assessed the presence of cerebral outflow anomalies in both, a population affected by MS and a population affected by MëS, demonstrating that not only there was a high prevalence of these anomalies in these two populations compared to a control population,10 but also and above all that there were different patterns in the two pathologies.

Their work underlines that CCSVI can be considered as a common mechanism, which differs as a pattern according to the pathology of MS or MëS, observing in MS patients anomalies mainly related to the J1 and J2 segments, compared to the population with MëS, which would present more anomalies in the J2-J3 segments, as well as jugular bulb anomalies and mediolateral or anteroposterior position that may determining encroachment of the surrounding structures.

Our team has first reported the treatment of a type 1 CCSVI, the so-called hydraulic form, due to venous anomalous outflow related to vascular obstacle in a MëS patient confirmed by phlebography and treated through IJV angioplasty.

In case of Type 2 CCSVI, the called mechanical form, due to muscles compression of IJV, the only described treatment in literature is a surgical procedure through an OM surgical transection together with a patch angioplasty.12

In a population of 56 patients affected by CCSVI, Zamboni et al. showed interesting results by combining to the procedure of endophlebectomy with removal of the defective valves (used as an alternative to the IJV angioplasty), the muscular resection, in cases where a muscular compression on the IJV or VV was present. In this article Zamboni showed how endophlebectomy could be a viable alternative to angioplasty (because of IJV angioplasty is burdened by recurrences of anomalous outflow, especially in cases of nonmobile valve leaflets), associating muscular resection, in the course of the intervention, in case of pattern III CCSVI (the mixed form due to vascular anomalies and muscle compression), re-establishing a complete normalization of cerebral outflow.13

Here we describe first the treatment of a MëS patient with IJV entrapment by SCM and OM and anomalous venous outflow with a not invasive option. The physiotherapy treatment has allowed a progressive muscles decontraction and a directly proportional IJV caliber improvement, avoiding a surgical intervention. ECD analysis performed both during the treatment and at the end of a three months cycle, confirmed the normalized venous outflow and the patient improvement quality of life, confirmed during one-year follow-up.

The conservative treatment, represented by a purposeful decontracting physiotherapy of the OM and SCM muscles (as described in Table 1) and their compression exerted on the IJV, may represent the first-line treatment for such patients, because clearly less bloody than muscular resection, the only treatment so far available.

The surgical option, clearly, can be a second choice treatment in case the targeted physiotherapy is not able to resolve the entrapment of the IJV.

The present case seems to indicate a possible first line treatment by physiotherapy, reserving surgical resection to not responders. Further studies with a wider sample of patients are warranted.

### Table 1. SCM and OM muscles physiotherapy treatment.

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<tr>
<th>Physiotherapy treatment for muscular compression</th>
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<tr>
<td>- Physiotherapy and osteopathical approach, releasing vertebrae and bone fragments where these muscles are attached, giving it back their proper functionality, just as much as to relevant metameres innervation</td>
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<td>- Fascial approach starting from the myofascial tissue shall be carried out manoeuvring of pompage, overstretching and mild compression of the muscle tissues until a feeling of their re-lease</td>
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<td>- Both approaches should be implemented with repositioning methods on the planes (sagittal, coronal, transverse)</td>
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![Figura 1. A) SCM and OM compression of IJV (blue arrow) during trasversal ECD analysis. The IJV appears oval, losing its natural shape. B) Longitudinal ECD analysis with evidence of IJV entrapment (blue arrow) and the absence of flow at PW Doppler. C) Partial muscles relaxation and IJV caliber improvement after one month physiotherapy-apy treatment: longitudinal ECD analysis confirmed the partial muscle compression resolution with a minimal flow detected at PW Doppler (D). E) Complete IJV caliber improvement at the end of physiotherapy treatment, confirmed in longitudinal axis and a normalized outflow in PW Doppler (F).]
Conclusions

The recovery of cerebral venous haemodynamics and inner ear concurrent to a progressive improvement of the patient conditions and to the desappearance of MeS symptoms raises different matters: i) what role can we actually give to the muscular components and to the compression that they exert altering and impairing the venous outflow; ii) if the variability of muscular tone could be related in a meaningful way to a more or less correct changing of the posture held by the patience, to his job, to his daily physical activities, so acting on the venous aspect a fairly substantial and decisive compression; iii) if this muscular compression variability could be reflected to the typical variability and fluctuations of symptoms of MeS patients, representing the physiotherapy treatment a valid option with effective benefits, rather than the surgical muscle resection.

References