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ORIGINAL ARTICLE

Vestibular findings in patients with Vogt-Koyanagi-Harada syndrome

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Abstract
Objective: To identify and describe vestibular abnormalities in patients with Vogt-Koyanagi-Harada syndrome (VKH).
Materials and method: Prospective, cross-sectional, observational, and descriptive study. Patients with VKH referred by an ophthalmological centre were interrogated and physically examined in search of signs of vestibular abnormalities, and if positive, they underwent videonystagmography, computerized dynamic posturography, tonal audiometry, and tympanometry.
Results: Out of 21 patients with VKH, only 10 were included in the study due to presenting data of vestibular abnormalities (10/10 with vestibular symptoms and 9/10 with abnormalities in the physical examination). The mean age was 37.8 years. The videonystagmography was mainly abnormal in ocular saccades test (10/10). The posturography showed a higher alteration of the visual (4/10) and vestibular (4/10) afferents. A diagnosis of benign paroxysmal positional vertigo was mostly concluded (6/10). None presented abnormalities of the middle ear nor data of central pathology, 6/10 presented abnormalities in tonal audiometry.
Conclusions: Peripheral vestibular disorder is often present in the population with VKH.

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PALABRAS CLAVE
Vogt-Koyanagi-Harada;
Síntomas vestibulares;
Videonistagmografía;
Posturografía

Hallazgos vestibulares en pacientes con síndrome de Vogt-Koyanagi-Harada

Resumen
Objetivo: Identificar y describir alteraciones vestibulares en pacientes con síndrome de Vogt-Koyanagi-Harada (VKH).
Material y método: Estudio prospectivo, transversal, observacional y descriptivo. Se interrogó y exploró físicamente a pacientes con VKH referidos de un centro oftalmológico, en busca de datos de alteración vestibular. De ser positiva, se les realizó videonistagmografía, posturografía dinámica computarizada, audiometría tonal y timpanometría.
Resultados: De 21 pacientes con VKH, sólo se incluyeron a 10 por presentar datos de alteración vestibular (10/10 con síntomas vestibulares, 9/10 con alteraciones en la exploración física). Media de edad, 37,8 años. Videonistagmografía principalmente alterada en prueba de sacadas oculares (10/10). Posturografía con mayor alteración de las aferencias visual (4/10) y vestibular (4/10). Se concluyó mayoritariamente el diagnóstico de vértigo postural paroxístico benigno (6/10). Ninguno presentó alteración del oído medio ni datos de afección central; 6/10 tuvieron alteraciones en la audiometría tonal.
Conclusiones: La población con VKH frecuentemente sufre afección vestibular periférica.

Introduction

The Vogt-Koyanagi-Harada (VKH) syndrome is a systemic inflammatory disease that affects the eye (bilateral panuveitis with severe iridocyclitis, serous detachment of the retina, diffuse oedema of the choroid, and hyperaemia of the optic disc), inner ear (tinnitus, hearing loss, and vertigo), skin (alopecia, hypersensitivity to touch, poliosis, and vitiligo), and meninges (headache, neck stiffness, and focal neurological signs).[1] It has a worldwide distribution but is most common in the Oriental, Hispanic and Native American ethnic groups.[1-3] Its frequency is not well known; in Mexico, Arellanes García et al[1] reported 6.4%of cases of uveitis in a reference ophthalmology centre. The gender more often affected is females in a ratio of up to 2:1.[4] It occurs most often between the second and fourth decade of life,[1] but may occur outside this age range.

Many studies have noted that it results from a cellular immune process directed against melanocytes, since the absence of melanocytes in the basal epithelium of skin and their scarcity in the choroid and the inner ear have been demonstrated. Normally, melanocytes and/or free melanin are found in the posterior labyrinth in the vicinity of ridges and maculae (utricle), membranous ducts, common cross, and saccule.[5,6]

The diagnosis is mainly clinical and ophthalmic. The disease is divided into 4 stages: prodromal, uveitic or acute, convalescent, and recurrent. Cytologic abnormalities may occur in the 4 stages, usually concurrent with ocular inflammation. Vestibular manifestations have been reported less often than audiological ones, but they are considered uncommon[2] in the form of vertigo, horizontal nystagmus, abnormal vestibular ocular reflex, absence of response in caloric tests, and abnormal slow tracking eye movement.[7,8] There are few vestibular publications on VKH, so they are analyzed in this paper.

Materials and method

Patients referred by the Eye Hospital with a diagnosis of VKH were recruited. Once informed consent was obtained, we carried out a history with a neuro-otological approach, with an emphasis on semiotics and physical examination in search of data on vestibular alterations.

Specific studies were conducted to determine the vestibular function only in patients with VKH who reported vestibular symptoms and/or in whom physical examination provided compatible data (alteration of measurements, diadochokinesias, Romberg, Babinsky-Weill progress or in tandem). A videonystagmography was also conducted (CHARTR VNG software for Windows, videogoggles and visual stimulator [luminous bar] placed 1.22 m away from the patient). We conducted saccadic eye tests (140 s recording of pseudo-random moving visual stimulus, target amplitude 5-30º), a search for spontaneous nystagmus with visual fixation (eyes open looking to the centre, right and left and a 140 s recording for each position), pendulum tracking (3 cycles with each of the following frequencies: 0.2, 0.3, 0.4, 0.5, 0.6, and 0.7 Hz, motion amplitude 16.7º, 140 s recording), optokinetic (right and left at 20º/s, 140 s recording), Dix-Hallpike (in search of nystagmus with cervical traction to the right and left with 140 s recording on either side), caloric tests at 30 and 44ºC, using 20 mL of water for 30 s, in the following sequence: right ear, hot water; left ear, hot water; and then cold water in the same order. With the Jongkees formula, we considered a difference of >22%% in the response between the 2 ears with both caloric stimulations, hot and cold, to be canalicular paresis, and a predominance of more than 28% in ocular response in either direction with the same caloric stimuli to be preponderance.

Next, a posturography test (EquiTest® System, Version 8.0) was carried out by conventional techniques in 6 situations

for the study of sensory organization: *1*) eyes open, fixed visual environment, and support platform; *2*) eyes closed and fixed support platform; *3*) eyes open, mobile visual environment, and fixed support platform; *4*) eyes open, fixed visual environment, and mobile support platform; *5*) eyes closed and mobile support platform; and *6*) eyes open, mobile visual environment, and mobile support platform. Afterwards, the computer system automatically analyzes sensory radii dividing the average balance of 2 or more situations to identify deficiencies in individual sensory systems, according to the necessary inputs to maintain balance (visual, vestibular, and somatosensory[9]). This establishes 4 basic radii that can be combined to indicate afference with deficit in the proper maintenance of equilibrium (Table 1 and Figure 1). There is also an aphysiological pattern that, in general, shows a better performance in the more difficult situations (e and f) rather than in the easy ones (a and b) of the posturography and regular oscillations without reaching a fall.

In a soundproofed chamber, we carried out standard techniques using an Orbiter 922 audiometer (ANSI regulation S-3.6) for conventional audiometry with air and bone pathways. Then we performed tympanometry (Zodiac 901 tympanometer, Madsen Electronics, ANSI regulation S-3.39), to rule out middle ear alterations or other disorders which by themselves probably conditioned a vestibular disturbance.

Results

Of the 21 patients diagnosed with VKH (2 in acute uveitic stage, 14 in convalescent, and 5 in reactivation), we only included the 10 who met the criteria of signs and/or symptoms of vestibular alteration (2 in acute uveitic stage, 5 in convalescent, and 3 in reactivation).

The mean age of the patients included was (37.8 [11.6] years), 9 women and only 1 male. The time since diagnosis of VKH was an average of 26 (7) months.

None had any relevant hereditary or family history of the current disorder nor any neuro-otological disease data clearly distinguishable from the disorder being studied under interrogation or clinical or instrumental examination. Among the personal data of importance to the vestibular disorder, we found 1 patient with arterial hypertension under adequate medical supervision, 1 with a history of head trauma apparently without sequelae, and 5 who used labyrinthine toxics (1 with occasional gentamycin and others with cytotoxics: 1, methotrexate and 3, cyclophosphamide, for more than 3 months as part of the treatment for VKH). It is important to note that none of these 10 patients had any other risk factor such as age >60 years or diabetes mellitus.

Vestibular symptoms are specified in Table 2. The time of evolution of these symptoms was 7.2 (6.3) months, which is less than the mean time since diagnosis of VKH commented previously. In 7 patients, the duration of vestibular episodes was <1 min, in 2 patients, 1-30 min, and only 1 reported a duration of hours. The frequency of these episodes was reported as 1 to 3 times a week in 4 patients, 4 to 6 times a week in 2, once a month in 2, daily in 1 and fortnightly in 1. Only 4 of the 10 patients with vestibular symptoms reported vagal cortex; 4 patients reported vestibular symptoms in relation to sudden posturocephalic movements; another 4, the start of movement, and 2 others during movement. In 2 cases it was mentioned that symptoms worsened in conditions of darkness.

Signs of vestibular disorder demonstrated by physical examination are described in Table 2. The measurements and diadochokinesias were normal in all patients.

None of the patients presented alteration of the middle ear by tympanometry. Audiometry was abnormal in 6 patients; in 1 it presented a selective drop at 125 Hz; in 4 more, selective drops at 4 and/or 8 kHz bilaterally and 1 unilaterally at the same frequencies. In no case was the fall >35 dB HL.

Videonystagmography

In the saccadic eye test, 8 patients presented bidirectional hypometric saccadic dysmetrias; 1 presented bidirectional hypermetric saccadic dysmetrias and 1 both hypermetric and hypometric bidirectional; none presented spontaneous

Table 1 Sensorial analysis by posturography

Radius[a]	Comparison	Functional relevance
Somatosensory (SOM)	Situation 2 and situation 1	Evaluates the ability of the patient to use somatosensory afference to maintain balance. A low score means that the patient makes little use of somatosensory references
Visual (VIS)	Situation 4 and situation 1	Evaluates the ability of the patient to use visual afference to maintain balance. A low score means that the patient makes little use of visual references
Vestibular (VEST)	Situation 5 and situation 1	Evaluates the ability of the patient to use vestibular afference to maintain balance. A low score means that the patient makes little use of vestibular references
Visual preference (PREF)	Situation 3+6 and situation 2+5	Evaluates the ability of the patient to use somatosensory afference to maintain balance. A low score means that the patient acts on visual afferences, even if they are inexact

Source: Data Interpretation Manual, Equi Test® System, Version 8.0, NeuroCom® International, Inc.; 2001.
[a]The radius identifies the deficiency of individual sensory systems.

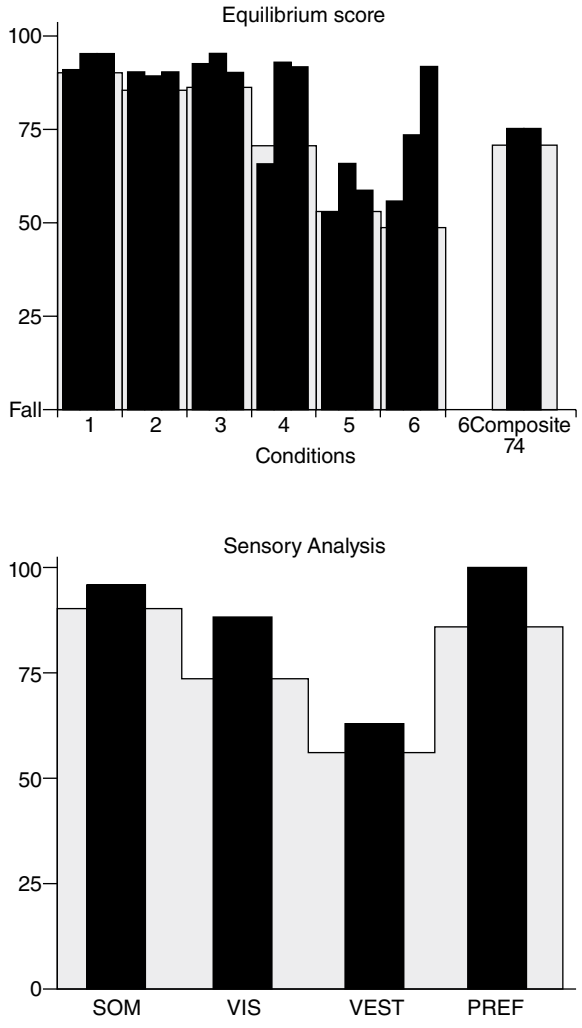

Figure 1 Normal computerized dynamic posturography. The upper graph shows the score awarded in the six posturography situations (abnormal values would be plotted within the shaded area). The bar labelled "Composite" refers to a general equilibrium result. The lower graph refers to the score of individual sensory systems (abnormal values would be plotted within the shaded area).

nystagmus; in pendulum tracing, 9 patients had adequate monitoring of the cycle with occasional interference eye movements; only 1 presented right saccadic movements. In the optokinetic test at 20 and 40º/s to the left and right, all records were symmetric and with adequate direction. No patients had alterations during the search for spontaneous nystagmus with visual fixation. In the search for alterations with the realization of the Dix-Hallpike manoeuvre, 6 patients presented nystagmus, all with features consistent with canalithiasis of horizontal semicircular canal (bilateral in 4 patients and unilateral in 2). In the caloric tests, 2 patients presented canalicular paresis on the right side and 1 on the left; there was also abnormal directional preponderance to the right in 2 cases.

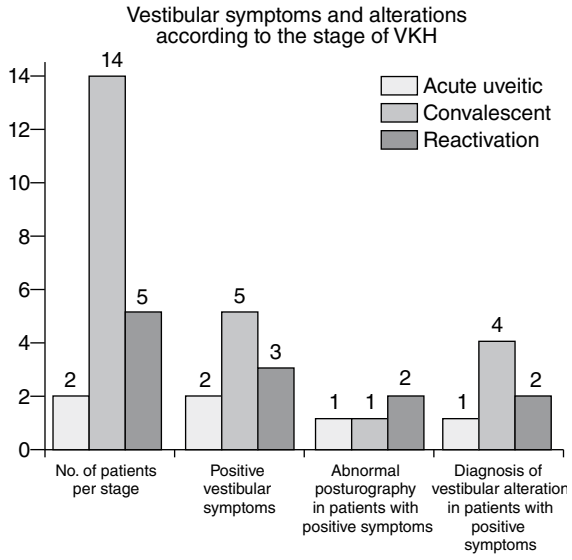

Figure 2 Vestibular symptoms and alterations according to the stage of the Vogt-Koyanagi-Harada (VKH) syndrome. BPPV: benign paroxysmal positional vertigo.

Computerized dynamic posturography

Posturography was normal in 4 cases; only 2 cases showed exclusive vestibular sensory deficit and the other 4 had unique or combined visual deficit with other posturography patterns. No data were found in any patients to show aphysiological patterns. Situation 4 was the most frequently affected (4 patients, combined in one of them with alteration of situations 2 and 3), followed by 5 (2 patients, in one of whom it was combined with alteration in situation 6). It should be noted that in 5 of these 6 patients the altered situations involved open eyes, which reveals an inappropriate use of visual afference. In all 6 cases, the overall outcome of balance was affected, but no falls were recorded.

The following diagnoses were made: benign paroxysmal positional vertigo in 3 patients, benign paroxysmal positional vertigo associated with vestibular dysfunction in 3 other cases, 1 case of pure vestibular dysfunction; 3 patients were classified as with no evidence of vestibular dysfunction because we were unable, through these studies, to reach a diagnosis and the physical examination and the semiology data were very non-specific.

Table 2 and Figure 2 summarize the main points in the semiology, physical examination, and test results for each patient with vestibular symptoms. Figure 2 also shows the total number of patients by stage, both with and without symptoms or signs of vestibular disorders.

Discussion

Almost 50% of the patients with VKH presented vestibular symptoms. Dizziness was the most commonly reported symptom, and vertigo, the least, which disagrees with previously published findings that characteristically mention

Table 1 Relevant data in patients with VKH and vestibular alteration

Questioning						Physical examination			Test results								
										Videonystagmography						Posturography	Vestibular diagnosis
										Oculomotor tests			Dix-Hallpike	Spontaneous nystagmus	Caloric tests		
Stage of VKH	Patient	Age	Months since diagnosis	Vestibular risk factor gait	Vestibular symptoms	Sensitized Romberg	Babinsky-Weill gait	Tandem gait	Tonal audiometry	Ocular saccades	Pendular tracking	OPK			Paresis	Preponderance		
Acute	3	26	6	MTX	Di	+	+	Stable	–		–	–	+	–	–	–	N	BPPV
	15	36	2	C	Di	–	–	Stable	–		M	–	–	–	–	–	Vis, S	NorDil
Convalescent	4	31	36	HT WS	Di	+	–	Stable	–		–	–	+	–	–	–	Ves	BPPV
	5	20	72	C	V	+	+	Stable	+	↑	–	–	+	–	+	–	Vis	BPPV, VD
	6	33	60	G	Di	+	+	Stable	–		–	–	+	–	+	+	N	BPPV, VD
	19	49	6	–	Di	+	+	Stable	+		M	–	–	–	–	+	N	VD
	21	26	8	–	I	+	–	Unstable	+		M	–	–	–	–	–	Ves	NorDil
Reactivation	1	43	18	–	Di, I	+	+	Stable	+		M	–	–	–	–	–	N	NorDil
	1	47	36	C	Di	+	+	Unstable	+		M	–	+	–	+	–	Vis, Ves, S	BPPV, VD
	20	56	16	CAHT	I	–	+	Stable	+		Sa	–	+	–	–	–	Vis, Ves	BPPV

– indicates without alteration; +, alteration in the test mentioned; ↑, hypermetries; , hypometries; C, cyclophosphamide; CAHT, controlled arterial hypertension; G, gentamycin; I, instability in gait, M, aggregate ocular movements, Di, dizziness; MTX, methotrexate; OPK, optokinetic at 20 and 40o/s; S, somatosensory; Sa, saccades; HT WS, head trauma without sequelae; V, vertigo; VD, vestibular dysfunction; Ves, vestibular; Vis, visual; BPPV, benign paroxysmal positional vertigo.

vertigo as a vestibular symptom,[1,2,4,7,8,10,12] although it can be sensed that a possible habituation of patients to dizziness and vertigo diminish the mention of these symptoms during anamnesis. Two patients commented that vestibular symptoms worsened in dark situations, indicating that in them visual input remained compensatory and, therefore, being deprived of it made symptoms worsen.[13] Vestibular symptoms were not mentioned as early symptoms of VKH, contrary to the affirmations of some authors.[10]

Although some of our patients with vestibular disorders presented risk factors, in this study they were not a clear reason for their appearance. The potential use of labyrinthine toxics, managed at appropriate doses, may not trigger vestibular damage; 5 vestibular patients used such drugs, but another 4 patients with VKH without vestibular data, also reported using them, mostly as part of the treatment for VKH.[14] Controlled arterial hypertension in 1 patient and the head trauma of 1 patient were not clearly related either with vestibular symptoms, although though both may be a cause of BPPV.

An alteration of the ocular-vestibular reflex and of the ocular movements of slow tracking (pendular tracking)[7,8] has been reported in patients with VKH, which may be due to the alteration in visual input which they present, which in turn may cause inadequate visual fixation. In our study, the ocular saccadic and pendular tracking tests (both oculomotor) were characteristically altered.

The dysmetria and ocular saccades data recorded in the saccadic and pendular tracking tests, respectively, may be indicative of central vestibular alteration; however, these were isolated data that did not coincide with other vestibular tests or with the clinical presentation to determine central structural alteration, therefore we believe that they were the result of inadequate visual input modifying the integration of information at a central level.

In 6/10 patients with vestibular symptoms, there was some alteration in posturography; in 4 of these cases there was a pattern of visual deficit, indicating that this afference is not contributing to the correct integration of balance. However, in 1 patient, although only vestibular alteration was recorded by posturography, alterations were also found in situation 4, which means that even with open eyes, there was no correct visual afference. A vestibular pattern reinforcing the idea of involvement of the posterior labyrinth in this disorder was also documented in 4 cases.

Endolymphatic hydrops is one of the alterations of autoimmune inner ear disease proposed by Kumagami and Harada.[8] This has been related to the aetiology of BPPV[15] and Ménière's disease. We shall delve further into the hydrops, since it can explain many of the symptoms presented by our patients, in whom the most common vestibular diagnoses were BPPV and vestibular dysfunction.

According to Goycoolea,[16] endolymphatic hydrops can cause vestibular disturbances under 3 theories: *a*) electrolytes, whereby the increased pressure in the labyrinth may cause rupture of Reissner's membrane, thus causing contamination of endolymph with perilymph and the attacks of vertigo by abrupt stimulation of the sensory epithelium of the vestibule; *b*) mechanics: the increased pressure in the membranous labyrinth has a mechanical effect on the sensory cells, causing vertigo; and *c*) osmosis: endolymphatic hydrops causes a physical and chemical

event with osmolar changes that are sufficient to cause vertigo symptoms. The scenarios described may, as already mentioned, result in BPPV-like symptoms, but could also produce cases similar to Ménière's disease. Although our patients in some cases presented audiometric curves consistent with Ménière's disease, they did not meet other clinical features of this condition.

BPPV may also coincide with the theories of cupulolithiasis and canalithiasis, where remnants of otoliths adhere to the dome or are free in the ducts respectively. These otolithic remains may have originated in the otolithic macula.[15] The otolithic macula, the common cross and the utricle have dark cells,[5] at the base of which melanocytes are located. The autoimmune attack against melanocytes in these areas may lead to their degeneration and, in the case of macular degeneration, may appear as BPPV. Under normal conditions, free pigment is found in the membranous ducts, surrounding the crests and in ampullary endings; the alteration of this pigment may also have vestibular consequences. Our results were consistent with canalithiasis of the horizontal semicircular canal (the least common presentation of BPPV[15]), but we do not know the cause of such high frequency in our study.

We must not forget that melanin has been attributed a probable role in the microcirculation of the inner ear and the development of endolymph and, therefore, if its characteristics change, it may alter the vestibular function of the inner ear[5] and cause a vestibular dysfunction to occur, similar to vestibular dysfunction secondary to vascular disorders of other aetiologies such as metabolic. The entrapment of circulating immune complexes in the vascular stria, mediomodiolar vessels and perilymph, originating from cerebrospinal fluid or the bloodstream, has been described in autoimmune vestibulopathy, as well as biochemical and circulatory alterations by vasculitis. These autoimmune phenomena can cause vestibular dysfunction.[17]

Instability is characteristically described in the autoimmune vestibulopathy rather than plain vertigo,[17] which is broadly consistent with our results.

Of our patients in acute phase, 100% complained of vestibular symptoms, followed by the recovery phase (60%) and finally (35%) the convalescent stage. The same behaviour was present in the verification, by videonystagmography, of damage to the vestibular function. An altered posturography pattern was found more frequently in the acute uveitic stage, in second place in the convalescent stage and finally in the recovery stage.

Vestibular symptoms were common in patients with VKH, vestibular lesions were more evident in early stages, perhaps due to a compensatory mechanism which made them less obvious at later stages.

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Conflict of interests

The authors have indicated there is no conflict of interest.

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