Vestibular Neuritis in Pregnancy

Mădălina Georgescu, Sorina Stoian

Institute of Phono-Audiology and ENT Functional Surgery, Bucharest

Acknowledgments: This paper is written within the SECTORAL OPERATIONAL PROGRAMME HUMAN RESOURCES DEVELOPMENT, funded by the European Social Fund and the Romanian Government, as stipulated in Contract nr. POSDRU/89/1.5/S/64109.

Abstract

The authors present a case report of vestibular neuritis, which occurred during pregnancy (week 30), during viral infection of the upper respiratory tract. Once the diagnosis established, we were able to decide on appropriate treatment plan, in collaboration with an obstetrician. The patient was included in a customized vestibular rehabilitation program which led to rapid and almost complete compensation of the abrupt vestibular loss.
Keywords: *vestibular neuritis, viral infection, vestibular rehabilitation*

Acute unilateral vestibulopathy with an abrupt onset, known under the name of vestibular neuritis, is the second peripheral cause of vertigo in terms of frequency[1], with an incidence of 3.5/100.000[2,3]. It can occur at any age, yet it is quite rare in children. Although in most cases the cause cannot be ascertained, the epidemic occurrence of this condition and the histopathological studies proving the viral aetiology of the changes noticed at the level of the vestibular nerve[4,5] (atrophy of vestibular sensory nerve and epithelium) support the viral aetiology of vestibular neuritis. Furthermore, the detection of herpes simplex HSV 1 DNA in the vestibular ganglion, using polymerase chain reaction (PCR), support the role of the virus in the pathogenesis of vestibular neuritis[6-9].

The onset of vestibular symptoms is often preceded by a viral infection of the upper respiratory tract or digestive tract, less than two weeks prior to the appearance of balance disturbances. The vestibular nerve transmits the information sensed at the level of the receptors in the inner ear, meant to maintain balance when moving the head. Maculae receptors monitor static equilibrium (head position with respect to gravity when the body is still). Cristae receptors in the semicircular canals monitor dynamic equilibrium (movement). Impulses from the vestibular apparatus travel along the vestibulocochlear nerve to appropriate brain areas. These centers start responses that fix the eyes on objects and stimulate muscles to maintain balance and avoid dizziness while we are making routine movements. In this way, the normal function of the two inner ears ensures the stabilization of the image on the retina during the head movement, which is synonymous with the lack of dizziness produced by the common movements of the head. When one of the inner ears/vestibular nerves is affected, an informational asymmetry occurs in the central vestibular structures, which is misinterpreted as rotation and triggers a compensatory movement of the eyes. The patient will experience vertigo (the feeling that things are spinning), or dizziness and it is visible as spontaneous nystagmus , although he rests and does not move his head. Function of the unilateral deficit, the vertigo/dizziness can vary in severity, affecting the quality of the patient's life to various degrees.

The predominant symptom in the clinical picture is vertigo, very intense and prolonged (7-10 days), accompanied by nausea and vomiting. The vertigo is increased by any head movement, forcing the patient to lie still in bed. If he gets up, the patient presents severe disturbance of erect posture and gait, needing assistance. Hearing is not normally impaired, as the vestibular nerve is separate from the auditory nerve, both forming the eighth pair of cranial nerves, i.e. the vestibular acoustic nerve. When vestibular neuritis occurs during an infectious disease - mumps, measles, mononucleosis - neurosensory hearing loss can appear at the same ear where vestibular neuritis occurred. Vestibular symptomatology is not accompanied by associated neurological signs (paresthesia, vision or speech disturbances, stroke) to suggest a cerebral tumour or CVA.

The otoneurological examination on presentation reveals severe spontaneous nystagmus (often third degree, so maximum severity), with direction towards the healthy ear. The nystagmus is horizontal, peripheral, having the same direction irrespective of the direction of the gaze, and is fixation suppressed. The Romberg test and head tilt reaction are positive on the side of the affected ear[1]. Watch the movement of the body in relation to a perpendicular object behind the subject (corner of the room, door, window etc.). A positive sign is noted when a swaying, sometimes irregular swaying and even toppling over occurs. The essential feature is that the patient becomes more unsteady with eyes closed. When you ask the subject to walk with eyes closed, stand close by as a precaution in order to stop the person from falling over and hurting himself or herself.

The objective vestibular tests (Electronystagmography (ENG), which registers and quantifies nystagmus, Posturography, vestibular potentials) reveal the partial or total vestibular nerve deficit on the affected side, being of use in monitoring patient evolution under treatment.

In the cases in which a clinical diagnosis cannot establish the cause of vertigo, it is recommended to resort to imaging investigations - cerebral contrast-enhanced MRI, focused on the inner auditory canal, the ponto-cerebellar angle and posterior fossa. Ponto-cerebellar angle tumours, cerebellar infarctions or strokes are thus excluded. Gadolinium administration allows visualisation of the swelling at the level of the vestibular nerve, which is enhanced.

The spontaneous evolution of vestibular neuritis is positive, by the central compensation of the unilateral vestibular deficit. This physiological phenomenon is slow (lasting 3-6 months) and is not perfect, some patients present residual imbalance, especially at abrupt head movements, because of the irreversible lesion produced by vestibular neuritis. Damage of both branches of the vestibular nerve - superior and inferior - occurs in most cases of vestibular neuritis, which explains the singular occurrence of this type of pathology in a patient's life.

Sometimes, the vestibular nerve is only partially affected[15,16], the inferior vestibular nerve remaining unharmed. It will continue to transmit information from the inferior posterior semi-

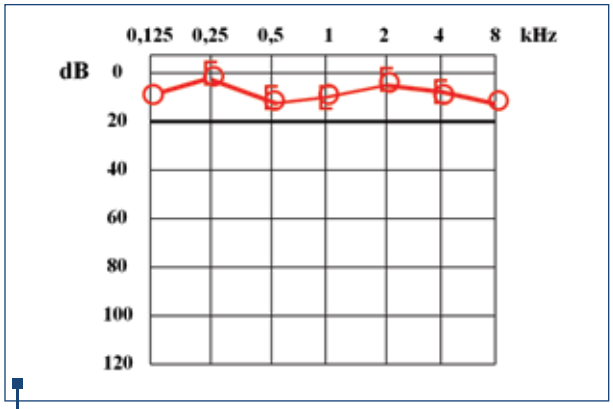

Figure 1

circular canal and saccule, thus explaining the possible complications in the evolution of vestibular neuritis. The damage of the superior vestibular nerve explains the malfunction of the utricle (sensory macular organ of the ear, innervated by the superior vestibular nerve), with the consecutive degradation of otoliths at that level and migration of otoconial debris into the posterior semicircular canal. This weight surplus will stimulate vestibular receptors in the posterior semicircular canal and will transmit excitatory information to the functional inferior vestibular nerve, determining the onset of Benign Paroxysmal Positional Vertigo (BPPV), a complication that is encountered in the evolution of partial vestibular neuritis. It is not a relapse, as patients may think, but a new pathology which complicates the recovery in incomplete vestibular neuritis.

Case report

We present the case of a 27 year old patient, with a 30 week pregnancy, who, within the context of a viral infection, had an abrupt, persistent attack of vertigo, accompanied by nausea and vomiting, and severe gait disturbances. The obstetrician under whose observation she was, requested multidisciplinary consultations, i.e. neurological and ENT, to establish the cause of vertigo. On presentation to the ENT department, the patient complained of vertigo with a history of 40 hours, associated with vomiting, difficulty to maintain an erect position and incapacity to walk without assistance. There had been no acoustic- vestibular symptoms prior to this episode. There was also no history of significant past diseases, neuritis occurring thus suddenly at a young, healthy patient.

The clinical otoneurological exam revealed the following:

- hearing within normal limits (figure1);
- spontaneous third degree Nystagmus - figure 2 (ENG registration, proving that the left superior vestibular nerve was affected);
- pathological head tilt to the left shoulder;
- positive head impulse test (HIT) on the left (figure 3) - which proves reduction in horizontal semicircular canal function in the affected ear (affected superior vestibular nerve);
- great difficulty in the Unterberger Test (stepping on the spot). The patient tended to fall to the left when closing her eyes. During the 50 steps on the spot, the patient pathologically rotated to the left (image in the mirror in the figure presented) and stepped on a much larger support surface (figure 4);

Figure 2

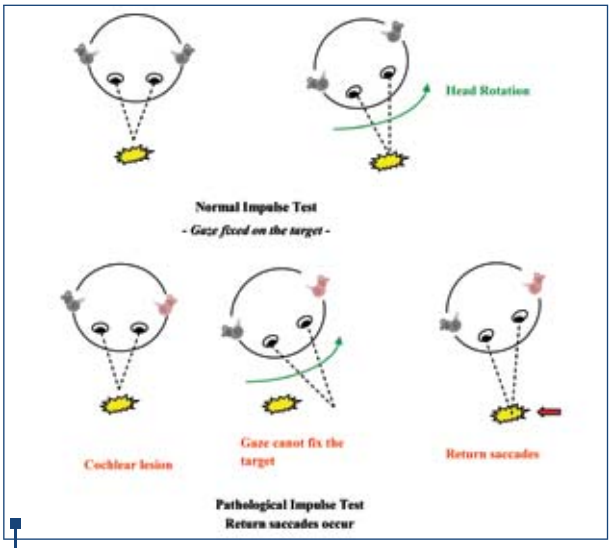

Figure 3

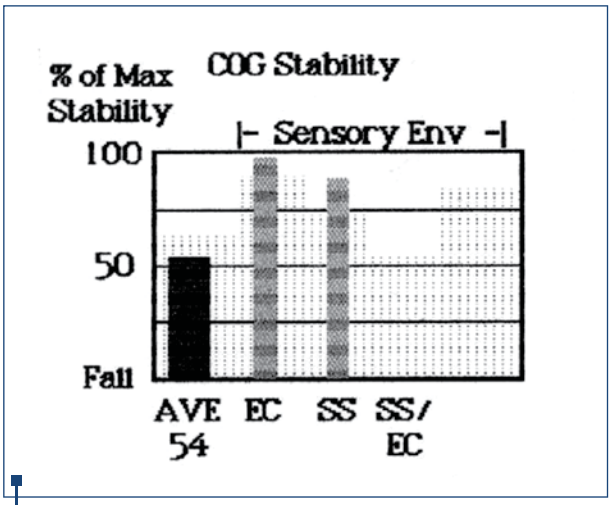

Figure 4

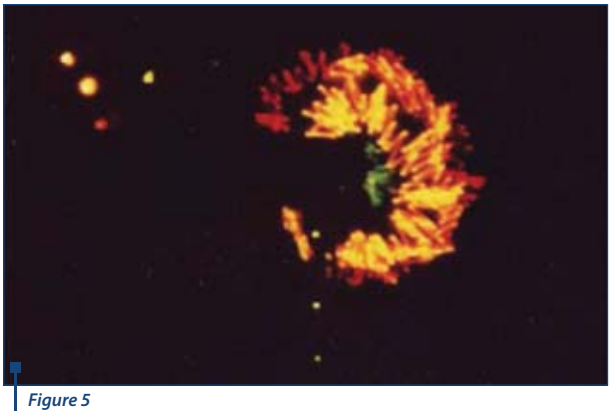

Figure 5

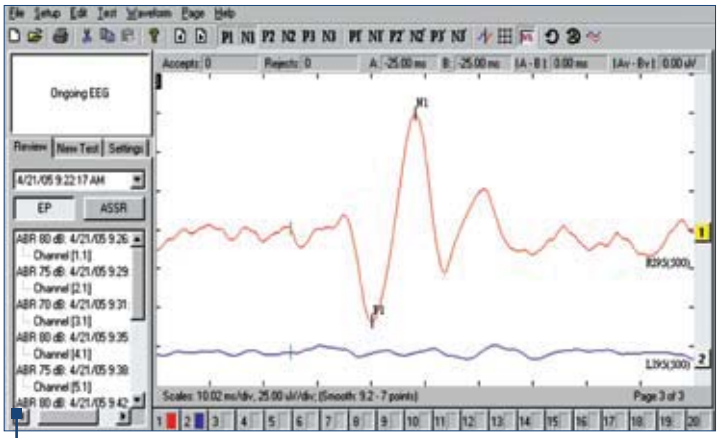

Figure 6

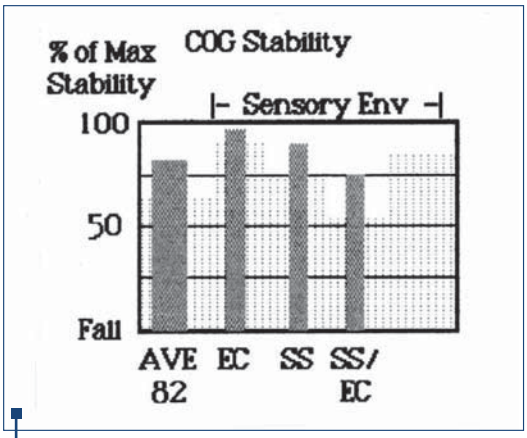

Figure 7

■ posturography - balance disturbances of vestibular origin - (stability 0% when testing the patient, eyes closed, on a mobile platform) figure 5;

■ vestibular evoked myogenic potential (VEMP) testing: absent on the left ear (figure 6), which denotes the viral inflammatory process has affected the inferior vestibular nerve.

After history taking, the neuro-otologic examination completed with a paraclinical audiovestibular one. The diagnosis established was acute complete left vestibular neuritis (both branches of the vestibular nerve being affected).

Our usual therapeutic scheme for vestibular neuritis in the acute phase consists in administration of vestibular sedatives for a maximum of three days and antiemetics (Diazepam and Metoclopramid), to which, from the first day, we associate an injectable steroidal anti-inflammatory treatment for five days and a vestibular neuromodulator (Betahistine, 48 mg/day). The latter will be continued for at least 6 weeks, associated to a personalised vestibular rehabilitation program (physical exercises adapted to each patient). Administration of antivirals (Acyclovir) did not prove to improve the recovery rate or pace, the steroidal anti-inflammatory treatment being statistically more effective[9].

In the present case, after consultation with the obstetrician, we chose as therapeutic protocol only the administration of Betahistine, simultaneous with exercises of vestibular rehabilitation. Vestibular rehabilitation is meant to re-teach the central vestibular structures to coordinate body movements with precision, so that the patient can regain balance in various situations of movement (walking on a kerb, so a narrow surface, rotation, head movements while walking, poor illumination, when the information essential for keeping the balance is diminished). In addition, vestibular rehabilitation aims at enhancing the vestibular ocular reflex (VOR) on the side of neuritis, so that the head movement should not trigger the movement of the visual field which gives the vertigo, often accompanied by nausea and discomfort.

For our patient, the rehabilitation exercises were adapted to the limited movement and effort capacity given by the 30 week pregnancy, and, as the vestibular rehabilitation program lasts at least six weeks, a period of advanced pregnancy followed during which she needed to perform physical exercise.

Four weeks later, the patients presented at follow-up: clinically, her balance was much improved; she no longer had vertigo or nausea. The check-up tests revealed her stability was again within normal limits (figure 7) and there was visible improvement in the Unterberger Test - the patient still rotated to the left (55 degrees, the normality limit being 49 degrees) and did no longer use a large support surface (figure 8).

Because of the advanced pregnancy, Electronystagmography (ENG) (caloric stimulation of the vestibular system) was not performed as it would have involved strenuous physical effort (approximately half an hour in dorsal decubitus, the trunk raised at 30 degrees) and could have produced nausea at the stimulation of the healthy ear.

Discussion

In 1949, Hallpike first described vestibular neuronitis as vertigo determined by an organic disorder localised at the peripheral vestibular level and differing from Ménière's disease[10,11]. In 1969, Coates defined vestibular neuritis as an acute unilateral peripheral condition, with persistent vertigo, without hearing loss, accompanied by ipsilateral semicircular canal paresis at caloric test; symptoms disappear completely in 6 months. Yet, in 1999, Brandt supports the term of vestibular neuritis as a better description of the condition, demonstrating that it is the vestibular nerve fibres that are affected rather than its ganglion cells. Furthermore, Schuknecht et al. report clinical cases with recurrent or bilateral vestibular dysfunction, arguing that ipsilateral vestibular dysfunction persists longer, over 6 months. At present, these terms are deemed synonymous. Recovery of patients with vestibular neuritis involves a combination of (1) incomplete restoration of labyrinthine function, (2) proprioceptive and visual substitution of unilateral vestibular deficit, (3) central vestibular compensation of functional asymmetry [12]. A prospective study of randomized therapy showed that vestibular rehabilitation exercise significantly improves postural stability (measured through posturography) in patients with vestibular neuritis, as compared to a control group who were not included in a vestibular rehabilitation program.

Case particularities

The standard treatment protocol of vestibular neuritis presented previously could not be applied in the case under discussion, as vestibular sedatives (Diazepam) are contraindicated in pregnancy. The administration of Diazepam

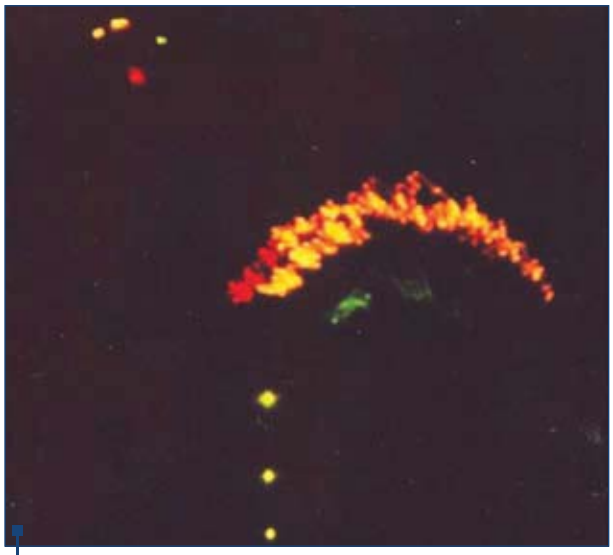

Figura 9

in pregnancy, and especially in the last term, significantly increases the risk of benzodiazepine withdrawal syndrome, floppy infant syndrome, refusal to breastfeed, drowsiness, sleep apnoea, cyanosis and hypothermia[13].

Together with the obstetrician, we limited the treatment of the vestibular condition to administration of Metoclopramid, to alleviate nausea, for two days, two tablets a day.

The studies made on large cohorts of newly born from women in Israel who had received Metoclopramid during pregnancy, revealed no increased risk of congenital malformations, small weight at birth, prematurity or perinatal mortality[14]. This fact extended the acute period of the disease with vertigo and balance disturbances, when the patient was virtually bedridden for a long period of time, as her head movements worsened her symptoms. Prolonged rest in bed impairs the quality of recovery as it delays the onset of vestibular compensation.

As well as that, pregnancy did not allow steroidal anti-inflammatory treatment, which rapidly and significantly reduces lesion inflammation and recovery of vestibular deficit. There is controversy regarding cortisone administration in pregnancy, considering the risk of hyperglycaemia and arterial hypertension that the drug can induce, as well as the increased risk of malformations in the foetus (Pregnancy category C drug - especially administered per os during the first term of pregnancy). Although pregnancy was past the first term and as the treatment should have lasted a minimum of five days, we decided against the use of this drug, for fear of arterial hypertension. All the above mentioned factors delayed patient recovery, extending the period of discomfort and limitation in the patient's daily activities. Yet, thanks to her youth and lack of pathological vestibular antecedents, the recovery of the vestibular deficit induced by vestibular neuritis progressed well, the check-up after four weeks of combined treatment with drugs and physical exercise revealed an important increase in the patient stability and quality of life (no vertigo, whether spontaneous or induced by slight head movements).

From what we know, there have been no other cases of vestibular neuritis reported in pregnancy, which does not allow a pertinent statistical estimation of pathology incidence in pregnancy. In our personal practice, we have diagnosed seven pregnant patients with vestibular neuritis, out of a total of 5000 patients consulted in the past three years. The question that still needs an answer is whether this small number is due to the low incidence of this pathology in pregnancy or to low addressability, considering the scarcity of specific references in the medical data bases. The severity of vestibular neuritis symptomatology, as well as the important discomfort induced by it, especially in a pregnant woman, requires the acknowledgement of this 'benign' condition and referral to the ENT consultant specialised in vestibular pathology.

Conclusions

Vestibular neuritis represents a benign peripheral vestibular pathology, which severely limits the patient's quality of life through the severe unilateral deficit abruptly installed.

The severe, long-lasting symptoms at the onset of this disease has a dramatic emotional impact on the patients, who in their majority believe they must have had a stroke or they are going to die shortly; and it has a physical impact (severe balance disturbances, which may trigger the patients' belief in their incapacity to walk in the street on their own and perform their daily tasks even long after the acute episode).

There are no predisposing factors for vestibular neuritis, as it is a viral disease, which may affect any perfectly healthy person. Pregnant women are probably more prone to it, as their condition modifies the immune status and increases susceptibility to common viral infections.

Pregnancy limits the administration of the right treatment in the acute phase, through the possible consequences of vestibular sedative treatment (Diazepam) and the administration of cortisone on both the mother and the foetus, thus prolonging the recovery of vestibular deficit.

The only possible methods of treatment remain: supporting central compensation of vestibular deficit (Betahistine) and balance recovery (vestibular rehabilitation).

In the majority of cases, the vestibular nerve is completely affected conferring the character of singularity to the attack, which will not be repeated. ■

References

1. Susan J. Herdman, Vestibular Rehabilitation - 3rd Edition, F.A. DAVIS COMPANY, Philadelphia, 2007.
2. Sekitani T, Imate Y, Noguchi T, et al. Vestibular neuritis: epidemiological survey by questionnaire in Japan. Acta Otolaryngol suppl 1993;503:9-12.
3. Brandt T. Vertigo: Its Multisensory Syndromes. London, England, Springer, 2004.
4. Herdman SJ, et al: Single treatment approaches to benign paroxysmal positional vertigo.
5. Otolaryngol Head Neck Surg 1992;107:399.
6. Schuknecht HF, Kitamura K. Second Louis H. Clerf Lecture. Vestibular neuritis. Ann Otol Rhinol Laryngol Suppl 1981;90:1-19.
7. Theil D, Arbusow V, Derfuss T, et al. Prevalence of HSV-1 LAT in human trigeminal, geniculate, and vestibular ganglia and its implication for cranial nerve syndromes. Brain Pathol 2001;1:408-13.
8. Arbusow V, Theil D, Strupp M, et al. HSV 1 not only in human vestibular ganglia but also in the vestibular labyrinth. Audiol Neurootol 2001; 6:259-62.
9. Theil D, Derfuss T, Paripovic I, et al. Latent herpesvirus infection in human trigeminal ganglia causes chronic immune response. Am J Pathol 2003; 163:2179-84.
10. Strupp M, Zingler VC, Arbusow V, et al. Methylprednisolone, valacyclovir, or the combination for the vestibular neuritis. N Engl J Med 2004; 351: 354-61.
11. Hallpike CS. The pathology and differential diagnosis of aural vertigo. In: Proc 4th Int Congr Otolaryngol. London, UK: British Medical Association, 1949:514.
12. Adour KK. Sprague MA. Hilsinger RL Jr. Vestibular vertigo. A form of polyneuritis? JAMA 1981; 246: 1564-7.
13. Strupp M, Arbusow V. Acute vestibulopathy. Curr Opin Neurol 2001; 14. 11-20.
14. McElhatton PR. (Nov-Dec 1994). "The effects of benzodiazepine use during pregnancy and lactation". Reprod Toxicol. 8 (6): 461-75.
15. Matok I, Gorodischer R, Koren G, Sheiner E, Wiznitzer A, Levy A. The safety of metoclopramide use in the first trimester of pregnancy. N Engl J Med 2009;360(24):2528-35.
16. Fetter M, Dichgans J. Vestibular neuritis spares the inferior division of the vestibular nerve. Brain 119:755-763, 1996.
17. Goebel JA, O'Mara W, Gianoli G. Anatomic considerations in vestibular neuritis. Otol Neurotol 22:512-518, 2001.