Pregnancy causes not only physiological changes in metabolism, hormone status, and autonomic nervous system but also impact of emotional stress. Sex hormones influence the central nervous system and provoke changes in hearing levels.

Sudden deafness is defined as the rapid (72 hours) decline of hearing (> 30 dB) in at least 3 contiguous frequencies without any identifiable cause. Sudden deafness is a cause of cochlear cell damage or cochlear nerve disturbances. Its etiology is sometimes difficult to determine and may include vascular, allergic, traumatic, inflammatory, degenerative (ie, multiple sclerosis) oncologic, or genetic conditions. It is rarely observed in pregnant women, especially in the beginning of the first trimester. It is postulated that the sex hormone changes may increase thrombogenic risk and interrupt cochlear microcirculation, causing the development of sudden deafness.

The effective treatment of sudden deafness in pregnant women is a challenging problem. In the literature there are some analysis of sudden deafness during pregnancy with complete recovery or persistent hearing loss after medication or spontaneous recovery after delivery. We did not find any case of repeatable, completely regressed sudden deafness in a woman during her first and second pregnancy.

**CASE REPORT**

A 25 year old, 27 week pregnant primipara was admitted to the Department of Otolaryngology with right-side sudden hearing loss and tinnitus lasting 2 days. There was no associated vertigo, viral infection, or history of trauma or medication intake. She was a nonsmoker. Her previous medical history was unremarkable, and the pregnancy was not complicated. Tonal audiometry revealed unilateral cochlear hypoacusis with hearing loss reached 60 dB in 1000 and 2000 Hz frequencies (Figure 1, A).

Tympanometries of the both ears were of type A. Stapedius reflex was present at the normal levels during stimulation of the healthy ear and was detected ipsilaterally during stimulation the affected ear with the tone of 500 Hz; absent for the 1000, 2000, 4000 Hz impulses; and present on the opposite side for 1000 and 2000 Hz stimulation at the level of 115 Hz. Audiological-evoked potentials of short latency (BAER) presented normal latencies of the waves I-V, normal range of latencies between the waves and the sides, and normal morphology and amplitudes. Evoked otoacoustic emissions demonstrated the lower energy of external cochlear cells answer to the frequency 4000Hz on the affected side (Figure 2, A).

The laboratory tests, morphology, blood glucose level, and the parameters of coagulation were normal. After 2 days of treatment using vasodilators, steroids, and B vitamins, the hearing loss disappeared, and audiometry was normal (Figure 1, B). The slight tinnitus that previously accompanied the hearing loss disappeared simultaneously with hypoacusis.

Two years after her first delivery, she was pregnant a second time. During the fourth week of pregnancy, she felt sudden hearing loss on the opposite side than during the first time. Tonal audiometry revealed unilateral cochlear hypoacusis with hearing loss reached 60 dB in 1000 and 2000 Hz frequencies and B, normal hearing after therapy.

**FIGURE 1**

Unilateral cochlear hypoacusis

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Omometry manifested the unilateral hypoacusis: the deepest hearing loss came to 60-40 dB in the frequencies 1500, 2000, 3000, and 4000 Hz. The other ear was completely normal (Figure 3, A). Tympanometry demonstrated type A on both sides, and the presence of stapedius reflex during stimulation of both the healthy and affected ear ipsilaterally and contralaterally the answer to 500 and 1000 Hz stimulus was present. Speech audiometry was normal with the discrimination distinguishing level for both sides was 5 and 10 dB, and the distinguishing level of 10 dB on the healthy side and 25 dB in the affected ear. The short increase sensitivity index (SISI test) achieved 0% on the healthy side and 15% on the affected side (500, 1000, 2000, 4000 Hz).

Laboratory tests were extended: apart from morphology, blood glucose level and coagulation factors (lipidogram, electrolytes, protein fraction pattern, Biernacki’s index, C-reactive protein) were analyzed, and the data were physiological. During electrystagmography (ENG; it records the vestibuloocular reflexes). The acute episode of vertigo is usually accompanied by spontaneous nystagmus, and the frequency, amplitude, and angular velocity can be measured by ENG and consequently observed for recovery. The whole ENG testing consists of various tasks: with visual, positional impulses and irrigating the ear with warm and cold water to search for the eventual labyrinth weakness. The cervical nystagmus when the head was turned to both the right and left side was observed.

No additional pathological signs (no spontaneous nystagmus, asymmetry of optokinetic nystagmus, disturbances of eye-tracking test, gaze nystagmus, asymmetry of labyrinth responses to caloric stimulations) were observed. The patient was not treated. She asked to be discharged from the hospital. The auditory checking that was performed after 5 months was completely normal (Figure 3, B). Both tonal and speech audiometry and the SISI test were normal.

**Comment**

The hormonal systems of women are unique because of the cyclical changes during pregnancy, the menstrual cycle, and menopause. The sex hormones may increase thrombogenic risk and interrupt cochlear microcirculation, leading to the development of sudden deafness. On the other hand, these changes could result on osmotic disturbances water and sodium retention. The shift of fluid osmolarity may affect the inner ear like in Menier’s disease.

Hormonal alternation also influences the cardiovascular system. Decreased erythrocyte deformability, increased plasma viscosity, and erythrocyte aggregation because of increased fibrinogen are observed. A thromboembolic episode involving the cochlear artery may be a result of a hypercoagulable state associated with pregnancy. Sennaroglu and Belgin reported the gradual decrease in the pure tone at 125, 250, and 500 Hz from first trimester to the third trimester of pregnancy. The speech audiometry and impedence audiometry in pregnancy were normal.

Tandon et al observed a higher threshold of wave V eliciting on the base of brainstem auditory responses (ABR) in pregnancy. In the ABR study, the increase of the absolute latency of wave V and the I-V interpeak latencies were recorded.

The therapy of sudden deafness during pregnancy is very controversial. In some cases the therapy consist of bed rest and carbogen therapy. On the other hand, when the microcirculation disturbances existed, the Dextran 40 seems to be the promoting agent. This therapy decreased blood viscosity and reduced cochlear hypoxia without side effects on the mother or the fetus. The steroid is not recommended for the treatment of sudden deafness in pregnancy because of side effects on the fetus: serious changes of the metabolic and endocrine balance of.

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**Figure 2**

Evoked otoacoustic emissions demonstrated the lower energy of external cochlear cell answer to the frequency 4000 Hz on the affected side.

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**Figure 3**

Deepest hearing loss

A, The deepest hearing loss to 60-40 dB in the frequencies 1500, 2000, 3000, and 4000 Hz and B, completely hearing recovery after 5 months.
various fetus organs can be taken into consideration.1

CONCLUSIONS
All complaints of hearing problems or tinnitus in a pregnant woman should be taken seriously. A complete audiol-logic diagnosis is mandatory in such a case. Recovery from a sudden hearing loss during pregnancy could be spontaneous after delivery or require medical treatment. The consideration of Dextran 40 infusion is necessary because of its safety. When a sudden hearing loss occurred during pregnancy, it could be a predictable factor of another episode during next pregnancies.

REFERENCES