Sudden (reversible) sensorineural hearing loss in pregnancy

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Abstract

Background  Sudden hearing loss directly associated with pregnancy or birth is a little known and rare occurrence. The temporary, unilateral, low-frequency sensorineural hearing loss in this case was reported after the birth of the patient’s first child, and again during the third trimester of her second pregnancy.

Aims  This paper discusses the different explanations as to why hearing losses occur due to physical changes within the body during pregnancy and birth. It is probable that this patient had significant anatomical asymmetry with one patent and one non-patent cochlear aqueduct, allowing increased pressure unilaterally. The mechanical restriction of the inner ear hair cells caused the hearing loss that returned to normal, when the pressure returned to normal.

Conclusions  Our case demonstrates that pregnancy can lead to hearing loss in two sequential pregnancies. Mechanisms are discussed in detail. Clinically it appears that the hearing loss and tinnitus associated with pregnancy can spontaneously recover.

Keywords  Pregnancy · Unilateral · Sensory neural · Hearing loss · Endolymphatic pressure

Introduction

Pregnancy can affect the hearing of inner ear in a number of ways, including infections such as rubella. However, sudden, temporary hearing loss is rare. We present a case where this happened to a patient during two consecutive pregnancies. The hearing returned to normal on each occasion.

History

This case study is of a woman who presented with hearing loss and tinnitus associated with pregnancy. She had experienced the same symptoms 4 years earlier when she gave birth to her first child.

A 42-year-old woman gave birth to her first child in 2001 when she was 38 years old. At that time, an ENT consultation was sought as the patient complained of right deafness for a week when she was postpartum 5/7. She also reported tinnitus but no otalgia, otorrhea or dizziness. Treatment included Carbogen gas 5 min in 1 h, thymoxamine 40 mg t.d. and prednisolone 20 mg q.d.

Daily audiograms were undertaken (Figs. 1, 2).

In view of her pregnancy, steroid treatment was stopped when hearing improved after 1 day. Subsequent hearing tests showed hearing had returned within the normal range.

In 2005, she was pregnant again, and at 38 weeks gestation reported noticing decreased hearing on her right side. Audiometry confirmed a low frequency hearing loss on that side (Fig. 3).

It was decided that use of a large quantity of steroids might be more of a hindrance than help, in view of the late period of gestation. But it was expected that since she had a similar episode during her previous pregnancy that the likelihood is that the hearing would return post-partum.
Fig. 1  Audio day 1: 8 August 01. Speech discrimination 97%

Fig. 2  Day 2 audio: 9 August 01

Fig. 3  Audio 9 June 04
A repeat audiogram was undertaken on 24 June 2004 within 12 h of delivery. The hearing had improved but as a precaution steroid treatment was started (Fig. 4). Hearing returned to normal by the 29 June 04, 5 days post-partum.

**Discussion**

The case study here demonstrates several different possible aetiologies for hearing loss associated with pregnancy and birth. The hearing loss in this case was reported after the birth of her first pregnancy, and also during the third trimester of her second pregnancy.

Sudden hearing loss directly associated with pregnancy is a little known and rare occurrence. Hearing losses associated with epidural procedures for pain relief during birth have been reported on occasions but are still not commonplace. There seems to be a number of different explanations as to why these hearing losses occur. The first explanation would be the hearing loss of mechanical nature due to increased pressure in the endolymph, caused by increased pressure in CSF [1–3]. The second explanation would be due to hormonal changes affecting level of oedema [3–6]. Finally another explanation could be of a vascular interruption [7]; this could be due to physical changes within the body during pregnancy and birth. Each of these three explanations will be examined in turn.

A case report [4] was published last year of a 34-year-old pregnant woman suffering from a significant bilateral sensorineural hearing loss and tinnitus in the third trimester of the pregnancy, in this case, there was spontaneous recovery 3 weeks after delivery. The possible reasons given were either electrolyte imbalance with an increase in extracellular fluid volume leading to the formation of perineural oedema, or the influence of hormones, particularly that of oestrogen. Other possibilities given were viral infection and immunosupression. Each of these possible explanations will be explored here, but starting first with the alternative explanation of a mechanical changes causing the hearing loss.

**Lumbar puncture/pressure changes explanation**

It is known that loss of cerebrospinal fluid due to a lumbar puncture can cause headaches and low tone hearing losses, which are readily rectified by giving an epidural blood patch. There is one reported case, in which a lumbar puncture gave rise to a unilateral hearing loss [8]. A study by Walsted et al. [1] demonstrated that there are small but significant changes in low tone hearing following spinal anaesthesia. They suggested that the explanation of the hearing loss could be a biological mechanism similar to endolymphatic hydrops, resulting from perilymphatic hypotension due to “loss of liquor during and after the spinal anaesthesia”. However, Finegold et al. [9] tested the hypothesis that women undergoing elective caesarean delivery with a subarachnoid block (SAB) suggesting that they may experience hearing loss due to lumbar puncture and cerebrospinal fluid leakage causing hypoacusis. They could find no difference in the hearing levels in the period immediately before and 2 days after the caesarean. Michel et al. [10] explain this apparent contradiction when they suggest that hearing loss following lumbar puncture is a rare complication, which would appear to occur in patients with a wholly or partially patent cochlear aqueduct, which allows loss of perilymphatic fluid into the cerebrospinal space. They cite nine non-pregnant patients who experienced hearing loss, three of whom were left with residual hearing loss.

The link between the cochlear and CSF fluids is important in the “pathogenesis of auditory dysfunction in several patient groups” [11]. Hearing losses have been
associated with the pressure changes in the CSF that occurred due to the injection of the epidural anaesthesia [2, 12]. In pregnancy, the epidural space is less compliant because of the increased epidural venous blood flow. Anaesthetists alter their treatment for pain relief in pregnant women, as epidural block is higher in pregnancy compared to non-pregnant age-matched control. When a patient reports headaches and the low-tone hearing loss with an epidural procedure, but in the absence of evidence of a lumbar puncture, rather than a CSF leak causing the symptoms, it could simply be the effect of the changes of pressure in the CSF with the epidural injection.

In pregnancy, there are several possible causes of changes in CSF pressure including the hormone and oedema explanations discussed later. If the cochlear aqueduct is wholly or partially patent then the inner ear is more liable to perilymphatic pressure changes during pregnancy compared to their non-pregnant age-matched controls due to the epidural space being less compliant. Consequently, the changes in CSF pressure could presumably have a similar effect to that of epidural injection with a raised CSF pressure producing a hearing loss of mechanical nature due to increased pressure in the endolymph. So that in both cases, when the CSF pressure returns to normal levels the apparent sensorineural hearing loss resolves with hearing returning to normal levels.

Hormone changes explanation/oedema

Excessive salt and water retention are common in pregnancy, the electrolyte imbalance causes an increase in the extracellular fluid volume, which leads to the formation of perineural oedema. The higher incidence of carpal tunnel syndrome which has been described in pregnancy [3, 13] appears to be of similar pathology. In late luteal phase of the menstrual cycle, some women experience oedema, there is evidence to show that this may have a direct influence on the pattern of endolymphatic hydrops or Meniere’s disease [5]. Raised perilymphatic pressure [14] is often associated with variation in the hormone levels of women with menstrual irregularities, pregnancy and the menopause and the presumed patency of the cochlear aqueduct. Some authors also attribute neurological impairments during pregnancy, such as cranial nerve palsy/ facial paralysis, to electrolyte balance and hormone fluctuations [4, 6, 7, 15].

Hormone fluctuations alter the maintenance of the chemical composition of both perilymph and endolymph in the inner ear, and the ion transport processes between them. The link between the perilymph and the endolymph is maintained by hydrostatic pressure via the cochlear aqueduct. Therefore, the patency of the cochlear aqueduct is key, to whether the effect on the hearing will be to a greater or lesser extent, with the changes in composition and pressure of the CSF during pregnancy. Statically the cochlear aqueduct grows in length with children growing, and then becomes less patent with the ageing process. Since most pregnancies are with young women (<45 years old), the cochlear aqueduct will usually be patent, whether in pregnancy the patency of the cochlear aqueduct will usually be patent, whether in pregnancy the patency of the cochlear aqueduct changes in the same way that the epidural space becomes less compliant is unknown. But it is interesting to note that all except one [16] of the few case studies published regarding hearing loss during pregnancy are in cases of older women (>35 years old).

Mild low tone hearing differences have been detected in pregnant women compared to their post-partum hearing tests and non-pregnant controls, but these hearing differences were so small that statistically the hearing remained within the normal range for hearing [3]. There appears to be little research into the relation between pregnancy and the pattern of endolymphatic hydrops, but there is one case study in which a woman with Meniere’s disease has been reported as having increased attacks during early pregnancy, directly related to the serum osmolality [13].

An alternative explanation may be that, in pregnant woman the autoimmune system is suppresses and this in itself may have an effect on the way in which the endolymphatic pressure and subsequently the hearing may be affected. Some researchers suggest that allergy and immunologic factors play a role in Meniere’s disease, citing that the inner ear may be the target, directly or indirectly, of an allergic reaction [17, 18]. If this is true then it is possible that the pregnant woman is at risk of experiencing symptoms of endolymphatic hydrops, when her autoimmune system might otherwise counteract an allergic reaction.

Vascular explanation

Sudden sensory-neural hearing loss during an otherwise uncomplicated pregnancy, may be linked to increased activation of both blood coagulation and fibrinolysis (‘hypercoagulable state’) which occurs during normal pregnancy. This state may lead to vascular occlusion in the microcirculation of the inner ear by microemboli.

A literature review also suggests the influence of hormones, particularly that of oestrogen. In this regard a study by Ben David [7] and others looks at the relationship between facial paralysis and oestrogen levels, where he tests the theory that oestrogen causes ischaemia of the vasa-nervosa and this leads to affecting of the cranial nerves, however, he was not able to prove such changes in pregnancy, but it still may be responsible for a higher incidence of Bell’s palsy during pregnancy. Pregnant
women have a predisposition to Bell’s palsy compared to non-pregnant women, which again is often unilateral.

Unilateral explanation

Having described the possible causes for a sensori-neural hearing loss, the difficulty of explaining a temporary unilateral hearing loss remains. In this case an explanation for there being a unilateral hearing loss during pregnancy might be due to the altered anatomy and physiology of the epidural space associated with pregnancy. Whilst in the examples given in literature are generally binaural hearing losses, there is the possibility of having a unilateral sensori-neural hearing loss. It may be that there is a non-symmetrical anatomy that causes it to be unilateral. Unilateral high epidural blocks have been reported in pregnancy resulting in Horner’s syndrome or facial numbness, despite otherwise low level of epidural block [19].

Conclusion

There are many changes that occur in pregnancy that could be attributed to causing a bilateral sensory-neural hearing loss including vascular, autoimmune, oedema, and CSF pressure related. If the cause were vascular in nature, for the length of time that the effect was present, some residual hearing loss would be expected with death of hair cells due to the poor blood supply. The suppression of the autoimmune system in pregnancy may itself have an effect on the way in which the endolymphatic pressure and subsequently the hearing may be affected.

Alternative explanations include oedema, electrolyte balance, and hormone fluctuations; these have previously been attributed to raised perilymphatic pressure. This raised perilymphatic pressure being caused either: by altering the maintenance of the chemical composition of both perilymph and endolymph in the inner ear, and the ion transport processes between them. Or the presumed patency of the cochlear aqueduct could allow transmission of pressure from the increased pressure in the CSF, to the perilymphatic fluid. This increase in the perilymphatic fluid pressure consequently mimicked Meniere’s-type symptoms, of a low tone hearing loss with tinnitus. This raised pressure and associated symptoms recovered spontaneously as the CSF levels returned to normal since the link between the perilymph and the endolymph is maintained by hydrostatic pressure via the cochlear aqueduct. But each of these possible explanations fails to explain a unilateral hearing loss. In this case it would appear that there was a significant anatomical asymmetry which allowed the pressure in one inner ear to lead to hearing loss and tinnitus being in one rather than both ears. It would appear more likely that this patient had one patent and one non-patent cochlear aqueduct and that the most likely cause is a mechanical restriction of the hair cells in the inner ear, due to the endolymphatic pressure being raised. Thus, when the pressure returned to normal the hearing likewise returned to normal levels.

Clinically it appears that the hearing loss and tinnitus associated with pregnancy can spontaneously recover. Other possible aetiologies need to be excluded, in the instance of a sudden sensorineural hearing loss during pregnancy. In terms of treatment, it leaves the ENT medics having to decide whether they should administer steroid drugs for acute hearing loss, as it may recover spontaneously.

More research is needed to establish if there are changes in the cochlear aqueduct patency during pregnancy. Also research is needed to examine the true levels of hearing change in women before and during and after pregnancy to establish whether it is usual to have some mild hearing loss or not.

References