

Epidural Hematoma After Cochlear Implantation In A 2.5-Year-Old Boy

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Objective: Report a case of an epidural hematoma after cochlear implantation in a 2.5-year-old boy, the diagnostic and therapeutical emergency management, as well as the post-interventional course and rehabilitation of the child.

Study Design: Retrospective case review.

Patient: Two and a half-year-old boy, suffering from early onset, profound sensorineural hearing loss had been diagnosed at an age of 1.5 years, which had been more severe on the right side initially, but had progressed to bilateral deafness.

Intervention and Complication: Cochlear implantation on the left side, followed up by an extensive epidural hematoma, causing intracranial compression with a midline shift of 15 mm to the right side. This initial complication was followed up by

arterial infarction not only of the middle but also the posterior as well as the anterior cerebral artery, most likely caused by incarceration through the tentorium.

Results: After immediate neurosurgical intervention and intensive care treatment including low-dose anticoagulation and high doses of corticosteroids, the child recuperated completely within 3 weeks without any remaining neurologic deficits.

Conclusion: In cochlear implantation, especially if placement of the implant housing is associated with considerable bone work, epidural hematoma has to be considered as an extremely rare, but life-threatening, complication.

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Cochlear implantations have become a routinely performed and very successful surgical intervention in both children and adults, performed so far in more than 10,000 patients worldwide. Generally, this procedure is associated with rather low complication rates, regardless of the individually chosen cochlear implant device. Different reports have detailed possible medical, surgical or audiologic complications (1–6).

We are reporting to our knowledge the first case of an epidural hematoma after cochlear implantation in a 2.5-year-old boy, resulting in a critical and life threatening situation. The diagnostic and therapeutical emergency management as well as the postinterventional course and rehabilitation of the child are discussed.

CASE REPORT

A cochlear implantation was performed in a 2.5-year-old boy suffering from early onset profound sensorineural hearing loss had been diagnosed at an age of 1.5 years, which had been more severe on the right side

initially but had progressed to bilateral deafness. An initial brainstem evoked response audiometry had shown responses at 100 dB on the right and between 100 dB and 120 dB on the left side. However, repeated evaluation, including brainstem evoked response audiometry as well as otoacoustic emissions throughout the 12 pre-operative months displayed better results on the left side. Cardiologic workup had revealed a ventricular right-to-left shunt. The boy appeared healthy otherwise without any evidence of autoimmune or syndromal disease. Pre-operative imaging showed normal cochlear and tympanic structures bilaterally and symmetrical pneumatization of both mastoids. The operation was planned, and informed consent was achieved from both parents for a cochlear implant on the left side.

The cochlear implantation was performed using the Med EL Combi 40+ device (Innsbruck, Austria). This implant requires rather extensive bone work to create an implant bed posterior to the mastoid cavity for firm and proper placement of the implant body. Dummies of the device are routinely used to exactly adapt the size of the bony cavity to the actual size of the implant. Using this type of implant, we routinely drill the bone down to a thin layer covering the dura at the edges of the implant bed and leave a thin shell of bone in the center. In the

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presented case, the surgical procedure was uneventful, and the electrode array was completely inserted into the cochlea through the cochleostomy by way of the facial recess. Intraoperative blood loss was within normal range, and minor bleeding was observed from stitches during wound closure; however, there was no evidence of arterial bleeding at any time, and the wound was dry and normal upon termination of the procedure. Recurrent adenoids after a previous operation elsewhere had been removed at the beginning of the operation.

General anesthesia was well tolerated and uneventful. No adverse events or remarkable observations were reported during the immediate postoperative period, and the child was transferred back to the pediatric unit of our department. The child was awake with a regular and stable cardiovascular as well as neurologic status and nurses as well as the parents did not observe any abnormalities during the immediate postoperative period.

Four hours postoperatively, the child developed progressive somnolence and appeared to have intermittent minor bleedings from his nose. The immediate laboratory workup revealed a blood sugar of 380 mg/100 ml, combined with a metabolic acidosis. Because of the observed bleeding from the nose, the nasopharynx as well as the retroauricular incision were reinspected in the operating room. No significant bleeding was detected in the nasopharynx, and the retroauricular wound appeared normal without evidence of a subcutaneous hematoma, so the wound was not opened at this time. A repeated blood analysis displayed a hemoglobin level of 8.1 mg/dl, indicating a significant decrease from the preoperative value 12.5 mg/dl, which could not be explained. Except for the reduced vigilance, the child was still free of severe neurologic symptoms with bilateral adequate pupillary reflexes to light.

The child was now transferred to the pediatric intensive care unit (ICU) and a cranial computed tomography (CT) scan was performed. This CT scan revealed an extensive epidural hematoma compressing the left hemisphere with a midline shift of 15 mm to the right side (Fig. 1). Neurosurgical revision and draining of the epidural hematoma was performed immediately. The arterial bleeding, originating from a dural branch of the middle meningeal artery, was located centrally underneath the implanted processor, right at the edge of the bony cover, and was stopped by bipolar coagulation. Most likely, this vessel had been eroded by pressure caused by the processor in the absence of a complete bony shell on top of the dura. Questionable unrecognized intraoperative trauma caused by the performed drilling is a factor that might have led to some initial weakening of vascular structures of the dura.

After this revision, the child was transferred back to the pediatric ICU for further close monitoring. The repeated hemoglobin analysis now revealed a value of 6.1 mg%. Packed blood cells were given during the after hours, followed up by an increase of the hemoglobin back to 11.5 mg%. The boy was extubated in the early

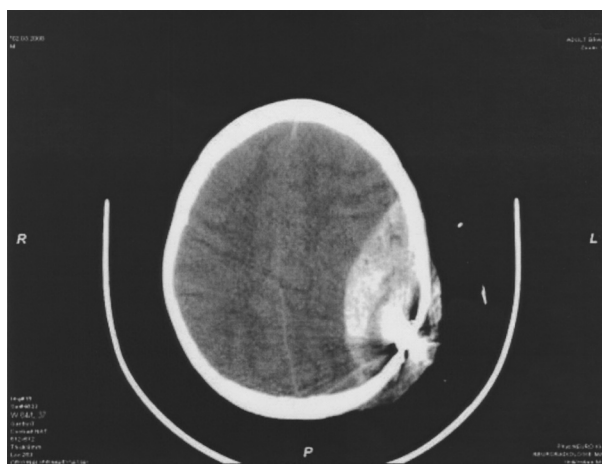


FIG. 1. Cranial computed tomography scan displaying a large epidural hematoma on the left side with a 15 mm midline shift.

morning of the next day. Throughout that day, he was still tired and drowsy with inadequate reactions to stimuli provided by nursing staff or his parents.

A repeated cranial CT scan was performed after 36 hours, which again unveiled a complication of more severity than expected. A large area of the left temporal and temporooccipital lobe displayed radiologic hypodensity, matching a venous cerebral infarction, possibly with consecutive hemorrhage. After first analysis of these scans indicated signs of a possible thrombosis of the superior sagittal sinus, the question of a high-dose anticoagulation had to be discussed, including the risk of severe life-threatening massive hemorrhage into the infarcted area. To further clarify the current developments, the child was now evaluated by sophisticated magnetic resonance imaging sequences focusing on the venous and arterial blood flow. These enabled us, without having to take the additional risk of an angiogram, to exclude the differential diagnosis of a sinus thrombosis. Two important findings lead to the final diagnosis of an arterial infarction, most likely caused by incarceration through the tentorium, instead of the suspected venous thrombosis: the infarction included areas supplied by not only the middle but also the posterior as well as the anterior cerebral artery. No sinuses, venous, or arterial vessels displayed any abnormalities (Fig. 2). These findings could only be explained by a transient interruption of the arterial blood supply during the compression caused by the epidural hematoma with herniation and subsequent incarceration through the tentorium.

After this diagnostic procedure, the anticoagulation was kept at a low dose, and the child was without any sedating medication. High doses of corticosteroids were applied intravenously in an effort to minimize and soon reduce intracranial edema and swelling. During the proceeding 48 hours, the boy slowly began to recuperate and regained adequate vigilance. By then we were able to reestablish a current neurologic status. The expected right hemiparesis presented as a weakness of the right

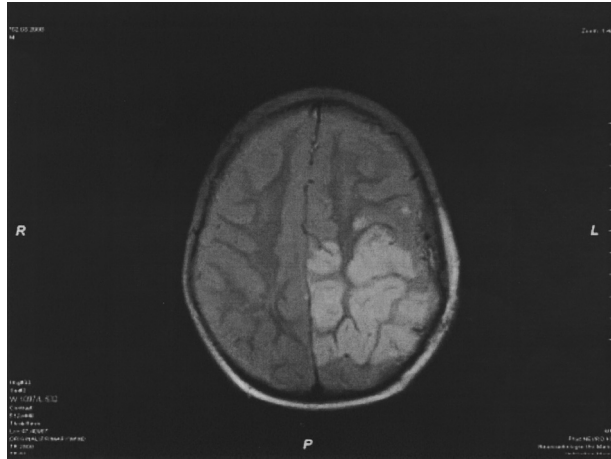


FIG. 2. Magnetic resonance imaging 36 hours postoperatively showing a large cerebral infarction after arterial ischemia caused by transtentorial herniation caused by the epidural hematoma.

arm and reduced reflexes in the muscles of the left leg. Furthermore, the child developed a centrally caused diabetes insipidus, which was controlled medically. A possible hemianopia was difficult to rule out completely because of the deafness of the boy and the absence of speech. However, a pediatric perimetric analysis performed in the department of ophthalmology at our institution showed no evidence of hemianopia 2 weeks postoperatively.

Throughout the next 10 days, supported by intensive physical therapy, the neurologic deficits completely resolved with minor motor weakness and the child being able to run and play in a normal manner. The diabetes insipidus also turned out to be of a transient nature, and medical therapy could be discontinued. Thirteen days postoperatively, the child was dismissed from the ICU and transferred to a pediatric ward before being dismissed from our institution at day 16 postoperative.

The cochlear implant, which had to be removed during the emergency procedure of draining the epidural hematoma, was checked for technical integrity by the manufacturer and showed to be intact. A revision was

scheduled 4 weeks postoperatively to evaluate the status of the cochleostomy and the intracochlear lumen. In case of a free cochlear lumen, a reimplantation on the identical side was intended; however, in case of obliteration by scarring, a cochlear implantation on the right side would be suitable after the audiologic and radiologic findings.

CONCLUSIONS

This is to our knowledge the first report of an epidural hematoma occurring after a cochlear implantation. This report suggests that this extremely rare, life-threatening complication needs to be considered as a differential diagnosis in case of sudden inadequate vigilance in the postoperative period, possibly accompanied by metabolic dysregulation and unexplained decrease of hemoglobin. Immediate revision of the wound should be recommended in this situation on the basis of the clinical findings even without waiting for imaging studies in an effort to minimize possible cerebral compression and ischemia.

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