

Life-threatening posterior fossa cyst induced by pseudomeningocele after operation for acoustic neuroma

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Abstract

Background: Pseudomeningocele is the term used to describe fluid accumulation due to the leakage of cerebrospinal fluid into the surrounding soft tissue. It may cause complications such as cosmetic deformities, chronic meningitis, and/or impingement on vital structures resulting in neurological deficits; nevertheless, life-threatening posterior fossa cyst formation is a rare event.

Case Description: We report a case of posterior fossa cyst formation induced by pseudomeningocele with brain stem compression leading to coma with pupillary dilation. These symptoms occurred after an operation for left acoustic neuroma. After emergent decompression and dural repair, the patient recovered well without experiencing any further neurological deficits.

Conclusion: We discuss the clinical features, possible pathophysiological mechanisms, and treatment options for pseudomeningocele. Although most cases of pseudomeningocele follow a benign course and need only conservative treatment, the potential attendant complications, such as an enlarged cyst, may still have fatal consequences. We believe that it is beneficial to take an aggressive attitude toward this condition and to consider the possibility of surgical interventions more seriously.

Key Words: Acoustic neuroma, cyst, pseudomeningocele

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INTRODUCTION

Pseudomeningocele is the term used to describe cerebrospinal fluid (CSF) accumulation after a meningeal tear over an extradural cavity.^[6] The incidence of postoperative pseudomeningocele is around 4.5% with neurotologic procedures. It may cause complications such as cosmetic deformities, chronic meningitis, and/or neural tissue compression with neurological deficits. Few patients with pseudomeningocele need to undergo an operative procedure to achieve resolution of symptoms; instead, most are advised nonoperative management, such as pressure dressing, bed rest, and lumbar spinal drainage.

In addition, fatal complications can be caused by a cyst induced by pseudomeningocele, although this is extremely rare. Although the incidence of pseudomeningocele is not high, and most cases can be resolved without surgery, cases involving the area of cerebellopontine angle (CP angle) and posterior fossa deserve closer examination, since complications in these regions are more life-threatening than those involving the supratentorium area.

CASE REPORT

A 69-year-old female with a history of medically controlled hypertension was referred to our clinic for a CP angle mass

with obstructive hydrocephalus. The patient presented with unsteady gait, which she had experienced for about one year. In addition, dizziness, urinary incontinence, left hearing loss, and mild facial palsy were noted. Brain magnetic resonance imaging (MRI) showed a large left CP angle mass; based on this finding, we suspected this to be acoustic neuroma [Figure 1a]. We performed a retrosigmoid craniotomy for removal of the tumor, and the pathology revealed a schwannoma composed of both Antoni A and B areas. The wound showed good healing 2 weeks after the operation; however, a left postauricular cystic mass was noted. Brain computer tomography (CT) showed fluid collection in the region of the left parietal-occipital scalp [Figure 1b]. Pseudomeningocele was diagnosed by physical examination, patient's history, and radiological findings. She received needle aspiration and conservative treatment as well as regular follow-up visits to our outpatient department. The protruding mass seemed to improve with treatment; however, one month after the operation, the patient complained of disturbed consciousness and was admitted with an initial Glasgow coma scale of E2M5V1. Brain CT showed a left cerebellar cyst, which communicated with a pseudomeningocele that severely compressed the brain stem and had induced obstructive hydrocephalus [Figure 2a]. We performed emergency surgery to achieve decompression of the brain stem and repair the dura defect. Postoperatively, the patient regained her clarity of consciousness, and a follow-up brain MRI [Figure 2b] revealed a smaller cerebellar cyst and the absence of pseudomeningocele.

DISCUSSION

Pseudomeningocele refers to fluid accumulation in the brain due to CSF leakage from surrounding soft tissue.^[4] The pathophysiology of pseudomeningocele is controversial. Some scientists believe that pseudomeningocele is caused by problems in the subarachnoid space, such as intradural defects, while others believe it may be related to defective CSF

absorption, secondary to conditions such as subarachnoid scarring and hydrocephalus.^[3] The usual treatment of pseudomeningocele includes compression dressing, believed to minimize the potential space available and to allow absorption of CSF. In addition, patients are restricted to bed rest and asked to keep their heads elevated in an effort to minimize the rise in CSF pressure. Regular follow-up care is required and in the case of a recurrence, sterile needle aspiration or lumbar drainage should be performed.^[6] Although literature reports efficacy of lumbar drainage for CSF leakage to be about 90%,^[8] acute posterior fossa syndrome may occur following lumbar drainage due to rapid movement of CSF fluid into the cerebellar parenchyma. Pseudomeningocele may cause complication such as cosmetic deformity, chronic meningitis, and impingement of vital structures with neurological deficits when it enlarged.^[5] Surgery can be performed to prevent development of an enlarged pseudomeningocele and its attendant complications. The range of onset time for pseudomeningocele has been described as 5–115 days postoperatively, with a mean of 18.2 days.^[6] The timing of treatment is important; if left untreated, an enlarging pseudomeningocele may cause irreversible damage.

When pseudomeningocele is associated with a posterior fossa cyst, the CSF fluid exchanged among the subarachnoid space, pseudomeningocele, posterior fossa cyst, and the dura defect may compose a check-valve that can enlarge the cyst and cause mass effect to vital structures. A cyst is a fluid-filled epithelium-lined cavity, whereas a pseudocyst is a fluid-filled cavity with no epithelial lining.^[9] Clinically, “cysts” can represent both cysts and pseudocysts. The mechanisms of cyst formation are related to multiple factors that differ depending on the clinical cause. When arteriovenous malformations were managed using gamma knife surgery (GKS), cyst formation was reported to be the most common complication.^[9] The possible causes of cyst formations include increased permeability of injured blood vessels

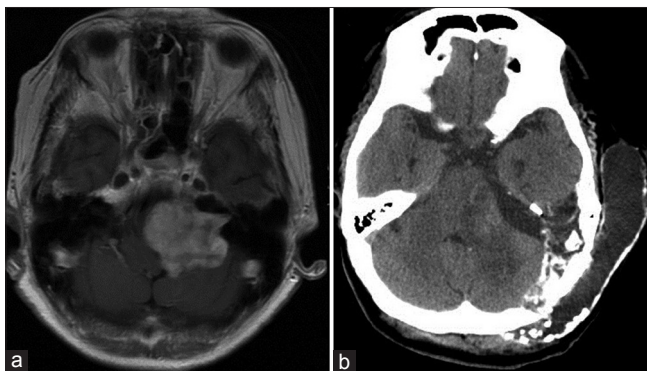


Figure 1: (a) Contrast axial T1-weighted brain MRI showed a large left CP angle acoustic neuroma with brain stem compression. (b) The axial brain CT showed left periauricular pseudomeningocele 2 weeks after the operation

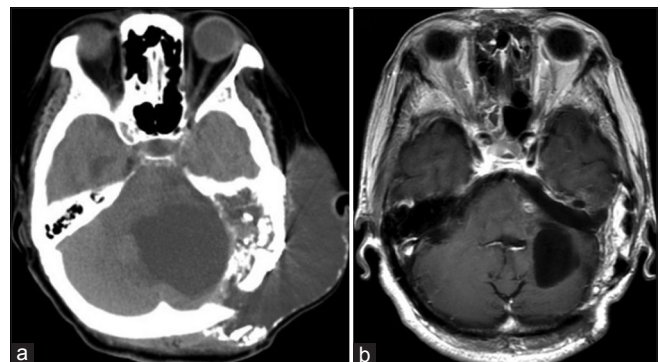


Figure 2: (a) One month later after operation, the axial brain CT showed a large left cerebellar cyst that communicated with a pseudomeningocele and severely compressed the brain stem. (b) The axial T1-weighted enhanced image of brain MRI showed a smaller cerebellar cyst after pseudomeningocele repair

of the nidus, liquefaction of coagulation necrosis, and a break in the blood–brain barrier. In the cases of cystic vestibular schwannoma with GKS, cyst formation may be related to the loss of the peritumoral arachnoidal plane and adjacent scarring due to radiosurgery.^[1] Moreover, proteinaceous tumor debris can cause a disruption of local CSF flow, resulting in an enlarged cyst.^[1] Breakdown of the blood–brain barrier and fluid-occupying lesions left by hematoma or brain tissue loss due to encephalomalacia following resection are reportedly the leading factors in the process of cyst formation.^[7]

In our case, a possible explanation for the patient's cyst formation is that CSF may drain out from a weakened dura with a one-way valve mechanism leading to pseudomeningocele formation. Enlarged pseudomeningocele may compress the normal CSF pathway of posterior fossa. After lumbar drainage or other treatment efforts, CSF flow changes and moves rapidly to move CSF from the pseudomeningocele to the intracerebellar parenchyma via a weak area of the dura, resulting in formation of an enlarged cyst with a mass effect to vital brain structures.^[2] The postoperative adjacent scarring, coagulation necrosis, brain tissue loss due to encephalomalacia following resection, and loss of the peritumoral arachnoidal plane are all possible leading factors of cyst formation in our case with the complication of pseudomeningocele. Since the best approach is prevention of cysts, we believe that we should have initially administered a more aggressive treatment such as surgical dural repair, rather than a conservative treatment, given the possibly fatal consequences of this condition.

In conclusion, pseudomeningocele following neurotologic procedures cannot be considered a rare event. Although most cases of pseudomeningocele follow a benign course and need only conservative treatment, the potential attendant complications, such as an enlarged cyst, may still have fatal consequences. We believe that it is beneficial to take an aggressive attitude toward this condition and to consider the possibility of surgical interventions more seriously.

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