

Case Report

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Delayed Cerebral Abscess as a Shunt Complication and Endoscopic Removal of the Ventricular Catheter and Abscess

Shunt infections are a common complication of ventriculoperitoneal (VP) shunts, but the formation of a brain abscess related to a shunt system is very rare. A 44-year-old woman had a VP shunt inserted for hydrocephalus secondary to a subarachnoid hemorrhage. She suffered an episode of meningitis and sepsis 8 months after the shunt operation. After recovering from the meningitis, she complained of a loss of cognitive function. An enhancing mass was found in the frontal lobe, around the frontal horn of the lateral ventricle, and the ventricular catheter was embedded inside the mass. The ventricular catheter and cerebral abscess were removed using neuroendoscopy. We present an interesting case of a shunt-related brain abscess which illustrates the usefulness of neuroendoscopy.

KEY WORDS : Brain Abscess · Ventriculoperitoneal shunt · Endoscopy.

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INTRODUCTION

A ventriculoperitoneal (VP) shunt is a standard procedure for symptomatic hydrocephalus. It is very effective for relieving the symptoms, although many complications are associated with shunt operations. Postoperative shunt infections occur in 2-17% of cases in most neurosurgical units worldwide^{5,9,11,15}. Despite the advent of modern neurosurgical techniques, new antibiotics, and imaging techniques, an infection after a VP shunt is still a serious complication^{5,13}. The clinical presentation of a shunt infection includes acute bacterial meningitis and the signs or symptoms of shunt malfunction or obstruction.

A delayed intracerebral abscess after a VP shunt is a very rare complication that has been reported in only a few cases^{1,2,6,10,14}. We report a patient who presented with a delayed shunt-related intracerebral abscess one year after placement of a VP shunt and who was managed successfully with an endoscopic procedure.

CASE REPORT

A 44-year-old woman suffered insomnia, general weakness, and decreased cognitive function that developed one month before admission. She had a complicated history. She had suffered from a spontaneous subarachnoid hemorrhage due to aneurysmal rupture and underwent aneurysmal neck clipping at another hospital in November 2003. Hydrocephalus was developed at 3 months postoperatively. She had a VP shunt placed in another hospital in March 2004. She visited our hospital for the first time in December 2004 because she was feeling drowsy and had a fever (38°C). Contrast-enhanced brain computed tomography (CT) did not show a space-occupying lesion or ventriculitis (Fig. 1). The culture of cerebrospinal fluid (CSF) withdrawn from the lumbar cistern on admission grew *Escherichia coli*, which was susceptible to first- and third-generation cephalosporins, aminoglycosides, and quinolones. The white blood cell count in the CSF was 558 (35% of polymorphonuclear leukocytes and 53% of lymphocytes), with a glucose level of 1 mg/dl and a protein level of 229 mg/dl. The white blood cell count in the blood was 13,500/μl with C-reactive protein of 33 mg/dl. Even though pathological organisms were not detected in both CSF and blood, which were sampled at the second hospital day, bacterial meningitis was strongly suspected, however, tuberculous meningitis could not be

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excluded. Intravenous cefotaxime and vancomycin and standard oral antituberculous medications were administered. Her consciousness was recovered fully and her fever subsided after the antibiotic treatment. The intravenous antibiotics were given for 2 weeks, and the antituberculous drugs were continued.

She complained of insomnia 6 months after her first admission to our hospital. Fever was not noted and laboratory findings of the blood such as white blood cell, erythrocyte sediment rate, and C-reactive protein were normal. Brain CT showed an abnormal enhancing lesion

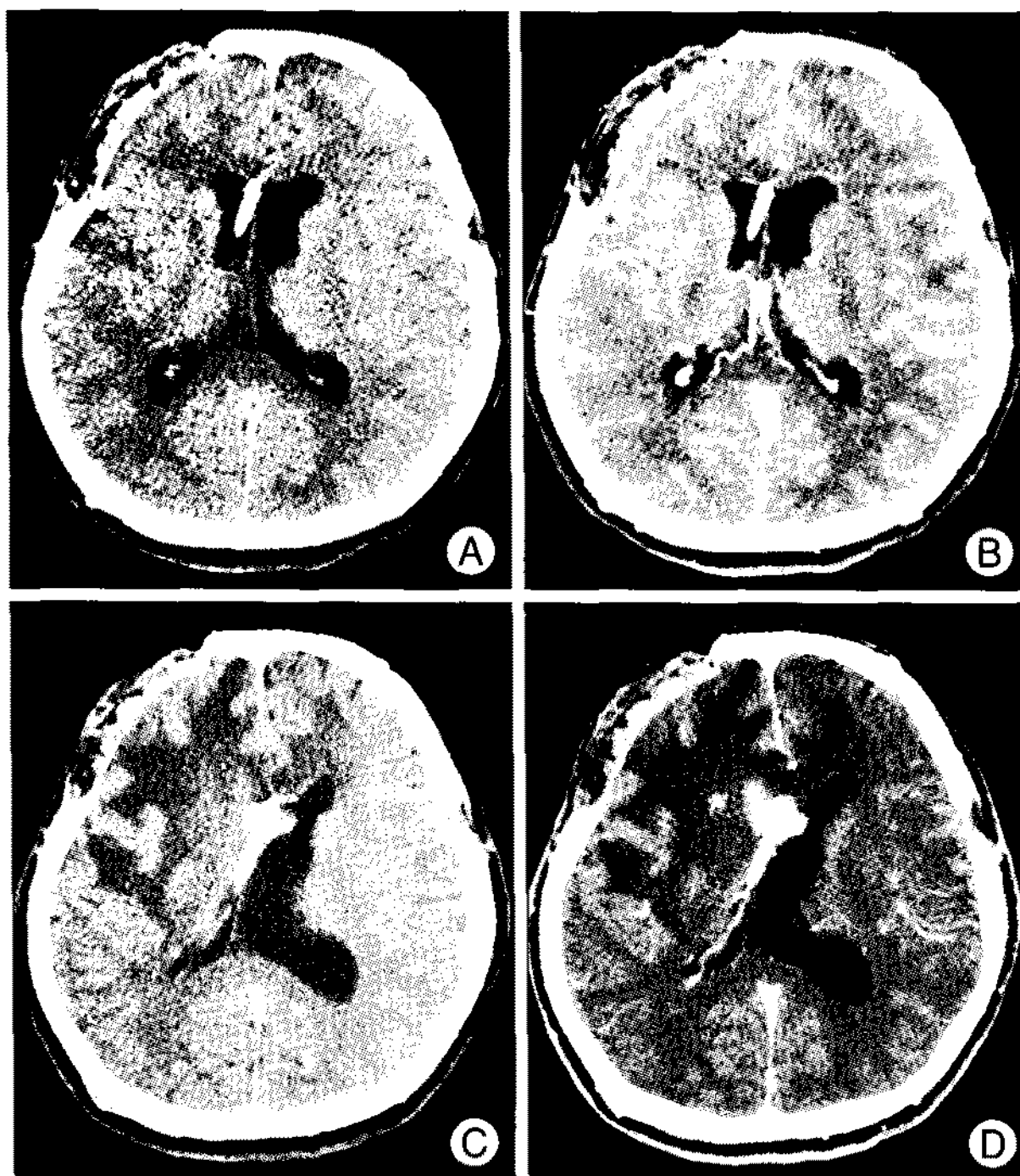


Fig. 1. Brain computed tomography during the periods of meningitis (A and B) and abscess (C and D). The ventricular catheter is located in the right lateral ventricle, embedded in the frontal lobe beyond the frontal horn of the lateral ventricle (A). There initially is no enhancing lesion (B). Six months later, an enhancing mass and edema are seen around the proximal tip of the ventricular catheter (C and D).

with perilesional edema in the deep white matter around the frontal horn of the right lateral ventricle (Fig. 1). The ventricular catheter tip was embedded in this lesion. She was still taking antituberculous medication at that time. We considered the lesion to be a catheter-related infection. We decided to perform neuroendoscopic surgery to remove the ventricular catheter and enhancing mass.

A neuroendoscope was introduced into the right lateral ventricle, which contained the reservoir for her shunt apparatus, via a parietal burr hole. The tip of the ventricular catheter had penetrated and was stuck in the brain parenchyma, as seen when the endoscope arrived at the frontal horn of the right ventricle (Fig. 2). When we tried to remove the catheter it would not move because it was adhered to the ventricular wall. A radiofrequency coagulator and forceps were introduced through the working channel of the endoscope, and the ventricular catheter was dissected away from the parenchymal lesion. After the ventricular catheter had been removed, the brain lesion appeared gray and hard, similar to granulation tissue. Complete hemostasis of the brain lesion and ventricular wall was accomplished with the coagulator. For CSF drainage, an external catheter was placed in the lateral ventricle.

Non-specific inflammatory cells and reactive gliosis were found in the pathological specimen (Fig. 2). The cultures of the ventricular CSF, brain mass, and abdominal catheter showed the growth of *Pseudomonas aeruginosa*, which was susceptible to first- and third-generation cephalosporins and aminoglycosides. *Escherichia coli* was also identified in the ventricular CSF. Therefore, intravenous cefepime (2 gm every 12 h) was administered.

The drainage catheter was withdrawn on postoperative day 3, and the antibiotics were continued. She became drowsy on postoperative day 10. Ventriculomegaly was seen on CT, and external ventricular drainage was performed through the right Kocher's point (Fig. 3). The

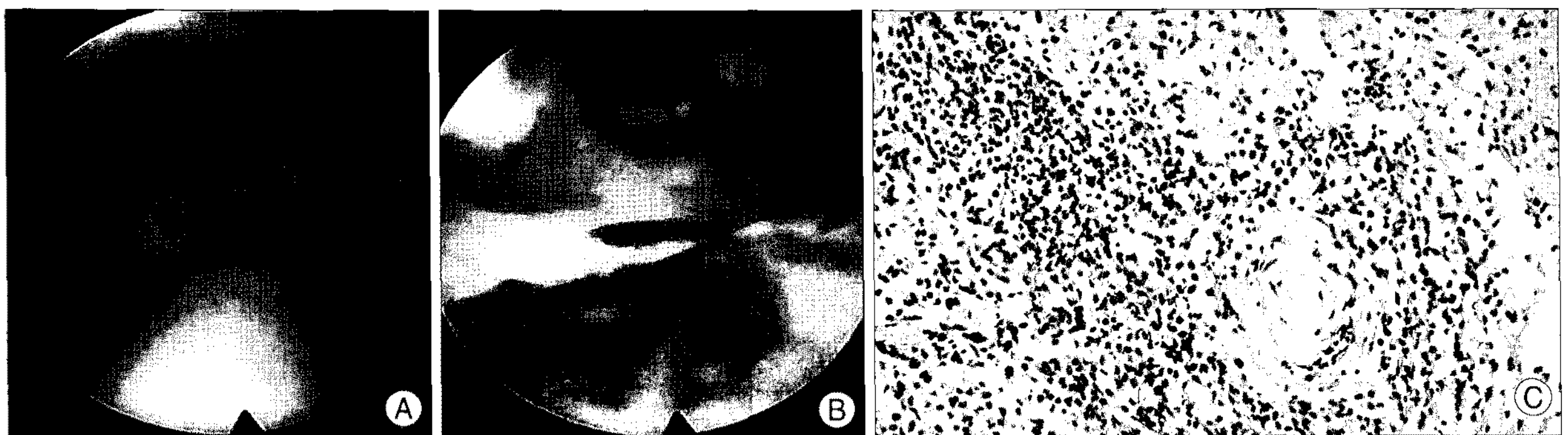


Fig. 2. Endoscopic view and photomicrograph of the mass. The catheter tip is embedded in the ependyma and brain parenchyma (A). Dissection and hemostasis with the radiofrequency coagulator are being performed (B). The histopathological examination (C) of the lesion reveals granulation tissue with non-specific inflammation and reactive gliosis (H & E stain, $\times 100$).

CSF study revealed signs of infection. The white blood cell count was 82/ml and *Pseudomonas aeruginosa* was seen in the culture of the CSF. Therefore, intrathecal gentamicin (8 mg/day) was administered for 7 days. The ventricular CSF was sent to the laboratory every day. On the 10th day of external ventricular drainage, the CSF was clean, with no evidence of infection. We sent CSF to the laboratory for the next 3 days to confirm that it was sterile. Subsequently, a VP shunt was placed in the left parieto-occipital point. Intravenous cefepime was given for 44 days. Her consciousness and cognitive function recovered fully, and the subsequent clinical course has been uneventful, with no shunt-related complications during one year postoperatively (Fig. 3).

DISCUSSION

Although shunt-related infections are the second most common complication of shunts, cerebral abscesses related to a shunt are very rare^{1,2,6,10,14}. In our case, the cerebral abscess was developed around the distal tip of the ventricular catheter. The abscess and ventricular catheter were removed successfully using endoscopic surgery.

Postoperative shunt infections occur in 2-17% of cases in most neurosurgical units worldwide^{5,9,11,15}. Although myriad infections such as meningitis and ventriculitis have been reported after a VP shunt, cerebral abscesses rarely develop. The common conditions underlying bacterial brain abscesses are chronic pulmonary disease and chronic otitis media⁸. However, the reported causes of cerebral abscesses in VP shunts include ascending infections of Gram-negative enteric organisms⁶ or infections colonizing the external surface of the ventricular catheter^{7,10,14}. The reported cerebral abscesses related to a shunt were located in the proximal part of the ventricular catheter, which lies in the brain parenchyma between the skull and ventricle. In our case, the abscess was developed around the distal tip of the ventricular catheter. The pathogen that caused the meningitis and ventriculitis was inoculated into the brain parenchyma via the embedded ventricular catheter. Using an excessively long catheter that can penetrate the remote brain parenchyma after passing along the ventricle may not be a good practice, because it may result in a delayed cerebral abscess, as in our case, and in difficulty removing the shunt system.

A non-functioning intraventricular shunt catheter has been shown by several authors that act as a site of bacterial colonization, leading to a progressive chronic infection and abscess formation^{1,7,14}. They have advocated the removal of a non-functioning shunt system because of its latent

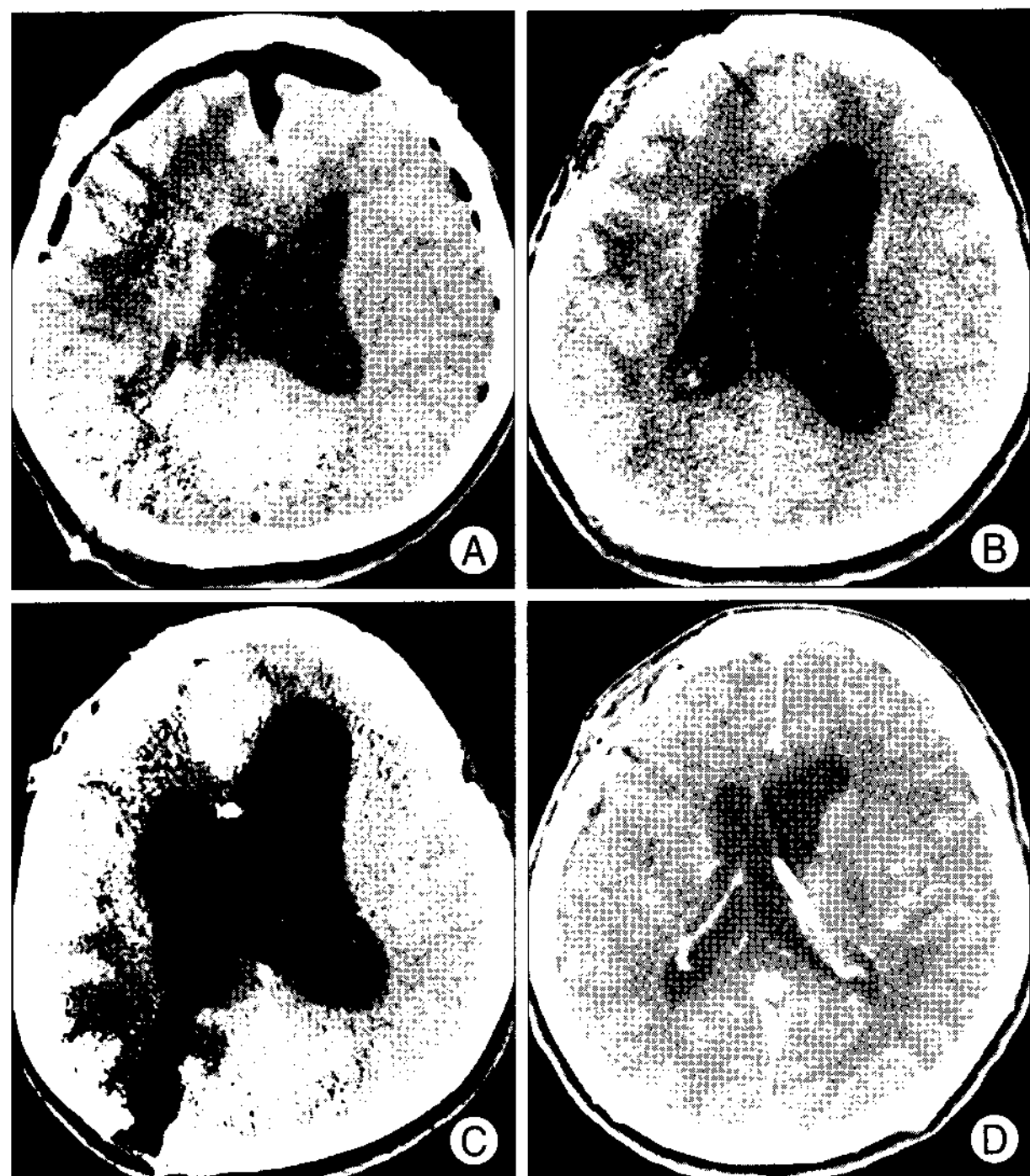


Fig. 3. Postoperative brain computed tomography (CT). Immediate brain CT shows no massive intraventricular hemorrhage (A). A catheter for external ventricular drainage (EVD) is placed in the right ventricle. After removing the EVD on the 10th postoperative day, ventriculomegaly is seen (B). The EVD was placed at the right Kocher's point (C), and intravenous and intrathecal antibiotics were administered. Brain CT at one year after the ventriculoperitoneal shunt at the left parieto-occipital point (D). There is no evidence of an abnormal enhancing lesion or hydrocephalus.

role in serious complications such as cerebral abscesses and ventriculitis. The pathogens isolated in our case were *P. aeruginosa* and *E. coli*. Therefore, we postulated that the previous meningitis laid the groundwork for the delayed brain abscess and that the pathogen was an ascending enteric pathogen, as there was no external wound around the shunt system.

Many complications, including intracerebral or intraventricular hemorrhage, can occur when removing a ventricular catheter, especially when it adheres firmly to the choroid plexus or ventricular wall³. Therefore, a non-functioning intraventricular catheter is sometimes left in place to avoid the serious complications that can occur when the shunt system is removed. An insulated suction tube attached to an electrocoagulation unit has been introduced for use in resecting and coagulating adhesions on a retained ventricular catheter⁴. The use of an endoscope for the revision of an adherent ventricular catheter reduces the risk of bleeding in these cases¹². In addition, the ingrowth of the choroid plexus, ependyma, or subependymal elements into the catheter lumen can be observed and divided under direct visualization. We

obtained a satisfactory surgical field in the ventricle through the ventricular catheter burr hole and controlled the bleeding from the inflamed parenchyma, while successfully removing the infected catheter and abscess. The procedure was accomplished with minimal brain injury.

CONCLUSION

A cerebral abscess can develop as a rare complication of a ventriculoperitoneal shunt. The ventricular catheter and cerebral abscess can be removed safely using neuroendoscopic surgery.

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