

## Case Report

# Cerebral Venous Thrombosis Complicated by Hemorrhagic Infarction Secondary to Ventriculoperitoneal Shunting

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While a delayed intracerebral hemorrhage at the site of a ventricular catheter has occasionally been reported in literature, a delayed hemorrhage caused by venous infarction secondary to ventriculoperitoneal shunting has not been previously reported. In the present case, a 68-year-old woman underwent ventriculoperitoneal shunting through a frontal burr hole, and developed a hemorrhagic transformation of venous infarction on the second postoperative day. This massive venous infarction was caused by bipolar coagulation and occlusion of a large paramedian cortical vein in association with atresia of the rostral superior sagittal sinus. Thus, to eliminate the risk of postoperative venous infarction, technical precautions to avoid damaging surface vessels in a burr hole are required under loupe magnification in ventriculoperitoneal shunting.

**KEY WORDS :** Hydrocephalus · Intracerebral hemorrhage · Postoperative complication · Venous infarction · Ventriculoperitoneal shunt.

## INTRODUCTION

Ventriculoperitoneal shunting is one of the most frequently performed procedure in neurosurgery, and a ventricular puncture through the cerebral cortex is an essential component of this procedure. The technical goal of a ventriculostomy is a safe placement of a ventricular catheter, without any development of intracerebral or intraventricular hemorrhage and with the tip located anterior to the foramen of Monro in the frontal horn of the lateral ventricle. Thus, any small cortical vessel in the burr hole is commonly coagulated by using a bipolar electrode<sup>13)</sup>. However, the iatrogenic occlusion of a surface vein of a certain caliber can cause regional brain edema and hemorrhagic venous infarction. The authors report on a case of massive hemorrhagic venous infarction following ventriculoperitoneal shunting via Kocher's point.

## CASE REPORT

A 68-year-old woman who underwent a successful right pterional craniotomy to clip a ruptured middle cerebral artery (MCA) aneurysm developed normal pressure hydrocephalus a few months later, presenting with gait disturbance and urinary incontinence. The patient then underwent ventriculoperitoneal shunting using a right frontal burr hole placed 1 cm anterior to the coronal suture in the midpupillary line. The patient did very well initially, and a computerized tomography (CT) performed on the first postoperative day showed appropriate placement of the ventricular catheter and no intracranial bleeding (Fig. 1A). However, on the second postoperative day, the patient developed left hemiplegia, along with deterioration in her mentality (Glasgow coma scale score 12). A CT scan demonstrated a massive intracerebral hemorrhage (ICH) around the ventricular catheter and an associated intraventricular hemorrhage (IVH) (Fig. 1B). The ICH was patchy and confluent within a subcortical hypodensity with a mass effect, suggesting a hemorrhagic transformation of venous infarction. The patient was then referred to our institution. There was no history of tendency of bleeding or hypercoagulability. Hematological tests, including the platelet count, prothrombin time, and partial throm-

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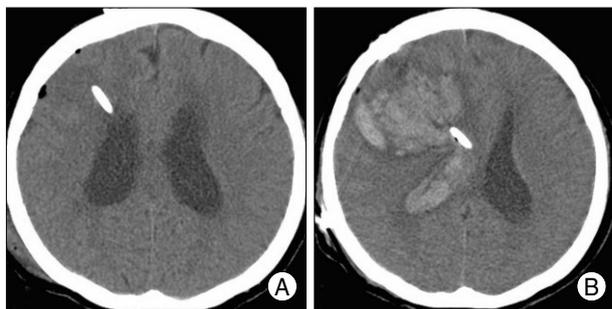
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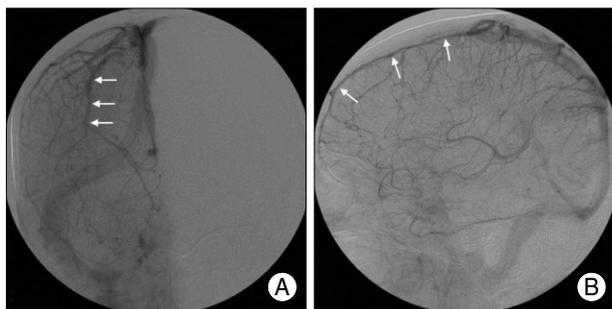
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boplastin time, were all within normal limits.

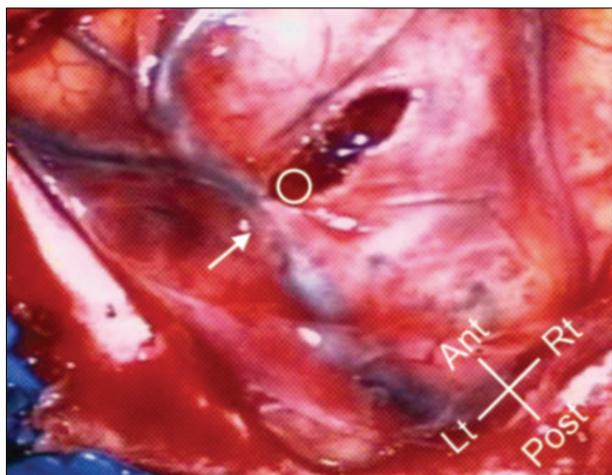
When reviewing the patient's previous catheter angiogram, no abnormalities were found, except the MCA aneurysm. However, the venous phase revealed aplasia of the anterior half of the superior sagittal sinus (SSS), which in turn was



**Fig. 1.** CT scans following ventriculoperitoneal shunting. A : On the first postoperative day, appropriate placement of the ventricular catheter and no intracranial bleeding are ascertained. B : On the second postoperative day, a massive intracerebral hemorrhage around the ventricular catheter and an associated intraventricular hemorrhage are revealed.



**Fig. 2.** Venous phase of angiograms. A : Anteroposterior view demonstrating a large paramedian cortical vein (arrows) coursing along the midpupillary line. B : Lateral view demonstrating a large paramedian cortical vein (arrows) joining the superior sagittal sinus behind the coronal suture. The anterior half of the superior sagittal sinus is aplastic.



**Fig. 3.** Intraoperative photograph. A thrombosed paramedian cortical vein was shrunk (arrow) due to the bipolar coagulation adjacent to the puncture site (circle) of the ventricular catheter. A cortical incision to remove the intracerebral hematoma was performed extending from the puncture site (circle).

drained by long paramedian cortical veins (Fig. 2). In particular, the large paramedian cortical vein on the right side coursed along the midpupillary line. The vein involved an extensive draining territory in the frontal lobe and had poor venous collateralization.

A craniotomy revealed the paramedian cortical vein beside the cortical puncture of the ventricular catheter. The vein was shrunk and pale adjacent to the cortical puncture and seemed to have been affected by the bipolar cautery (Fig. 3A). The absence of venous flow was confirmed using an intraoperative Doppler, and a vascular incision revealed intravenous thrombosis. Using a cortical incision starting at the cortical puncture site of the ventricular catheter, the ICH and IVH were removed. The ventricular catheter of the shunt was then re-inserted into the lateral ventricle.

A CT scan on the first postoperative day showed relief of the mass effect after removing the ICH and IVH. However, the territory of the venous infarction extended slightly further. At two months after the craniotomy, the patient recovered her mentality, yet still remained hemiplegic.

## DISCUSSION

Coronal (Kocher's point) and occipital sites are most frequently chosen for a ventricular puncture when performing a shunting procedure. A ventricular puncture at the coronal site is commonly performed through a burr hole placed just anterior to the coronal suture in the midpupillary line over the nondominant hemisphere<sup>7,9,11</sup>. After a dural incision in a cruciate form, the brain surface is inspected and any small cortical vessels are coagulated using a bipolar electrode before introduction of the ventricular catheter<sup>13</sup>. Some neurosurgeons also perform a small dura opening with coagulation using a monopolar cautery to ensure a tight dural catheter seal and reduce the risk of cerebrospinal fluid leakage around the catheter<sup>14</sup>. Yet, the present case emphasizes the importance of preserving large cortical veins and requirement of careful inspection of the brain surface in the burr hole under loupe magnification.

The SSS courses in the midline from the foramen cecum posteriorly to the torcular herophili. Each hemisphere has 8 to 12 superficial cortical veins entering into the SSS, and the cortical veins are quite variable in both number and configuration<sup>10</sup>. In the anterior frontal region, most of the superior cortical veins are small, making the sacrifice of such veins not normally hazardous. However, if the rostral SSS is atretic or hypoplastic, substitute parasagittal venous channels develop and drain most of the frontal lobe<sup>5,18</sup>. Thus, an acute occlusion of such a parasagittal vein can cause a rise in the venous pressure and tissue pressure within its drainage territory,

leading to a decrease in the blood supply and disruption of the blood-brain barrier, which eventually results in regional brain edema and venous infarction with or without a hemorrhagic transformation<sup>2,3,12,16</sup>.

Venous infarction develops ill-defined hypodense lesions that do not conform to the arterial distribution on a CT scan. Hemorrhages, mainly in the white matter, range from petechial to large, and surrounding cerebral edema is prominent. Clinical features include frequent headaches and seizures, in addition to focal deficits and disorders of consciousness<sup>2,3</sup>. The clinical course of venous infarction is usually slow and indolent until hemorrhagic transformation. The present case also showed delayed occurrence of remarkable neurological deficits two days after the venous occlusion.

A delayed ICH at the site of the ventricular catheter has occasionally been reported in literature<sup>1,4,6,8,15,17</sup>. The presumed mechanisms of the delayed ICH include delayed erosion of a cerebral vessel by the ventricular catheter, a hemorrhage from an occult vascular malformation, a coexisting bleeding disorder, and head trauma occurring after the shunt placement. However, venous infarction secondary to coagulation of a surface vein should also be included as a cause of a delayed ICH.

## CONCLUSION

Technical precautions to avoid damaging surface vessels are required under loupe magnification in ventriculoperitoneal shunting. Inadvertent coagulation of a cortical vein of a certain caliber can cause significant venous infarction.

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