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Case Report

Endoscopic third ventriculostomy for noncommunicating hydrocephalus by vertebrobasilar dolichoectasia: A case report

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ABSTRACT

Background: Vertebrobasilar dolichoectasia (VBD) is a vasculopathy characterized by the elongation, widening, and tortuosity of a cerebral artery. Rarely, hydrocephalus results when the extended basilar artery impairs communication of the cerebral ventricle and cerebrospinal fluid dynamics. We experienced such a case when a patient underwent endoscopic third ventriculostomy (ETV) for noncommunicating hydrocephalus with VBD.

Case Description: A 54-year-old man presented with cognitive dysfunction and was diagnosed with VBD by magnetic resonance imaging (MRI). Seven years later, he exhibited subacute impaired consciousness due to acute noncommunicating hydrocephalus, undergoing external ventricular drainage (EVD) that improved consciousness. After EVD removal, the noncommunicating hydrocephalus did not recur; however, 7 months later, subacute consciousness impairment due to noncommunicating hydrocephalus was again observed. MRI showed a significant dilation of both lateral ventricles and ballooning of the third ventricle while the right posterior cerebral artery shifted slightly posteriorly. The patient underwent ETV and clinical symptoms improved. One year after the treatment, MRI observed a patent ETV fenestration and no deleterious changes in clinical symptoms were observed.

Conclusion: ETV can be an effective treatment for the noncommunicating hydrocephalus with VBD when performed with preoperative assessment of vascular anatomy and attention to vascular injury.

Keywords: Countercurrent pulsation, Endoscopic third ventriculostomy, Non-communicating hydrocephalus, Vertebrobasilar dolichoectasia

INTRODUCTION

Dolichoectasia is a cerebral vasculopathy with deformation, mainly in the vertebral and basilar arteries (vertebrobasilar dolichoectasia; VBD), with prevalence estimates ranging between 0.2% and 4.4%. However, among patients presenting with stroke, the prevalence ranges between 2.6% and 17.1%.[14] This deformability of vessels is explained by arterial wall remodeling,[7] resulting in an elongated, dilated, and tortuous artery that causes variable neurological symptoms. While cerebral

infarction and compression are common,[2] hydrocephalus is a relatively rare complication in about 5% of patients with VBD.[7,12,14] There are some reports of ventriculoperitoneal (VP) shunting for noncommunicating hydrocephalus with VBD but, recently, endoscopic third ventriculostomy (ETV) is more commonly used.[1,4-6] However, there are no reports of ETV for noncommunicating hydrocephalus with VBD. Here, we report a case of noncommunicating hydrocephalus with VBD treated by ETV with an emphasis on precautions and endoscopic features.

CASE PRESENTATION

A 54-year-old man presented with cognitive dysfunction and was diagnosed with VBD at another hospital. Magnetic resonance imaging (MRI) showed that the basilar artery was elongated, protruding upward, and compressing the third ventricle floor [Figure 1a-d]. The patient underwent medical treatment for hypertension (nifedipine and imidapril hydrochloride), dyslipidemia (fluvastatin sodium), and dementia (donepezil hydrochloride). At home, his blood pressure was controlled at about 130–140/90–100 mmHg. Although he experienced no changes in clinical symptoms, 5 years later, MRI showed asymptomatic left internal carotid artery occlusion and further VBD elongation [Figure 1e-h] while SPECT showed no decrease in cerebral blood flow. One year later, he suffered a sudden loss of consciousness and MRI revealed dilatation of both lateral ventricles and the third ventricle. External ventricular drainage (EVD) was performed for acute hydrocephalus and consciousness status was immediately improved with no dilatation of lateral ventricles observed after EVD removal. As there was no recurrence of impaired consciousness, he was treated conservatively and, 7 months later, MRI showed no dilatation of the ventricles. However, 4 days after MRI, he exhibited subacute consciousness impairment again and was transported to our hospital.

MRI showed a significant dilation of both lateral ventricles, periventricular hyperintensity, and ballooning of the third ventricle [Figure 1i-l]. The right posterior cerebral artery had slightly shifted posteriorly in comparison to a condition without lateral ventricle dilatation. This was determined to be acute hydrocephalus and urgent EVD was performed, again improving consciousness status. Although the initial noncommunicating hydrocephalus improved with EVD alone, it recurred within a short period of time and we, therefore, planned a noncommunicating hydrocephalus repair. Since the basilar artery slightly deviated to the left with a certain distance between the basilar artery and the third ventricle floor, we decided that it was safe to perform ETV.

An insertion port was placed on the right frontal lobe toward the anterior horn of the right ventricle and a flexible endoscope was inserted [Figure 2a]. The posterior part of the third ventricle floor was stretched upward due to the extension of the right posterior cerebral artery [Figure 2b] and the third ventricle floor was seen to be ballooning while the tuber cinereum was thinning. Arterial pulsation was confirmed through the third ventricle floor [Figure 2c]. The tuber cinereum was fenestrated at the right posterior of the infundibular recess, avoiding the artery [Figure 2d]. The endoscope was inserted into the basal cistern through the fenestration at the tuber cinereum and the basilar artery adventitia was observed to be white from fibrotic change [Figure 2e]. Cerebrospinal fluid flow through the fenestration was confirmed.

On postoperative day 7, MRI showed a decrease in dilatation of both lateral ventricles and the periventricular hyperintensity had also improved [Figure 3a]. In addition, the cerebrospinal flow void at the fenestration on the third ventricle floor was confirmed and the right posterior cerebral artery had returned to the anterior position [Figure 3b]. The neurological status improved and the patient was discharged home on postoperative day 14. One year after the treatment, MRI showed a patent ETV fenestration, with no dilatation of the lateral ventricles. There were no changes in clinical symptoms.

DISCUSSION

Pathologically, dolichoectasia is a result of internal elastic lamina disruption from matrix metalloproteinase dysfunction and the migration of smooth muscle cells that induce breakdown and remodeling of the tunica media. As a result, affected arterial walls are prone to dilatation.[2,11] Noncommunicating hydrocephalus is a rare complication of VBD, with a 3.3% estimated 5-year risk of progressive hydrocephalus that occurs when the basilar artery bifurcation is well above the sellar diaphragms and impinges on the third ventricle.[2,14] Two mechanisms have been reported for noncommunicating hydrocephalus with VBD.[1,6,9] One is the true obstruction of CSF flow due to compression of the third ventricle or cerebral aqueduct while the other is impairment of CSF communication due to the combination of increased CSF pulse pressure and impairment of outward CSF flow by countercurrent pulsations of the basilar artery.[13] The previous reports showed that bilateral or unilateral VP shunt placement has been performed for the hydrocephalus with VBD.[1,3,6,9,10,15,16] However, even though ETV is more effective than shunt surgery for noncommunicating hydrocephalus because of fewer complications and lower mortality, there are no reports about ETV for hydrocephalus with VBD.[4,8]

We experienced a case of noncommunicating hydrocephalus with VBD and performed ETV with a good long-term outcome. The patient initially had stenosis in the third ventricle due to PCA compression caused by the upward

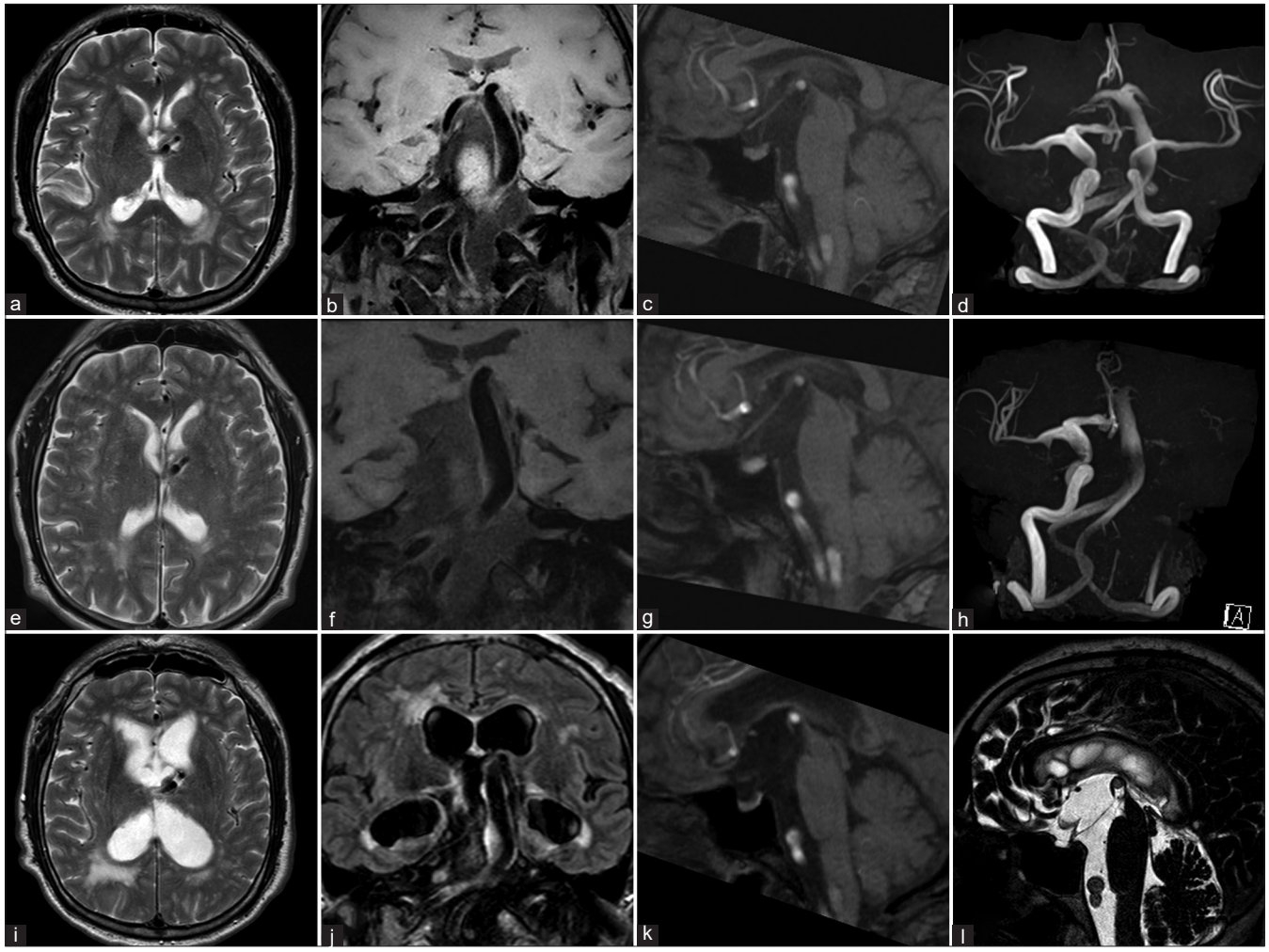

Figure 1: (a-d) Initial MRI (a: T2-weighted image, b: T1-weighted volume isotropic turbo spin-echo acquisition, c: sagittal reconstruction image of MRA, and d: MRA). The basilar artery was dilated and the top of the artery extended upward (b and d). The right posterior cerebral artery extended the floor of the third ventricle upward but did not touch the fornix (c). The enlargement of the ventricles was not observed (a). (e-h) MRI 5 years later (the second examination); (e) T2-weighted image, (f) T1-weighted volume isotropic turbo spin-echo acquisition, (g: sagittal reconstruction image of MRA, h: MRA). The left internal carotid artery was asymptomatically occluded (h). The basilar artery and right posterior cerebral artery extended further upward and the floor of the third ventricle touched the fornix (f and g). The enlargement of the ventricles was not observed (e). (i-l) MRI 1 year and 7 months later (the third examination, just before ETV); (i) T2-weighted image, (j) fluid-attenuated inversion recovery image, (k) sagittal reconstruction image of MRA, and (l) T2 drive image. The bilateral ventricle and the third ventricle were enlarged (I and j). The right posterior cerebral artery extended further upward and touched the fornix while deformities of the midbrain and pons were also observed (k). The floor of the third ventricle was observed to be ballooning (l).

extension of the BA. Moreover, the temporary increase in CSF pulse pressure caused by VBD may have dilated the third ventricle and deviated the PCA posteriorly, resulting in third ventricle obstruction. EVD reduced the size of the third ventricle, released the PCA deviation, and restored CSF flow, which may have temporarily improved the hydrocephalus thought to be caused by the impairment of CSF communication due to countercurrent pulsation rather than simple CSF flow obstruction. We chose ETV in the present case for the following reasons: first, ETV is the first choice for noncommunicating hydrocephalus. Second, considering the pathogenesis of hydrocephalus, we thought that ETV with spontaneous CSF drainage would have fewer complications than VP shunt placement with forced CSF drainage.[4,8] The most critical point during ETV is the vascular injury of the basilar artery during fenestration and it is necessary to confirm the position of the basilar artery and the fenestration on preoperative images. A previous report mentioned a case where the pulsation of the basilar artery at the third ventricle floor was observed endoscopically and ETV was not performed due to the possibility of vascular injury.[1] In contrast, we observed that the basilar artery was

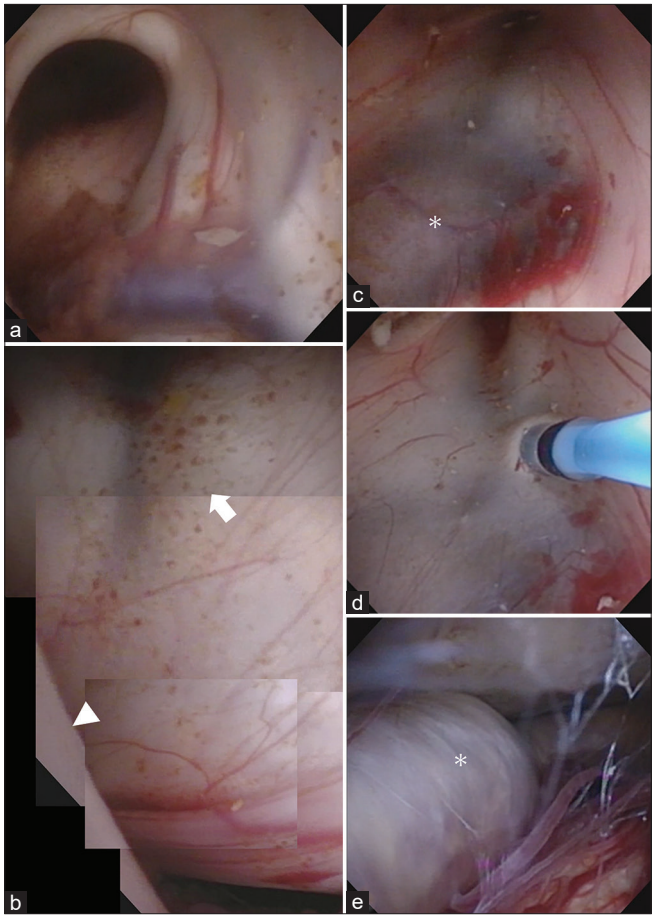

Figure 2: Images of the endoscopic third ventricle ventriculostomy. (a) The foramen of Monro was observed from the right ventricle, which was enlarged. (b) Consolidated images of endoscopic findings in the third ventricle. The floor of the third ventricle extended to the fornix due to the upward extension of the right posterior cerebral artery (white arrowhead). The mamillary body was stretched (white arrow). (c) The basilar artery (asterisk) was confirmed through the floor of the third ventricle. (d) The floor of the third ventricle was fenestrated to avoid injuring the basilar artery. (e) The vascular wall of the basilar artery (asterisk) had white sclerotic changes.

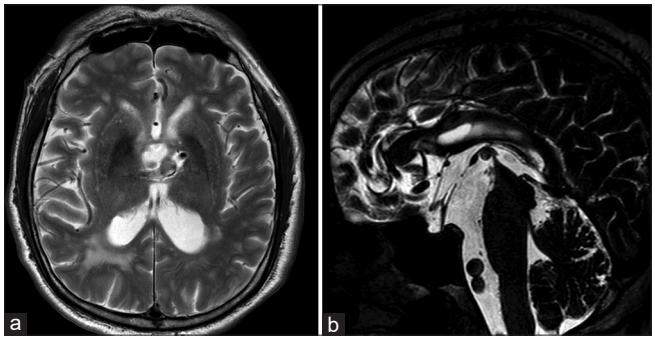

Figure 3: Postoperative MRI showed that the ventricles were shrinking (a: T2-weighted image) and the floor of the third ventricle was fenestrated (b: T2 DRIVE image).

transparent from the third ventricle floor, which could be safely opened while avoiding the basilar artery. Although there are no reports of mid-to-long-term outcomes in VP shunts, the present case showed no changes in imaging or clinical symptoms 1 year after the treatment.

We assert that hemodynamic change is a key trigger of the dolichoectatic process since the basilar artery began to extend upward after the ICA was asymptomatically occluded, although annual MRI images after the diagnosis of VBD showed no changes for 5 years. The increase in hemodynamic stress on the posterior circulation due to ICA occlusion was considered to have caused the upward extension of the basilar artery.[11] VBD may, therefore, progress when hemodynamic changes, such as vessel occlusions, occur and should be closely observed.

CONCLUSION

We treated a case of noncommunicating hydrocephalus with VBD by ETV. ETV for noncommunicating hydrocephalus with VBD can be an effective treatment when performed with a preoperative assessment of vascular anatomy and attention to vascular injury.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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