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Middle Meningeal Artery Embolization for Pre-existing Chronic Subdural Hematoma Enlarged After Ventriculoperitoneal Shunt Placement: A Case Report

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Abstract

Chronic subdural hematoma (CSDH) may develop or enlarge following ventriculoperitoneal (VP) shunt placement because of cerebrospinal fluid (CSF) overdrainage. Managing CSDH in patients requiring CSF diversion for hydrocephalus is challenging, as restricting CSF drainage may compromise hydrocephalus treatment. We report a case of a 78-year-old man with post-traumatic hydrocephalus and pre-existing left CSDH who underwent VP shunt placement. The initial programmable valve pressure was set at 12 cmH_2O. On the third postoperative day, computed tomography (CT) revealed enlargement of the CSDH. Middle meningeal artery embolization (MMAE) using 20% n-butyl cyanoacrylate was performed on the fifth postoperative day. Following embolization, no further hematoma enlargement was observed. The programmable valve pressure was subsequently reduced to 9 cmH_2O and then 5 cmH_2O to maintain adequate CSF diversion. Two weeks after MMAE, CT demonstrated reductions in both ventricular size and hematoma volume, accompanied by improvement in gait. The patient was discharged home, and further hematoma regression was observed approximately one month after embolization without additional intervention. This case suggests that MMAE may be a useful adjunctive treatment for managing pre-existing CSDH in patients requiring VP shunt placement for hydrocephalus.

Categories: Neurosurgery
Keywords: chronic subdural hematoma, csf overdrainage, hydrocephalus, middle meningeal artery embolization, ventriculoperitoneal shunt

Introduction

Ventriculoperitoneal (VP) shunting is an established and widely used treatment for symptomatic hydrocephalus of various etiologies, including idiopathic normal pressure hydrocephalus (iNPH) and secondary hydrocephalus [1,2]. However, cerebrospinal fluid (CSF) overdrainage following VP shunting may result in intracranial hypotension and contribute to the development or enlargement of subdural fluid collections, including subdural hygromas and hematomas [1-4]. The proposed mechanisms include downward displacement of the brain, stretching of bridging veins, and expansion of the subdural space [3,4]. When subdural collections develop after VP shunt placement, conventional management strategies include increasing the valve opening pressure to reduce CSF drainage. In more severe cases, temporary shunt ligation or surgical evacuation of the hematoma may be required [2,5]. However, these approaches may compromise adequate CSF diversion and potentially lead to recurrence or worsening of hydrocephalus. Therefore, the management of patients with pre-existing chronic subdural hematoma (CSDH) who require VP shunting for hydrocephalus presents a particular therapeutic challenge, as adequate CSF diversion must be balanced against the risk of hematoma enlargement. Middle meningeal artery embolization (MMAE) was initially introduced as a treatment for recurrent or refractory CSDH [6] and has subsequently gained increasing attention as a therapeutic option for both primary and recurrent CSDH [7]. Recent studies have suggested that MMAE may improve treatment outcomes in patients with CSDH, particularly when used as an adjunct to standard treatment [8-10]. However, its role in patients with pre-existing CSDH who require VP shunting for hydrocephalus remains poorly understood. Although MMAE has been reported as a treatment option for shunt-associated CSDH [11], evidence regarding its application in patients with pre-existing CSDH undergoing VP shunt placement remains limited. Here, we report a case of post-traumatic hydrocephalus with pre-existing CSDH that enlarged following VP shunt placement and subsequently regressed after MMAE, despite gradual reduction of the programmable valve pressure.

Case Presentation

A 78-year-old man was admitted for VP shunt placement approximately three months after sustaining a traumatic brain injury. At the time of the initial injury, he had traumatic subarachnoid hemorrhage, acute left subdural hematoma, and bilateral frontal and temporal cerebral contusions (Figure *1*). He was managed conservatively and remained hospitalized for approximately one month. Although cognitive impairment persisted, he was able to perform activities of daily living independently and was discharged home. The left

subdural hematoma persisted on follow-up imaging and gradually evolved into a CSDH (Figure 2). The maximum thickness of the left CSDH was 9 mm on computed tomography (CT) obtained two weeks before discharge and remained unchanged on subsequent outpatient imaging. Two weeks after discharge, gait deterioration was noted during outpatient rehabilitation. CT revealed ventricular enlargement, suggesting post-traumatic hydrocephalus, while the pre-existing left CSDH showed no interval enlargement (Figure 3). Further evaluation demonstrated an Evans index of 0.31, third ventricular ballooning, a callosal angle of 88°, and tight high-convexity sulci. A CSF tap test was performed. The CSF opening pressure measured during lumbar puncture was 14 cmH_2O, and 30 mL of CSF was withdrawn. Before the test, gait and cognitive function were assessed using the Timed Up and Go (TUG) test and Raven's Colored Progressive Matrices (RCPM), respectively. Manual muscle testing (MMT) revealed normal muscle strength (5/5) in all four extremities, without laterality. On the following day, the TUG time improved from 17 to 14 s, and the RCPM score increased from 24 to 28 points. However, these improvements were transient, and his symptoms returned to their previous levels approximately one week later. Based on these findings, VP shunting was planned. VP shunt placement was performed using a Miethke shunt system (Christoph Miethke GmbH & Co. KG, Potsdam, Germany), with cannulation of the posterior horn of the right lateral ventricle (Figure 4). The initial valve pressure was set at 12 cmH_2O, slightly below the CSF opening pressure measured during the tap test. Follow-up CT on the third postoperative day revealed enlargement of the left CSDH, with its maximum thickness increasing to 11.5 mm (Figure 5). At this time, neither gait nor verbal interaction had clearly improved compared with the transiently improved state observed after the tap test. MMAE was therefore performed on the fifth postoperative day. MMAE was performed under local anesthesia via right radial artery access. A 6 Fr Rist radial access guide catheter (Medtronic, Minneapolis, MN, USA) was positioned in the left external carotid artery. A Marathon microcatheter (Medtronic, Minneapolis, MN, USA), supported by a Guidepost slender distal access catheter (Tokai Medical Products, Inc., Aichi, Japan), was navigated into the parietal branch of the left middle meningeal artery using a CHIKAI-10 microguidewire (Asahi Intecc Co., Ltd., Aichi, Japan). Subsequently, 20% n-butyl cyanoacrylate (NBCA) was injected through the Marathon to achieve embolization (Figure 6). CT obtained the day after MMAE showed no further enlargement of the hematoma (Figures 7A, 7B). Given the absence of further hematoma enlargement and the persistent ventricular enlargement, the programmable valve pressure was gradually reduced from 12 to 9 cmH_2O. Three days later, follow-up CT showed no further hematoma enlargement, although ventricular enlargement persisted (Figures 7C, 7D). Therefore, the valve pressure was further reduced to 5 cmH_2O. These pressure adjustments were based on serial CT findings and clinical observations rather than a predefined protocol. The specific increments of pressure reduction were determined empirically, taking into consideration the need for adequate CSF diversion and the potential risk of CSDH progression. CT performed two weeks after MMAE demonstrated a reduction in both ventricular size and the left CSDH, with the maximum thickness of the hematoma decreasing to 9.5 mm (Figures 7E, 7F). His gait also improved on clinical observation, and he was discharged home. Before discharge, the RCPM score was 28 points, identical to the score observed after the tap test, and the TUG time was 15 s, representing improvement compared with the pre-tap test value of 17 s. Approximately one month after MMAE, follow-up CT demonstrated further reduction in the size of the left CSDH (Figures 7G, 7H). No additional intervention was required, and the final programmable valve pressure was maintained at 5 cmH_2O.

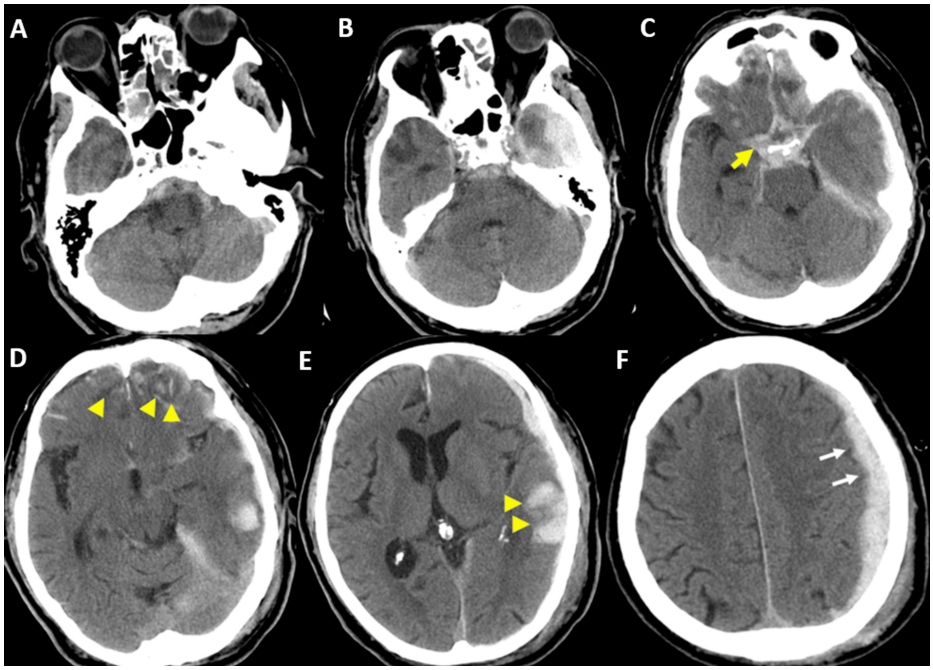


FIGURE 1: Baseline CT

(A-F) Head computed tomography (CT) obtained at the time of the initial injury showed traumatic subarachnoid hemorrhage (yellow arrow), an acute left subdural hematoma (white arrows), and bilateral frontal and temporal cerebral contusions (yellow arrowheads).

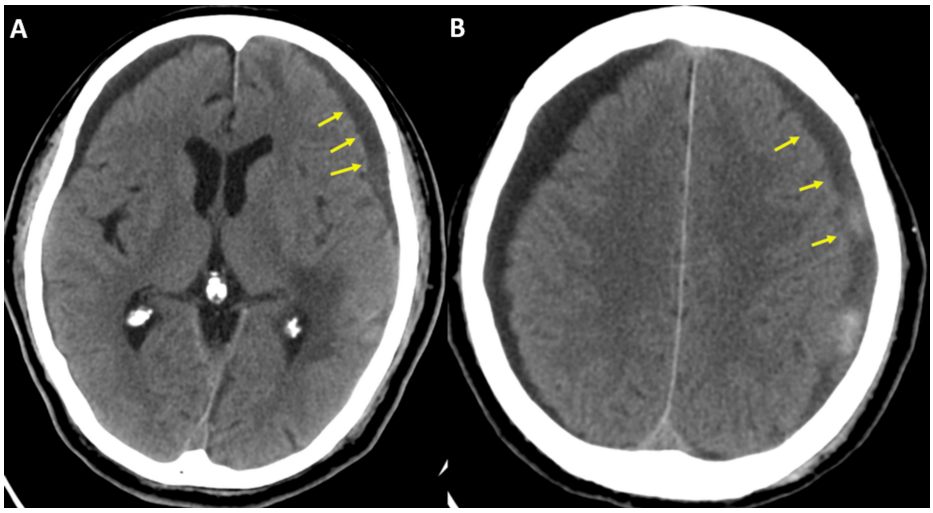


FIGURE 2: CT before discharge

(A, B) Follow-up computed tomography (CT) demonstrated a persistent left chronic subdural hematoma (CSDH) (yellow arrows).

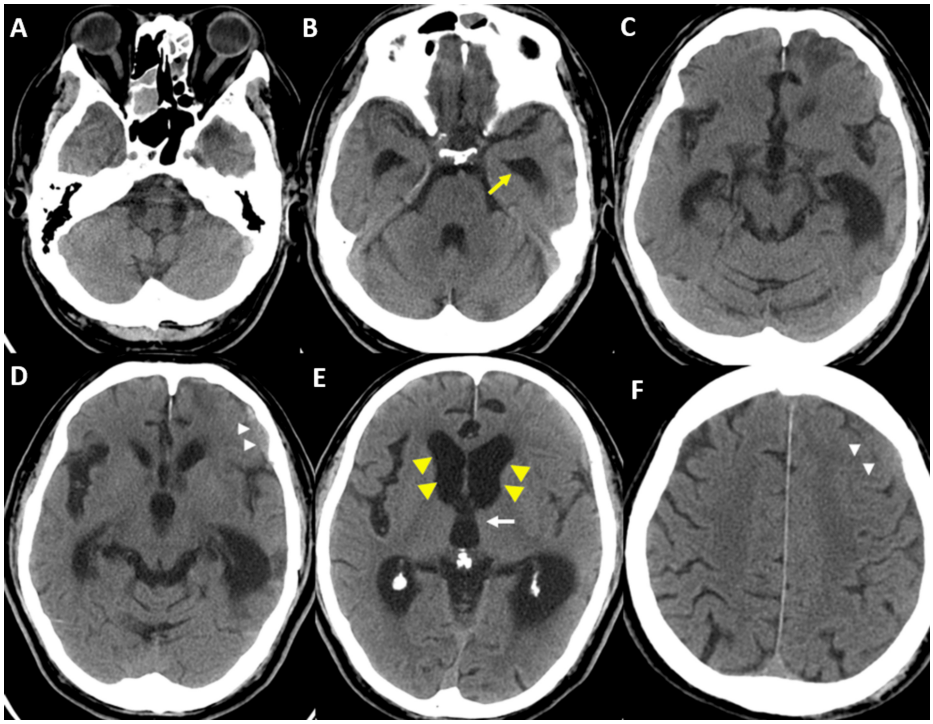


FIGURE 3: CT two weeks after discharge

(A-F) Head computed tomography (CT) demonstrated ventricular enlargement (yellow arrowheads), dilation of the temporal horns of the lateral ventricles (yellow arrow), and ballooning of the third ventricle (white arrow), with no interval enlargement of the pre-existing left chronic subdural hematoma (CSDH) (white arrowheads).

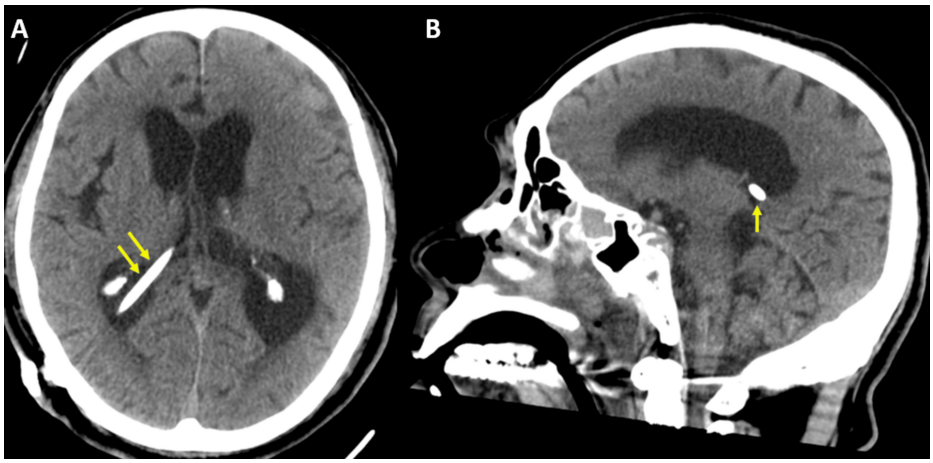


FIGURE 4: Postoperative CT after VP shunt placement

(A, B) A ventriculoperitoneal (VP) shunt was placed with the ventricular catheter inserted into the posterior horn of the right lateral ventricle (yellow arrows).

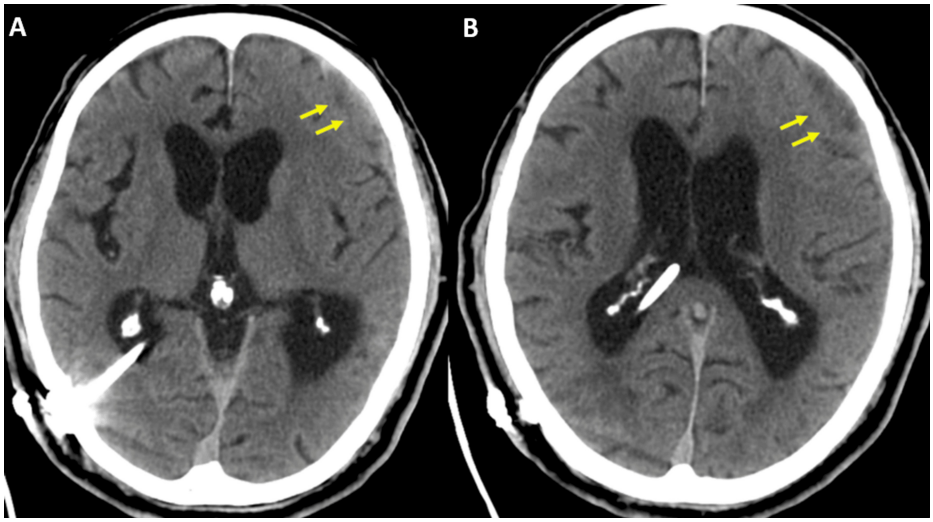


FIGURE 5: CT on postoperative day 3

(A, B) Follow-up computed tomography (CT) obtained on postoperative day 3 demonstrated enlargement of the left chronic subdural hematoma (CSDH), with its maximum thickness increasing to 11.5 mm.

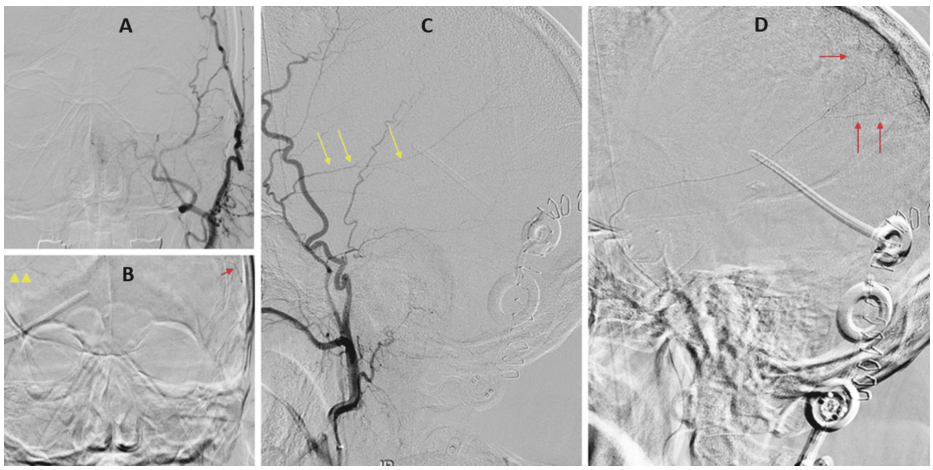


FIGURE 6: Angiographic images during middle meningeal artery embolization (MMAE)

(A-D) External carotid angiography (ECAG) demonstrated the posterior convexity branch of the left middle meningeal artery (MMA) (yellow arrows). Embolization was performed using 20% n-butyl cyanoacrylate (NBCA), injected through a Marathon microcatheter (red arrows), with embolization extending to the contralateral MMA (yellow arrowheads).

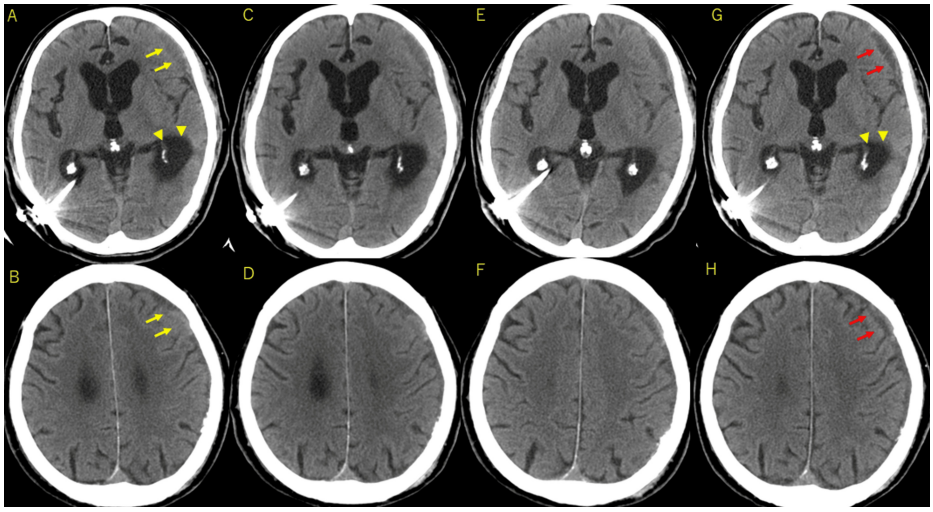


FIGURE 7: Serial CT findings after MMAE

(A, B) CT obtained one day after middle meningeal artery embolization (MMAE) showed no further enlargement of the left chronic subdural hematoma (CSDH) (yellow arrows), although dilation of the temporal horns of the lateral ventricles persisted (yellow arrowheads). (C, D) CT obtained three days after MMAE demonstrated persistent ventricular enlargement without further enlargement of the CSDH. (E, F) CT obtained two weeks after MMAE showed reductions in both ventricular size and the left CSDH, with the maximum thickness of the hematoma decreasing to 9.5 mm. (G, H) CT obtained approximately one month after MMAE demonstrated further reduction in the size of the left CSDH (red arrows). Yellow arrowheads indicate the temporal horns of the lateral ventricles.

Discussion

In this report, we present a case of post-traumatic hydrocephalus with pre-existing CSDH that enlarged following VP shunt placement but subsequently regressed after MMAE, despite further reductions in the programmable valve pressure. CSF overdrainage following VP shunt placement is a recognized risk factor for the development or enlargement of subdural fluid collections [1,12,13]. Excessive CSF drainage can lead to a reduction in intracranial pressure and brain volume, resulting in downward displacement of the brain and stretching of the bridging veins [12,13]. These changes may cause disruption of bridging veins and expansion of the subdural space, potentially leading to subdural hygroma or hematoma formation [1,12]. Furthermore, once a CSDH has developed, repeated microhemorrhages from fragile neovessels within the hematoma membrane may contribute to its progressive enlargement [14]. Thus, CSF overdrainage may not only induce new subdural collections but also exacerbate pre-existing CSDH. Conventional management strategies for shunt-induced subdural collections include increasing the programmable valve pressure, temporary shunt ligation or occlusion, and surgical hematoma evacuation when indicated [4,15-17]. Although these approaches can be effective in controlling hematoma enlargement, restricting CSF drainage may compromise the treatment of underlying hydrocephalus. Furthermore, hematoma evacuation alone may not prevent CSDH recurrence if excessive CSF drainage persists. In the present case, the patient demonstrated objective improvement in gait and cognitive function following the CSF tap test, supporting the need for CSF diversion. However, the pre-existing CSDH enlarged after VP shunt placement. Although observation or an increase in valve pressure could have been considered, we were concerned that these approaches might delay the treatment of hydrocephalus or limit the necessary CSF diversion. Therefore, MMAE was selected to address the vascular component of the enlarging CSDH while allowing subsequent adjustment of the shunt pressure to treat the hydrocephalus. This decision was based on an individualized assessment of the potential risks and benefits, rather than established evidence supporting routine MMAE in this setting. MMAE was initially introduced as a treatment for recurrent or refractory CSDH [6]. Since then, its application has expanded, and recent studies have suggested that MMAE may reduce treatment failure and the need for reoperation when used as an adjunct to standard treatment for CSDH [7-9]. MMAE has also been applied to CSDH occurring after CSF shunt placement. Meggyesy et al. reported their experience with MMAE in patients with shunt-associated CSDH, suggesting that it may be a treatment option in this population, although its benefit over conventional management remains uncertain [11]. The proposed mechanism of MMAE involves occlusion of the arterial supply to the vascularized outer membrane of the hematoma, thereby reducing blood flow to fragile neovessels and potentially suppressing repeated microhemorrhages and hematoma expansion [14,18]. However, MMAE does not directly address the mechanical factors associated with CSF overdrainage, such as brain displacement and stretching of the bridging veins. Nevertheless, by targeting the vascular component of CSDH progression, MMAE may help control hematoma enlargement even when the underlying mechanical factors persist. This approach may be particularly relevant in patients who require continued CSF diversion for the treatment of hydrocephalus. In the present case, the programmable valve pressure was gradually reduced from 12 to 9 cmH_2O and subsequently to 5 cmH_2O. These adjustments were guided by serial CT findings and clinical observations. The first reduction was made after CT confirmed no further hematoma enlargement following MMAE, while

ventricular enlargement persisted. The subsequent reduction was performed because ventricular enlargement remained evident on follow-up CT, again without further hematoma enlargement. The specific increments of pressure reduction were determined empirically based on clinical judgment rather than a predefined protocol. A gradual adjustment strategy was adopted to balance the need for adequate CSF diversion against the potential risk of further CSDH progression. Several factors may have contributed to the regression of CSDH in the present case. First, MMAE may have reduced the vascular supply to the hematoma membrane, thereby suppressing repeated microhemorrhages and allowing hematoma resorption to predominate. Second, although the programmable valve pressure was gradually reduced, this did not necessarily indicate a sustained increase in mechanical stress on the bridging veins, as the actual CSF drainage volume and intracranial pressure dynamics are influenced by multiple factors. Furthermore, changes in ventricular size and brain re-expansion may have altered the mechanical conditions affecting the subdural space. Taken together, these factors suggest that suppression of the vascular mechanisms underlying CSDH progression may have contributed to hematoma regression despite continued CSF diversion. At discharge, the patient's RCPM score was 28 points, matching the score observed after the tap test, while his TUG time was 15 s, improved from the pre-tap test value of 17 s. These findings indicate that the functional improvement observed after the tap test was maintained to some extent following treatment. However, the relative contributions of VP shunting and MMAE to the clinical improvement cannot be determined. Nevertheless, spontaneous hematoma resolution and the effects of valve pressure adjustments cannot be excluded. Although the CSDH regressed following MMAE despite further reductions in valve pressure, it remains unclear whether this outcome was attributable to MMAE, the natural history of the hematoma, or the combined effects of these interventions. Furthermore, the contribution of changes in CSF drainage and intracranial pressure dynamics cannot be determined in this case. As this is a single case without a comparison group, the relative contributions of these factors and a causal relationship between MMAE and hematoma regression cannot be established. Further studies are warranted to clarify the role of MMAE in patients with pre-existing CSDH who require CSF diversion for hydrocephalus.

Conclusions

In patients with pre-existing CSDH, careful management of shunt pressure is essential to balance adequate CSF diversion against the risk of hematoma progression. MMAE may represent a useful adjunctive treatment for managing pre-existing CSDH in patients who require VP shunt placement for hydrocephalus. This case suggests that MMAE may help control CSDH while allowing continued CSF diversion, even when further reduction of the programmable valve pressure is required. The management of these patients requires an individualized approach that considers both the progression of CSDH and the need for adequate CSF diversion. Further studies are needed to clarify the role of MMAE in patients with CSDH who require CSF diversion.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Kohei Funato

Acquisition, analysis, or interpretation of data: Kohei Funato, Daiki Ito, Keisuke Miura, Mao Yamamoto

Drafting of the manuscript: Kohei Funato, Mao Yamamoto

Critical review of the manuscript for important intellectual content: Kohei Funato, Daiki Ito, Keisuke Miura

Supervision: Kohei Funato

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **AI use statement:** The authors used ChatGPT (GPT-6, OpenAI, San Francisco, CA, USA) as follows: language polishing, translation in Abstract, Introduction, Case Presentation, Discussion, Conclusion. The AI-suggested grammar and spelling corrections were manually reviewed. The authors confirm that they have reviewed and verified all AI-assisted content and take full responsibility for the work. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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