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Case Report

A case of refractory chronic subdural hematoma and internal carotid artery stenosis sequentially treated with surgical drainage, middle meningeal artery embolization, and carotid artery stenting

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ABSTRACT

Background: Both chronic subdural hematoma (CSDH) and ischemic cerebrovascular disease are commonplace in the clinical context, and their combination is sometimes experienced. We describe a unique and challenging case in which both therapeutic interventions were indispensable and performed in a sequential manner. This report aims to discuss the management of hemorrhagic and ischemic conditions where CSDH and carotid artery stenosis coexist.

Case Description: An 83-year-old male presented with the left cerebral infarction due to the left internal carotid artery (ICA) stenosis. The coexisting left CSDH was surgically drained first. Then, the left middle meningeal artery (MMA) was endovascularly embolized to prevent hematoma recurrence under antiplatelet therapy, before the left carotid artery stenting (CAS) was successfully conducted. The subdural hematoma gradually grew but remained asymptomatic. However, he later presented with another stroke due to the progressive right ICA stenosis that had been conservatively treated initially. Emergency right CAS was required eventually.

Conclusion: Under the circumstances where CSDH is present but antiplatelet therapy is inevitable, MMA embolization could be a reasonable treatment option to avoid additional surgical procedures. Furthermore, early intervention should be considered even for asymptomatic carotid stenosis in terms of shortening the administration period of antiplatelet agents.

Keywords: Chronic subdural hematoma, Internal carotid artery stenosis, Middle meningeal artery embolization

INTRODUCTION

Both chronic subdural hematoma (CSDH) and ischemic cerebrovascular disease are common, and thus, their comorbidity is not rare; however, surprisingly, few reports exist where surgical interventions for both of them were necessary or considered.[2,9] We, herein, report a challenging case with CSDH and carotid artery stenosis because of the conflicting treatment policies. The purpose of this report is to share our experience and deepen our understanding of how to deal with the complex clinical factors. To the best of our knowledge, this is one of the few case reports that discuss specific and appropriate management.

CASE PRESENTATION

An 83-year-old male with a history of CSDH, previously treated with surgical drainage twice, presented with transient right hemiparesis. Head computed tomography showed the expansion of the left CSDH over 2 years [Figures 1a and b]. Magnetic resonance (MR) imaging revealed multiple brain infarctions in the left cortical border zone between the anterior and middle cerebral arteries [Figure 1c]. Subsequent MR angiography demonstrated the severe stenosis with an unstable plaque in the bilateral cervical internal carotid artery (ICA) (85% in the left, North American Symptomatic Carotid Endarterectomy Trial method), to which the brain infarctions were attributed [Figures 1d-f].

The transient right hemiparesis obviously resulted from the acute cerebral infarctions, which first required aspirin administration of 100 mg/day. Meanwhile, as the left CSDH expansion was expected under antiplatelet therapy, surgical drainage of the CSDH was concurrently performed. Furthermore, to avoid hematoma recurrence after upcoming carotid artery stenting (CAS) with long-term antiplatelet therapies, we conducted the left middle meningeal artery (MMA) embolization with platinum coils and n-butyl-2-cyanoacrylate (NBCA) 13 days after the evacuation [Figures 2a-c]. Then, 7 days after MMA embolization, the left CAS was successfully performed under dual antiplatelet therapy (DAPT) with aspirin and clopidogrel [Figures 2d and e]. Since the right carotid stenosis had been considered asymptomatic, we decided to conservatively observe the patient at that moment.

The patient was discharged with no complications. The left CSDH, though still asymptomatic, gradually expanded in 2 months [Figures 2f and g]. At the same time, the antiplatelet therapy was reduced to aspirin alone mainly because 2 months had passed since the left CAS, while paying attention to the growing hematoma. However, 2 months later, he presented with the left transient hemiparesis, and MR imaging on admission revealed the occlusion of the right ICA [Figures 3a and b]. The emergency right CAS was required [Figures 3c-e]. The symptom gradually improved and he was discharged with a modified Rankin scale score of 3. The left CSDH remained stable in size and also asymptomatic without any further treatment.

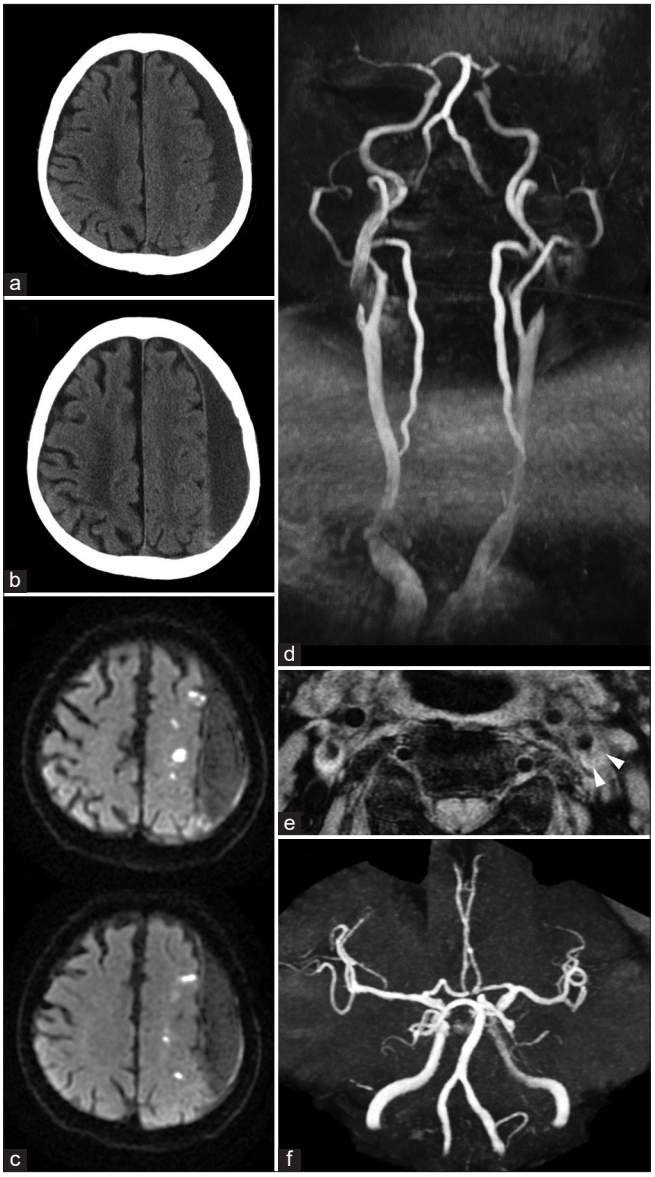

Figure 1: On admission, the expansion of the left chronic subdural hematoma was observed (b), compared to the finding 2 years before (a). Diffusion-weighted images showed the left watershed infarction between the anterior and middle cerebral arteries (c), which was resulted from the severe left carotid stenosis (d) with a T1 hyperintense plaque (e; arrowhead). Note that the contralateral carotid artery was also highly stenotic in the same degree. Intracranial large-vessel stenosis or occlusion was not present (f).

DISCUSSION

When both hemorrhagic and ischemic conditions coexist, we should assess how they will affect each other and progress in the future before formulating a treatment strategy. CSDH sometimes complicates or has a history of ischemic cerebrovascular diseases, and both of them tend to occur in middle-aged and older adults. The prevalence of CSDH incidentally combined with brain infarction or its history has been reported from 4% to 18%.[3,4,11] There are also the nonincidental cases in which brain infarction or ischemic symptoms were attributed to the mass effect of CSDH: compressive cerebral ischemia[7,16] or vascular transposition and malfunction[15], for instance. However, in actual clinical practice, CSDH is often induced or exacerbated by antithrombotic therapy for ischemic cerebrovascular diseases. Furthermore, as seen in this case, the well-

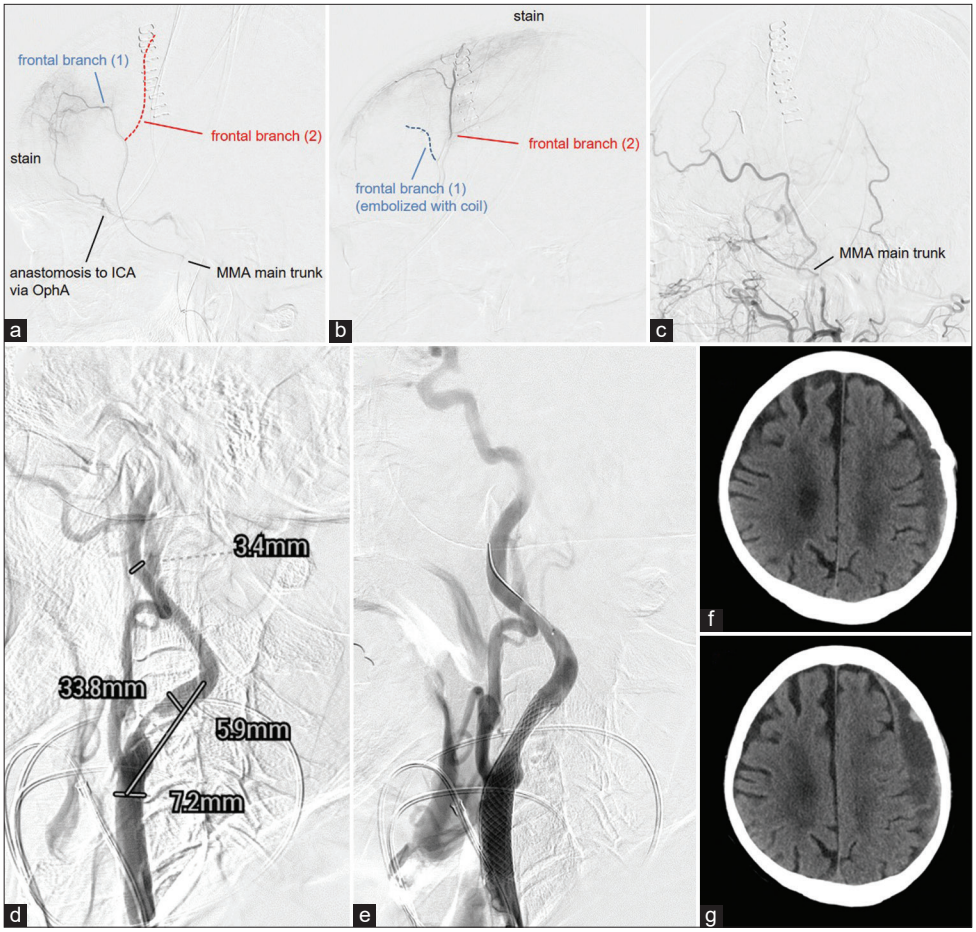

Figure 2: After the evacuation of chronic subdural hematoma (CSDH), the left middle meningeal artery (MMA) embolization was conducted. Angiography showed stains of the hematoma membrane and two frontal branches of the MMA were identified as feeders: one running anteriorly (1) and another superiorly (2). The former was embolized with platinum coils since the anastomosis to the internal carotid artery (ICA) through the ophthalmic artery was present (a), whereas the latter was treated with n-butyl-2-cyanoacrylate (b). The stains completely disappeared after embolization (c). Afterward, the left CAS was performed using a distal balloon protection device (d and e). Computed tomography scans over time showed the gradual expansion of the left CSDH in 2 months since the surgical drainage (f and g).

developed collateral circulation from the external carotid artery (ECA) due to severe ICA stenosis [Figure 2a] will result in the abundant blood supply through the MMA to the outer membrane of CSDH. This could lead to refractory CSDH. Therefore, it is difficult to make a strict distinction between incidental and nonincidental combination, and CSDH or ischemic cerebrovascular disease is related to each other both clinically and pathophysiologically to no small degree.

Antithrombotic agents for cerebral ischemia are essential, while they increase the risk of CSDH growth. An infarction lesion is irreversible; meanwhile, the symptoms of CSDH can be reversible with drainage procedures. Thus, the treatment of ischemia should be a priority, unless the mass effect of CSDH is immediately fatal. In this case, the initial surgical evacuation of CSDH before antiplatelet agents became effective, was a reasonable strategy. However, surgery alone cannot always control CSDH. Additional treatment options include endovascular therapy. MMA embolization for CSDH was first reported in 2000[10] and has been believed to reduce the recurrence of CSDH by devascularizing the outer membrane of subdural hematoma. Evidence for this treatment has been accumulating in recent years, and several clinical trials, including the evaluation of embolization alone strategies, are underway.[5,6,8,14] Some reports suggest that NBCA is an effective embolic material in preventing hematoma regrowth because of its high permeability to the peripheral vessels.[12,13]

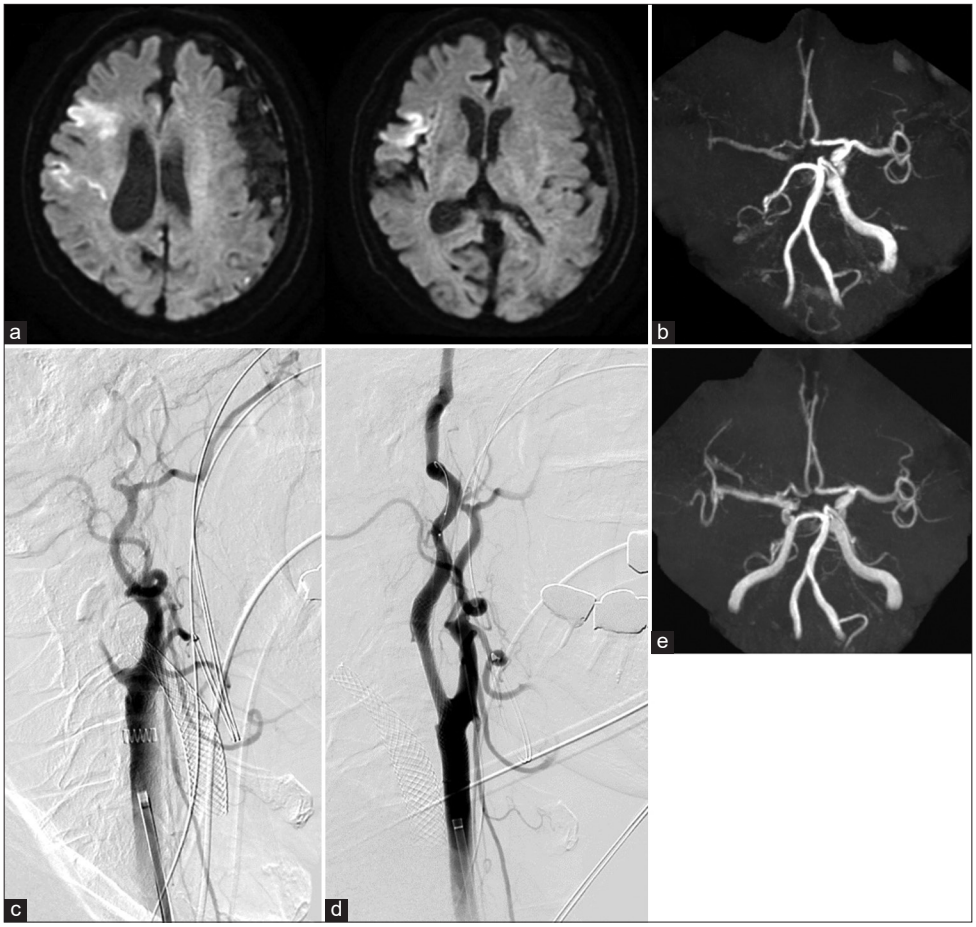

Figure 3: Diffusion-weighted images on the second admission showed acute cerebral infarction in the right middle cerebral artery territory (a). Magnetic resonance angiography revealed the right internal carotid artery occlusion and collateral circulation through the anterior communicating artery (b). The emergency right carotid artery stenting was necessary the next day (c and d), resulting in the flow restoration in the right cerebral hemisphere (e).

In this case, additional MMA embolization did not have a complete curative effect on CSDH. As discussed above, the well-developed collateral circulation through the ECA system might have increased the vascularity of the dura and accounted for refractory CSDH. Furthermore, aspirin combined with clopidogrel increased the risk of CSDH.[1] MMA embolization seems particularly promising for this kind of patient with a greater risk of CSDH recurrence and regrowth. Indeed, another surgery for CSDH was avoided in this case. However, it should be noted that the evaluation of the ECA vasculature is essential, and for vessels from which intracranial anastomoses obviously arise, as in this case, coil embolization would be safer instead of NBCA.

The right ICA was eventually occluded after clopidogrel discontinuation in the present case. Retrospectively, when and how to make an intervention for the right carotid stenosis, which was initially asymptomatic, is a considerable concern. Since DAPT longer than 3 months increases the risk of hemorrhagic complications,[17] a shorter period of DAPT administration is desirable while CSDH is present. In this case, one approach should have been to perform CAS for the right lesion not long after discharge, to minimize the duration of DAPT and reduce the risk of future stroke. We should be aware, however, that bilateral CAS in a short period of time could induce bradycardia and hypotension as a result of carotid sinus reflex. Another approach of conducting carotid endarterectomy in a drug reduction phase might have also been reasonable. Furthermore, repeated imaging of the asymptomatic right lesion, particularly after antithrombotic drug tapering, could have helped determine the appropriate timing of the additional intervention. Considering that an increase in CSDH will require a reduction in antiplatelet agents, which may then cause a rapid deterioration of the carotid stenosis, more aggressive treatment for an asymptomatic lesion than usual may be justified.

CONCLUSION

The case presented is unique in that both CSDH and ischemic cerebrovascular diseases required sequential interventions and challenging consideration of the treatment strategy. If CSDH is combined with cerebral ischemia requiring antiplatelet therapy, MMA embolization may contribute to avoiding additional surgical procedures for CSDH. The effect of MMA embolization on hematoma reduction still needs to be verified and the embolization should be conducted on the condition that dangerous anastomoses are not affected. At the same time, we should develop a treatment strategy so as to shorten the administration period of DAPT while CSDH is present. Thus, early intervention might be considered even for asymptomatic carotid stenosis. The overall treatment policy should be decided on a case-by-case basis.

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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