Radiology

Middle Meningeal Artery Embolization for Chronic Subdural Hematoma[1]

Seung Pil Ban, MD
Gyojun Hwang, MD, PhD
Hyoung Soo Byoun, MD
Tackeun Kim, MD
Si Un Lee, MD
Jae Seung Bang, MD
Jung Ho Han, MD
Chae-Yong Kim, MD, PhD
O-Ki Kwon, MD, PhD
Chang Wan Oh, MD, PhD

Purpose: To evaluate the effect of middle meningeal artery (MMA) embolization on chronic subdural hematoma (CSDH) and compare the treatment outcomes of MMA embolization and conventional treatment.

Materials and Methods: All consecutive patients 20 years or older with CSDH were assessed for eligibility. CSDHs with a focal location, a thickness of 10 mm or less, no mass effect, or underlying conditions were excluded. Seventy-two prospectively enrolled patients with CSDH underwent MMA embolization (embolization group; as the sole treatment in 27 [37.5%] asymptomatic patients and with additional hematoma removal for symptom relief in 45 [62.5%] symptomatic patients). For comparison, 469 patients who underwent conventional treatment were included as a historical control group (conventional treatment group; close, nonsurgical follow-up in 67 [14.3%] and hematoma removal in 402 [85.7%] patients). Primary outcome was treatment failure defined as a composite of incomplete hematoma resolution (remaining or reaccumulated hematoma with thickness > 10 mm) or surgical rescue (hematoma removal for relief of symptoms that developed with continuous growth of initial or reaccumulated hematoma). Secondary outcomes included surgical rescue as a component of the primary outcome and treatment-related complication for safety measure. Six-month outcomes were compared between the study groups with logistic regression analysis.

Results: Spontaneous hematoma resolution was achieved in all of 27 asymptomatic patients undergoing embolization without direct hematoma removal. Hematoma reaccumulation occurred in one (2.2%) of 45 symptomatic patients receiving embolization with additional hematoma removal. Treatment failure rate in the embolization group was lower than in the conventional treatment group (one of 72 patients [1.4%] vs 129 of 469 patients [27.5%], respectively; adjusted odds ratio [OR], 0.056; 95% confidence interval [CI]: 0.011, 0.286; P = .001). Surgical rescue was less frequent in the embolization group (one of 72 patients [1.4%] vs 88 of 469 patients [18.8%]; adjusted OR, 0.094; 95% CI: 0.018, 0.488; P = .005). Treatment-related complication rate was not different between the two groups (0 of 72 patients vs 20 of 469 patients [4.3%]; adjusted OR, 0.145; 95% CI: 0.009, 2.469; P = .182).

Conclusion: MMA embolization has a positive therapeutic effect on CSDH and is more effective than conventional treatment.

[1] From the Department of Neurosurgery, Regional Cardiocerebrovascular Center, Seoul National University Bundang Hospital, Seongnam, Korea (S.P.B., T.K., S.U.L., J.S.B., J.H.H., C.Y.K., O.K.K., C.W.O.); and Department of Neurosurgery, Hangang Sacred Heart Hospital, Hallym University, 12 Beodeunaru-ro 7-gil, Yeongdeungpo, Seoul 07247, Korea (G.H., H.S.B.). Received January 7, 2017; revision requested March 6; revision received June 19; accepted July 15; final version accepted August 14. **Address correspondence to** G.H. (e-mail: *storynlemon@gmail.com*).

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Complex pathophysiologic processes are involved in the formation and growth of chronic subdural hematoma (CSDH). Development of CSDH starts with the separation of the dural border cell layer, which triggers healing responses that include dural border cell proliferation, granulation tissue formation, and macrophage deposition (1–3). In this process, local inflammation is thought to contribute to CSDH formation (4,5) and leads to hyperfibrinolysis of the clot (6) and production of angiogenic factor, which induces neovascularization and bleeding from fragile capillaries (3,5,7). On the basis of these pathophysiologic mechanisms, various nonsurgical treatments have been proposed, but their effect is limited to selected patient groups (8–12). Therefore, surgical hematoma removal is the primary option in most CSDHs. Surgery is effective in relieving the mass effect of hematoma but does not change the underlying pathophysiologic mechanisms, and recurrence occurs frequently.

Advance in Knowledge

- Middle meningeal artery (MMA) embolization for treatment of repeated bleeding from chronic subdural hematoma membrane reduced the rate of treatment failure (incomplete hematoma resolution or surgical rescue at or before 6-month follow-up) as compared with conventional treatment including close follow-up and hematoma removal (one of 72 patients [1.4%] vs 129 of 469 patients [27.5%], respectively; adjusted odds ratio (OR), 0.056; 95% confidence interval (CI): 0.011, 0.286; P = .001); treatment-related complication rate was not different between patients undergoing MMA embolization and those undergoing conventional treatment (0 of 72 patients vs 20 of 469 patients [4.3%], respectively; adjusted OR, 0.145; 95% CI: 0.009, 2.469; P = .182).

CSDH has an outer membrane derived from the dura mater. Repeated bleeding from the membrane is currently considered to be a cause of the evolution of CSDH. This concept has been supported by imaging, advanced microscopy, and molecular biology studies (13–18). In theory, embolization of the middle meningeal artery (MMA), which is performed to inhibit blood influx into pathologic structures receiving blood through meningeal arteries, can control bleeding from the CSDH membrane and eventually enhance spontaneous resolution of the hematoma. This theoretical background is supported by several case reports (19–27). These case reports showed that complete resolution of recurrent CSDH refractory to repeated hematoma removal was achieved with simple bleeding control by using MMA embolization without direct hematoma drainage, although symptomatic hematoma required additional hematoma removal. Therefore, we hypothesized that MMA embolization could be used as a primary treatment option for CSDH. The purpose of this study was to evaluate the effect of MMA embolization on CSDH and to compare its treatment outcomes with the outcomes of conventional treatment.

Materials and Methods

Patients

This study was approved by our institutional review board. All patients provided written informed consent. All consecutive patients aged 20 years or older with CSDH were included in this study. We excluded patients with *(a)* CSDH that had a focal location (confined to the frontal or temporal base or the interhemispheric space without cerebral convexity involvement), was 10 mm or less in thickness, or had no mass effect (cortical flattening or midline shifting); *(b)* CSDH that developed with underlying conditions (vascular lesions, brain tumor, arachnoid cyst, spontaneous intracranial hypotension, or previous craniotomy); and *(c)* poor medical condition and with life expectancy of less than 6 months. We screened 104 patients between March 2015 and April 2016 to select study subjects for MMA embolization, and 32 patients were excluded: 28 with CSDHs of 10 mm or less in thickness or no mass effect, one with CSDH that developed with an arachnoid cyst, one with CSDH with spontaneous intracranial hypotension, and two with CSDHs after craniotomy. Therefore, 72 patients were prospectively enrolled in this study and underwent MMA embolization (embolization group). All patients followed the study protocol without refusal.

In the retrospective arm of the study, using the clinical data warehouse system of our institution, we reviewed 603 patients with CSDH who were given a diagnosis between May 2003 and February 2015, and 134 patients were excluded: 120 with CSDHs with focal location, thickness of 10 mm or less, or no mass effect; three with CSDHs with arachnoid cyst; seven with CSDHs with intracranial hypotension; and four with CSDHs after craniotomy. Finally, 469 patients who underwent conventional treatment were selected as the historical control group (conventional treatment group).

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Abbreviations:
CI = confidence interval
CSDH = chronic subdural hematoma
MMA = middle meningeal artery
OR = odds ratio

Author contributions:
Guarantors of integrity of entire study, G.H., O.K.K.; study concepts/study design or data acquisition or data analysis/interpretation, all authors; manuscript drafting or manuscript revision for important intellectual content, all authors; manuscript final version approval, all authors; agrees to ensure any questions related to the work are appropriately resolved, all authors; literature research, S.P.B., G.H., H.S.B., S.U.L., J.H.H., O.K.K.; clinical studies, S.P.B., G.H., T.K., J.S.B., J.H.H., C.Y.K., O.K.K., C.W.O.; statistical analysis, G.H., J.H.H.; and manuscript editing, S.P.B., G.H., J.H.H., O.K.K.

Conflicts of interest are listed at the end of this article.

Conventional Treatment

Symptomatic (headache unresponsive to analgesics or neurologic deficits) CSDHs were surgically managed with hematoma removal, including burr-hole drainage and craniotomy. For asymptomatic CSDHs, surgeons chose close follow-up or hematoma removal. When close follow-up was chosen, patients visited the outpatient clinic and underwent computed tomography (CT) every 2–4 weeks. The evaluation schedule was adjusted on the basis of serial CT scan findings. Patients were also instructed to visit the emergency department if any symptoms occurred. If the maximum diameter of the hematoma increased on serial CT images and eventually a clinical symptom developed during close follow-up, hematoma removal was performed.

The selection and technique of surgical methods were conventional. Patients undergoing antiplatelet or anticoagulation therapy were prepared with proper reverse method by means of platelet or factor transfusion or antagonist administration before surgery. An indwelling drainage catheter was used for several days in all patients and was removed after hematoma status was confirmed with CT. The antiplatelet or anticoagulation therapy was then resumed 1 month after the surgery, according to follow-up CT findings.

MMA Embolization Protocol

MMA embolization was performed as the primary option for all study subjects who were prospectively enrolled in the embolization group. MMA embolization was performed with polyvinyl alcohol particles and with local anesthesia. Systemic heparinization was not used. A 5-F guiding catheter was placed in the proximal external carotid artery or distal common carotid artery, and a microcatheter (Excelsior SL-10; Stryker Neurovascular, Fremont, Calif) was navigated into the MMA by using a guidewire (Synchro-14; Stryker Neurovascular). Before embolization, selective angiography through the microcatheter was performed to select target MMA branches and to detect potentially dangerous collateral vessels. MMA branches (frontal, squamosal, or parietal branches) supplying blood to convexity dura were selected as the target for embolization on the basis of findings at selective angiography. If no collaterals were observed, target branches were embolized with polyvinyl alcohol particles (Contour 150–250 μm; Boston Scientific, Marlborough, Mass). In cases with dangerous collaterals, the embolization was performed after the microcatheter was advanced more distally or collaterals were occluded with coils. When flow stasis of the MMA was confirmed, the procedure was concluded. The embolization procedure was considered successful when all target MMA branches were embolized without procedural complications.

According to the study protocol, asymptomatic patients underwent embolization as a sole treatment, whereas symptomatic patients additionally underwent hematoma removal as an adjunctive treatment for symptom relief after embolization. If the patient had a condition requiring antiplatelet drugs or anticoagulants, the drugs were not reversed and were maintained without discontinuation. Clinical and radiologic follow-up examinations with CT scans were performed at 1, 3, and 6 months after embolization.

Outcomes

The primary outcome was treatment failure, which was defined as a composite of incomplete hematoma resolution (remaining or reaccumulated hematoma with thickness > 10 mm at 6-month follow-up) or surgical rescue (hematoma removal for relief of symptoms that developed with continuous growth of initial or reaccumulated hematoma as discovered during follow-up). Secondary outcomes included surgical rescue as a component of the primary outcome and treatment-related complications, including all surgical and endovascular complications, for a safety measure.

Data Collection and Assessment

Demographic, clinical, laboratory, radiologic, and treatment-related data regarding study subjects were collected. Radiologic data included side, width, and stage of the hematoma; midline shifting; and the presence of brain atrophy. Hematomas were classified into four types (homogeneous, laminar, separated, and trabecular) and three stages (stage 1, homogeneous or laminar type; stage 2, separated type; and stage 3, trabecular type) according to a previously described staging system (28). Brains with widely dilated sulci and subdural space were considered to have atrophy (29).

All clinical and radiologic data regarding patients were electronically stored with only study subject number for identification (Amazon Elastic Compute Cloud; Amazon Web Service, Seattle, Wash). The independent data and safety committee members, who were blinded to patient information, accessed these data at their remote sites and adjudicated all primary and secondary outcomes.

Statistical Analyses

Recent reports demonstrated recurrence rates of 10% or less and 0% after burr-hole drainage and MMA embolization, respectively (26,30,31). However, because patients undergoing follow-up without hematoma removal were included in this study, we expected that treatment failure rates of the conventional treatment and MMA embolization would be increased, primarily based on a high treatment failure rate of close follow-up reported in several studies (9,10). Therefore, we assumed that treatment failure rates of conventional treatment and MMA embolization would be 12% and 2%, respectively, until the 6-month follow-up (rate difference, 10%). For comparison with 469 patients receiving the conventional treatment in the historical control group, we estimated that a sample size of 65 patients undergoing MMA embolization would provide 80% power to detect this rate difference of the primary outcome at a two-sided significance level of .05. Estimating a dropout rate of 10%, we enrolled the study candidates, targeting 72 patients undergoing MMA embolization.

The Wilcoxon rank sum test was used for continuous variables, and the χ^2 or Fisher exact test was used for nominal factors in comparisons of baseline characteristics. Comparison of outcomes between embolization and conventional treatment groups was performed by using logistic regression analysis, adjusted for factors showing baseline group difference at $P < .15$, and provided with an adjusted odds ratio (OR) and 95% confidence interval (CI). The penalized likelihood estimation method was used in logistic regression to deal with rare events.

Because the outcome of CSDH could be affected by various conditions, predefined subgroup analyses were added to explore the uniformity of the overall primary outcome difference. The primary outcome was compared in subgroups defined by hematoma removal, hematoma thickness (<20 vs ≥20 mm), presence of symptoms, and hematoma laterality (unilateral vs bilateral). Tests for interaction were performed to assess heterogeneity of treatment effect among subgroups. Because this study had a 2 × 2 factorial trial design consisting of MMA embolization and hematoma removal, we performed a post-hoc analysis to further evaluate the individual efficacy of MMA embolization and hematoma removal and interaction between the two treatments. According to a guideline for analysis and reporting of factorial trials (32), the efficacy of each treatment for primary outcome was estimated both "at the margin" and "inside the table." For "at the margin" analysis, the efficacy of each treatment was determined by comparing the primary outcome of all patients receiving the treatment with that of all patients not receiving the treatment. For "inside the table" analysis, the efficacy of each treatment was estimated by comparing the primary outcome in patients who received only the treatment with that in patients who received neither treatment. Testing for interaction between MMA embolization and hematoma removal was performed to evaluate whether the two treatments had efficacy for the primary outcome independently.

Statistical analyses were performed by using Stata Statistical Software, release 13 (Stata, College Station, Tex). Because comparison of secondary outcomes was an exploratory analysis, no adjustments for multiplicity were made. *P* values for interaction in subgroup analysis and comparisons of primary outcome in post-hoc analysis were corrected for multiplicity by using the Sidak method. Statistical significance was accepted for $P < .05$.

Table 1

Baseline Characteristics of Embolization and Conventional Treatment Groups

Characteristic	Embolization (*n* = 72, 13.3%)	Conventional Treatment (*n* = 469, 86.7%)	*P* Value
Male sex	48 (66.7)	347 (74.0)	.201
Age (y)*	69.3 ± 10.47	67.4 ± 13.16	.221
Trauma history	45 (62.5)	272 (58.0)	.522
Smoking	15 (20.8)	106 (22.6)	.879
Chronic alcoholism	12 (16.7)	75 (16.0)	.864
Hypertension	45 (62.5)	212 (45.2)	.008
Diabetes mellitus	16 (22.2)	90 (19.2)	.527
Cerebrovascular accident	8 (11.1)	39 (8.3)	.498
Coronary heart disease	13 (18.1)	48 (10.2)	.069
Liver disease	1 (1.4)	17 (3.6)	.491
Chronic renal failure	0 (0)	7 (1.5)	.602
Use of antiplatelet drugs or anticoagulants	29 (40.3)	121 (25.8)	.016
Platelet count (×10³/μL)*	219 ± 54.64	231 ± 74.68	.182
PT (INR)*	1.07 ± 0.209	1.07 ± 0.421	.922
aPTT (sec)*	35.5 ± 4.36	36.7 ± 10.65	.371
Brain atrophy†	21 (29.2)	106 (22.6)	.233
Ventriculoperitoneal shunt	2 (2.8)	8 (1.7)	.630
Symptomatic hematoma‡	45 (62.5)	278 (59.3)	.699
Hematoma side			
Right	25 (34.7)	149 (31.8)	.559
Left	28 (38.9)	213 (45.4)	…
Bilateral	19 (26.4)	107 (22.8)	…
Hematoma classification§			
Stage 1 (homogeneous or laminar type)	30 (41.7)	324 (69.1)	<.001
Stage 2 or 3 (separated or trabecular type)	42 (58.3)	145 (30.9)	…
Hematoma width (mm)*	19.6 (5.00)	20.3 (6.47)	.338
Midline shift (mm)*	6.6 ± 4.54	7.2 ± 4.82	.352
Hematoma removal			
None (observation)	27 (37.5)	67 (14.3)	<.001
Burr-hole drainage	43 (59.7)	394 (84.0)	…
Craniotomy	2 (2.8)	8 (1.7)	…

Note.—Except where indicated, data are numbers of patients, with percentages in parentheses. aPTT = activated partial thromboplastin time, INR = international normalized ratio, PT = prothrombin time.

* Data are means ± standard deviations.

† Brains with widely dilated sulci and subdural space on CT scan were considered to have atrophy (29).

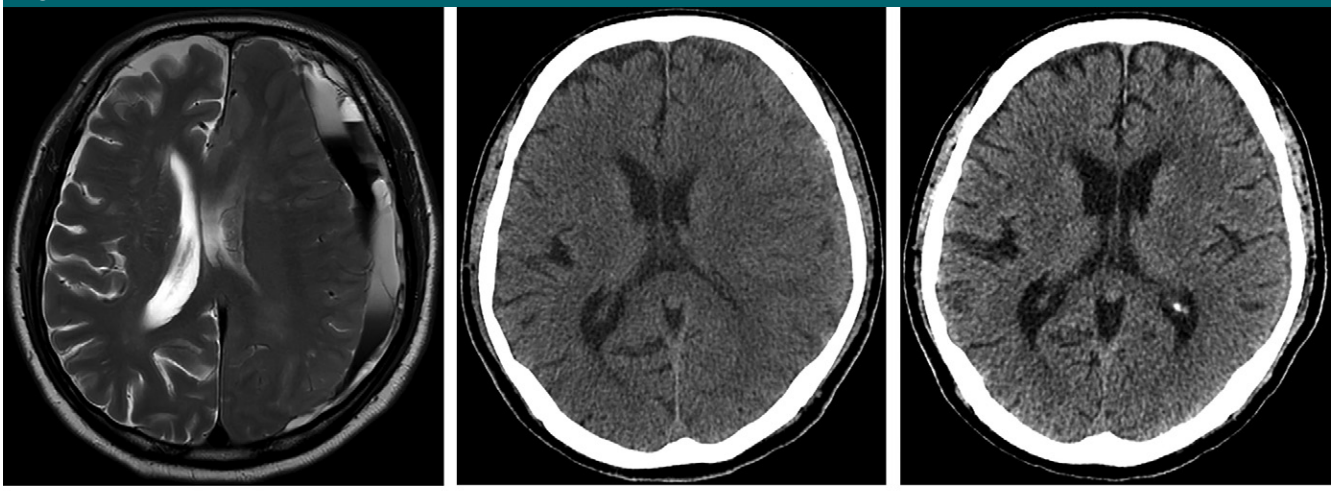
‡ CSDH that led to headache unresponsive to analgesics or neurologic deficits resulting from mass effect was considered to be symptomatic hematoma.

§ Hematoma was classified according to a prior staging system (28).

Results

Baseline characteristics of the embolization and conventional treatment groups are summarized in Table 1. More patients in the embolization group had a history of hypertension ($P = .008$), use of antiplatelet drugs or anticoagulants ($P = .016$), and separated or trabecular-type hematoma ($P < .001$). Although no statistically

Figure 1



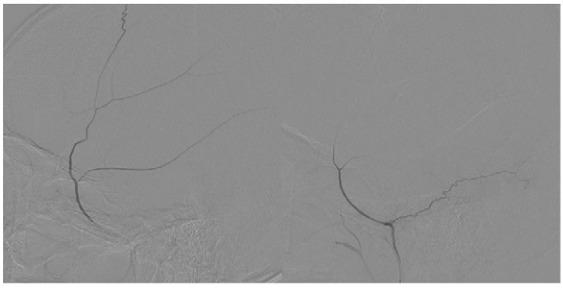


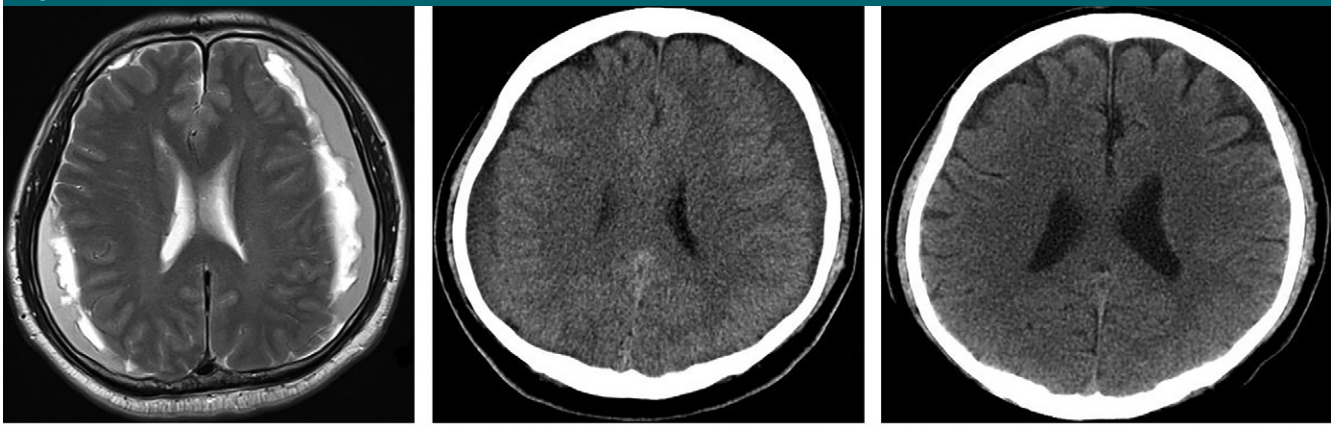
Figure 1: Images in a 67-year-old woman with asymptomatic CSDH treated with MMA embolization without direct hematoma removal. **(a)** Pretreatment axial T2 image shows multiseparated CSDH in left frontal, temporal, and parietal lobes (maximum hematoma width, 22.8 mm). **(b)** Selective angiography with the use of a microcatheter showed that frontal and parietal (squamosal) branches of MMA were main meningeal arteries supplying blood to convexity dura. Frontal and parietal branches were selected as target vessels and were embolized with polyvinyl alcohol particles. Petrosal branch of MMA was saved. **(c)** Axial nonenhanced CT scan at 1-month follow-up shows decrease in maximum width (10.2 mm) and mass effect of hematoma. **(d)** Three-month follow-up nonenhanced axial CT scan shows successful resolution of left-sided hematoma.

significant difference was shown, a history of coronary heart disease (P = .069) tended to also be more frequent in the embolization group. Hematoma removal, especially burr-hole drainage, was chosen more often than close follow-up for asymptomatic hematoma in the conventional treatment group, and thus the frequency of hematoma removal ($P < .001$) in the conventional treatment group was higher than in the embolization group.

Of 469 patients in the conventional treatment group, 191 (40.7%) were asymptomatic. Among these asymptomatic patients, close follow-up and hematoma removal were chosen as the first option in 67 (14.3%) and 124 (26.4%) patients, respectively. In 278 (59.3%) symptomatic patients, hematoma removal was performed as the primary treatment. Therefore, 402 (85.7%) patients underwent hematoma removal, and the remaining 67 (14.3%) patients underwent close follow-up. In 402 patients who underwent hematoma removal, the primary outcome occurred in 73 patients (18.2%) (incomplete resolution occurred in 41 patients [10.2%], and surgical rescue was performed in 32 patients [8.0%]). Among 67 patients who underwent close follow-up, spontaneous resolution occurred in 11 (16.4%), but surgical rescue was needed in 56 (83.6%) because of symptom development resulting from hematoma growth. Surgical complications occurred in 20 (4.3%; cerebral infarction in eight, acute epidural or subdural hematoma in seven, surgical infection in three, and intracerebral hemorrhage in two) of the 469 patients in the conventional treatment group. Cerebral infarction occurred in patients with carotid stenosis (three of 67, 4.5%) or atrial fibrillation (five of 38, 13.2%) after discontinuation of antiplatelet drugs or anticoagulants. Acute epidural or subdural hematoma occurred in patients with thrombocytopenia (two of 15, 13.3%) or residual effect of antiplatelet therapy (five of 93, 5.4%), despite platelet transfusion before surgery.

Of 72 patients in the embolization group, 27 (37.5%) were asymptomatic and underwent MMA embolization as a sole treatment. Forty-five (62.5%) symptomatic patients underwent MMA embolization and hematoma removal as an adjunctive treatment for symptom relief. MMA embolization was successful in all 72 patients. None of the patients with CSDH demonstrated treatment failure during follow-up after embolization (Figs 1, 2). However, hematoma reappeared in one patient, at 4 months after the initial embolization, following additional head trauma as a result of a traffic accident, and surgical rescue was eventually performed

Figure 2



a. b. c.

Figure 2: Images in a 77-year-old man with asymptomatic bilateral CSDHs receiving MMA embolization without burr-hole drainage. **(a)** Preembolization axial T2 image shows laminar-type chronic hematomas occupying left frontotemporoparietal and right frontoparietal areas (maximum hematoma width: left, 24.7 mm and right, 19.5 mm). **(b)** Axial nonenhanced CT scan obtained 3 months after embolization shows decrease in maximum width (left, 12.7 mm; right, 10.6 mm) and relevant mass effect of hematoma. **(c)** Six-month follow-up axial nonenhanced brain CT scan shows complete resolution of hematoma.

because of symptom recurrence. No complication related to endovascular or surgical procedures developed.

Comparison of Treatment Outcomes and Subgroup Analyses

The treatment failure rate during the 6-month follow-up in the embolization group was significantly lower than in the conventional treatment group (one of 72 patients [1.4%] vs 129 of 469 patients [27.5%], respectively; adjusted OR, 0.056; 95% CI: 0.011, 0.286; P = .001; Table 2). Subgroup analyses showed maintenance of this difference regardless of hematoma removal, hematoma width, presence of symptoms, and hematoma laterality, and lack of significant interactions (Fig 3). The frequency of surgical rescue in the embolization group was also significantly lower than in the conventional treatment group (one of 72 patients [1.4%] vs 88 of 469 patients [18.8%]; adjusted OR, 0.094; 95% CI: 0.018, 0.488; P = .005). MMA embolization did not increase treatment-related complication rate (0 of 72 patients vs 20 of 469 patients [4.3%]; adjusted OR, 0.145; 95% CI: 0.009, 2.469; P = .182).

Post-Hoc Factorial Design Analysis

Interaction between MMA embolization and hematoma removal was not significant (P = .128, Table 3). Therefore, MMA embolization ($P < .001$) and hematoma removal ($P < .001$) acted independently for reducing the treatment failure rate.

Table 2

Primary and Secondary Outcomes

Outcome	Embolization	Conventional Treatment	Adjusted OR*	*P* Value
Treatment failure	1/72 (1.4)	129/469 (27.5)	0.056 (0.011, 0.286)	.001
Surgical rescue	1/72 (1.4)	88/469 (18.8)	0.094 (0.018, 0.488)	.005
Treatment-related complication	0/72 (0)	20/469 (4.3)	0.145 (0.009, 2.469)	.182

Note.—Except where indicated, data are numbers of patients, with percentages in parentheses.

* Data in parentheses are 95% CIs. Each comparison of outcomes between the study groups was adjusted for histories of hypertension and coronary heart disease, use of antiplatelet drugs or anticoagulants, and hematoma classification.

Discussion

Results of this study revealed that controlling bleeding from the CSDH membrane with MMA embolization prevented further growth of hematoma and eventually led to spontaneous hematoma resolution without direct hematoma removal. Hematoma reaccumulation was also prevented by MMA embolization in symptomatic cases requiring hematoma removal. Embolization reduced the treatment failure rate as compared with conventional treatment performed in the historical control group. This effect was maintained regardless of various conditions that can affect the fate of CSDH.

Various medical treatments targeting inflammation and angiogenesis have been proposed but have failed to yield satisfactory results in clinical trials (8–12). We performed this study to further investigate effects of MMA embolization, because promising results were noted in previous case reports (19–27). Unlike previous researchers, we expanded the application of MMA embolization to a primary treatment option for new CSDHs and thus could compare its effects with those of conventional treatment.

The course of CSDH is determined by a balance of bleeding and absorption through its membrane. On the

Figure 3

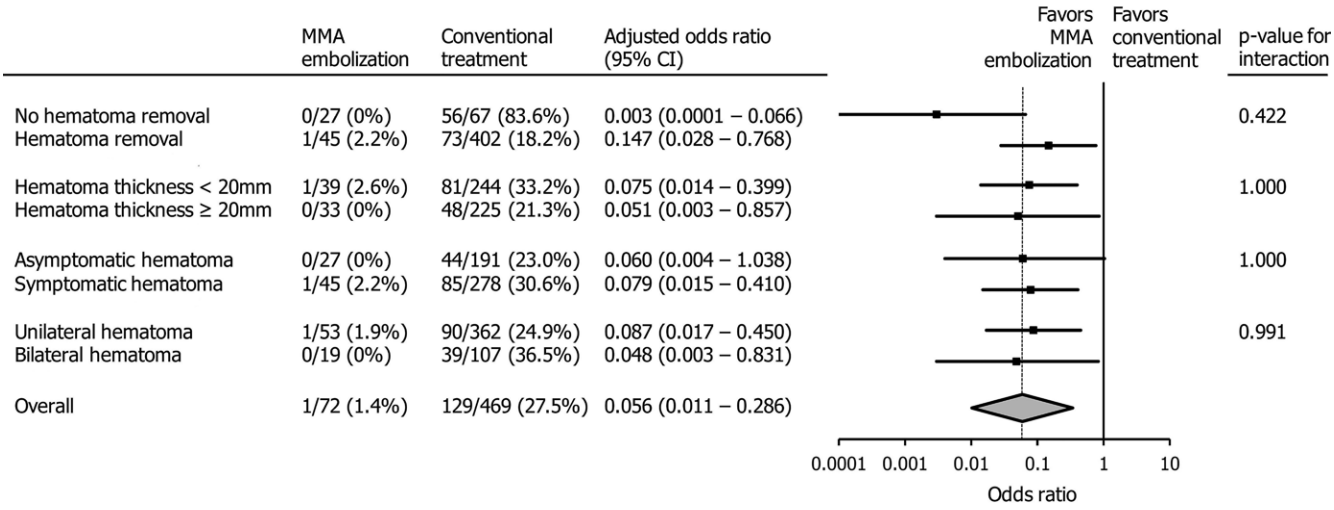


Figure 3: Graph shows subgroup analysis of primary outcome. *P* values for interaction were corrected for multiplicity by using the Sidak method.

basis of our observations of the historical control group and results of other studies (9,10), spontaneous resolution occurs less commonly, which suggests that bleeding exceeds hematoma absorption in most cases and that a more active approach than simple close follow-up is needed. No effective nonsurgical treatment has been developed, and thus many surgeons still prefer surgical removal, even of asymptomatic hematomas. MMA embolization can be used as an alternative to surgical hematoma removal for patients with asymptomatic CSDH.

The current surgical treatment cannot resolve the underlying pathophysiologic conditions. Because MMA embolization was found to overcome this drawback, MMA embolization followed by hematoma removal can be a better strategy for patients with CSDHs requiring surgical hematoma removal for symptom relief. In addition, we found that complications related to the surgical treatment were not uncommon in our historical control group, similar to other reports (30,31). Cerebral infarction caused by the discontinuation of antiplatelet drugs or anticoagulation and acute epidural or subdural hematoma in patients with thrombocytopenia or residual effect of antiplatelet drugs despite platelet transfusion were the most common complications. In planning this study, we assumed that discontinuation or reversal of these drugs would be unnecessary in patients undergoing embolization. In accordance with the study protocol, these drugs were maintained, and no related surgical complications developed. This finding suggests that MMA embolization can additionally contribute to the safety of surgical hematoma removal.

This study had the main limitation of being a single-center, nonrandomized study in which a historical control group was used. Therefore, a multicenter, randomized study is needed to support our findings. In addition, embolization may not be possible in cases with aberrant origin of the MMA and dangerous collaterals with the current endovascular technique, although we did not encounter such cases in this study.

In conclusion, this study demonstrated that MMA embolization facilitates resolution and prevents reaccumulation of CSDH and is more effective than conventional treatment without increasing treatment-related complications. Our results suggest that MMA

Table 3

Post-Hoc 2 × 2 Factorial Design Analysis

Analysis	Treatment Failure	OR*	*P* Value†
MMA embolization vs no MMA embolization			
At the margin	1/72 (1.4) vs 129/469 (27.5)	0.055 (0.011, 0.281)	<.001
Inside the table	0/27 vs 56/67 (83.6)	0.004 (0.0002, 0.651)	<.001
Hematoma removal vs no hematoma removal			
At the margin	74/447 (16.6) vs 56/94 (59.6)	0.136 (0.084, 0.219)	<.001
Inside the table	73/402 (18.2) vs 56/67 (83.6)	0.045 (0.023, 0.090)	<.001

Note.—Except where indicated, data are numbers of patients, with percentages in parentheses. Interaction between MMA embolization and hematoma removal, $P = .128$.

* Data in parentheses are 95% CIs

† *P* values were corrected for multiplicity by using the Sidak method.

embolization can be considered an alternative treatment for CSDH.

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