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Case Report

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Middle Meningeal Artery Embolization in the Treatment of Acute Subdural Hematoma: A Case Series Featuring Access Through a Carotid Stent

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ABSTRACT

The first objective of this case series was to describe the case of a patient with severe symptomatic left internal carotid artery stenosis after a recent stroke. Several days after stent placement by transcarotid artery revascularization, the patient developed a left-sided subdural hematoma. The patient then underwent embolization of the left middle meningeal artery (MMA) despite blocked access to the left MMA because of an internal carotid stent. The external carotid artery was accessed by passing a guiding catheter through the stent wall. We describe this method as "intrawall access." This allowed a coaxial system to deliver polyvinyl alcohol particles to the MMA for embolization. Embolization was successfully performed, with the stent integrity and blood flow through it remaining uncompromised. Overall, we demonstrated a new method of access through a previously placed internal carotid stent to gain neurointerventional access to the external carotid artery, which was jailed by a stent, for treating an acute subdural hematoma via MMA embolization. The second objective of this case series was to demonstrate the first MMA embolization in literature carried out in the acute or acute-on-subacute setting, in this case, and in four others.

Keywords: Acute subdural hematoma; Meningeal arteries; Carotid artery stenosis; Interventional radiology

INTRODUCTION

Atherosclerosis is a leading cause of stroke, particularly in the cervical or intracranial arteries, which is associated with 16% of ischemic strokes.[17] The internal carotid artery (ICA) is an area of specific interest when addressing stroke cause and prevention. ICA stenosis is associated with 8.0% of all ischemic strokes and ICA occlusion is associated with 3.5% of all ischemic strokes.[6] Treatment for carotid artery stenosis includes conservative medical management and surgical options of endarterectomy or stenting. In general, surgical options are considered in symptomatic patients with stenosis greater than 50% if perioperative mortality risk is less than 6%. For asymptomatic patients, surgical options are considered in patients with stenosis greater than 50%–60% and perioperative mortality risk of less than 3%.[20] In patients with a

Presentation

Case 1 was presented as an abstract and poster at the 2022 American Academy of Neurology Conference in Seattle, Washington, USA on April 6, 2022. The remaining cases (2-5) have never been presented before.

stenosis or occlusion of the carotid artery contralateral to the artery under operation, there is an increased risk of perioperative stroke or death with endarterectomy.[7] These increased risks were not noted in groups that underwent stenting.[15] Transcarotid artery revascularization (TCAR) is a method of carotid stenting in which the catheter is inserted at an incision in the supraclavicular area. It temporarily reverses the blood flow and re-routes it to a catheter in the femoral vein to prevent atherosclerotic debris from flowing into the brain. It is associated with the lowest risk of stroke or death both at the 30-day and 1-year mark compared to any other carotid stenting method.[18]

Subdural hematoma (SDH) is a hemorrhage between the dura mater and the arachnoid mater, mostly associated with a tear in the bridging veins. Over time, blood accumulates in the subdural space leading to migration of inflammatory cells. This promotes an upregulation of vascular endothelial growth factor and angiogenic factors angiopoietin 2. This leads to angioedema and neovascularization of the dura mater with immature arterial branches which are friable and permeable and can leak blood into the SDH.[8,19] These branches are anastomosed to the middle meningeal artery (MMA). Embolizing the MMA cuts off the blood supply to these neovascularizations and therefore promotes resolution of the hematoma by halting continued blood leakage to the area.[13] This development has led to the emergence of MMA embolization as a treatment for chronic SDH.[1,5,9,10,12,13] We seek to explore MMA embolization in the acute SDH setting.

In this case series, we begin by showing a case in which a patient with severe bilateral carotid atherosclerosis underwent TCAR carotid stenting after a stroke. Several days after, he developed an acute SDH ipsilateral to the stent. A guiding catheter was utilized to gain intra-wall access through the stent to reach the MMA to embolize the MMA. This is the first documented case of intra-wall access in the neurointerventional setting and it is the first documented case of MMA embolization done in the acute or acute on subacute SDH setting. We follow this case with four other examples of MMA embolization in the acute SDH setting.

CASE REPORT

Case 1

The patient is a 76-year-old male who presented after waking up with hemipararesis, facial droop and paresthesia of the left side as well as dysarthria. He had no known medical history and had not seen a physician in the previous 15 years.

Computed tomography (CT) angiography revealed 100% occlusion of the right proximal ICA and 90% stenosis of the left ICA (**FIGURE 1**). Magnetic resonance imaging confirmed an acute infarct in the right frontoparietal region, as well as acute punctate infarcts in the right frontal and left parietal lobes. The patient was eventually discharged to a rehabilitation facility in stable condition. The patient was on dual antiplatelet therapy with aspirin and clopidogrel. Intervention on the ICA atherosclerosis was planned later in the ambulatory surgical setting.

Three and a half months after the stroke, the patient underwent TCAR of the left common carotid artery and ICA. The left ICA was predilated with a Sterling™ balloon (Boston Scientific, Inc., Marlborough, MA, USA). Then, a 9 mm × 40 mm ENROUTE® transcarotid stent (Silk Road Medical, Inc., Sunnyvale, CA, USA) was placed. The patient tolerated the procedure well and was discharged on dual antiplatelet therapy.

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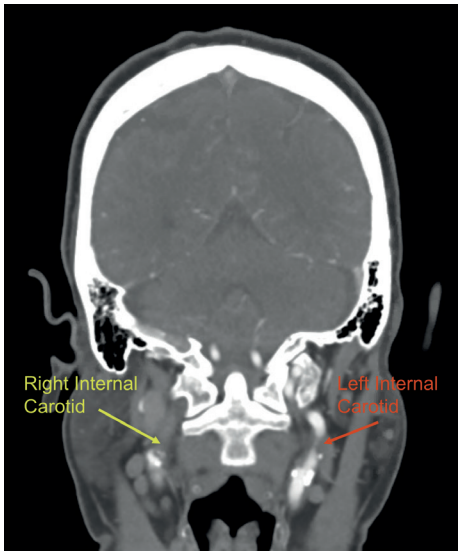

FIGURE 1. CTA noting internal carotid artery atherosclerosis.
CTA in coronal view shows 100% stenosis of the right internal carotid artery (yellow arrow) and 90% stenosis of the left internal carotid artery (red arrow).
CTA: computed tomography angiography.

Twelve days after the TCAR procedure, the patient presented to the emergency room (ER). He had developed progressive right upper extremity weakness since the TCAR procedure. Additionally, he reported a three-day history of headache. No trauma had occurred leading up to this event. CT of the head noted an acute left frontal SDH with left-to-right midline shift (**FIGURE 2A**). Antiplatelet therapy was held.

The patient then underwent MMA embolization. Arterial access was established through the right common femoral artery. A coaxial system was utilized using a 6 French BENCHMARK™ 071 guiding catheter (Penumbra, Inc., Alameda, CA, USA) over a Marathon™ microcatheter (Medtronic, PLC, Parkway Minneapolis, MN, USA) and a Synchro®-10 microguidewire (Stryker, Inc., Kalamazoo, MI, USA). This system was navigated under biplane roadmap conditions through the left external carotid artery trunk (**FIGURE 2B**). In doing so, the guiding catheter gained intra-wall access through the carotid stent. After passing through the stent, the guide catheter was navigated to the proximal left internal maxillary artery. The microcatheter was then navigated superselectively through the extracranial left MMA and then past the foramen spinosum and the pterional segment superselectively into the origin of the ascending frontal branch (**FIGURE 2C**). Under continuous fluoroscopic monitoring, Cook® polyvinyl alcohol particles with a size of 180–300 microns (Cook Group, Inc., Bloomington, IN, USA) were injected to devascularize the distal vascular bed. A superselective study via the microcatheter demonstrated complete obliteration of the frontal (**FIGURE 2D**), parietal, and petrosquamosal trunks of the MMA.

The patient tolerated the procedure well with no complications.

Carotid Doppler ultrasound done 8 days (**FIGURE 3A**) and 5 months (**FIGURE 3B**) after the embolization noted widely patent left ICA stent with normal antegrade blood flow.

Multiple surveillance CT head studies were done to monitor the progress of the hematoma, which each showed progressive resolution of the bleed. The last CT to note chronic blood was

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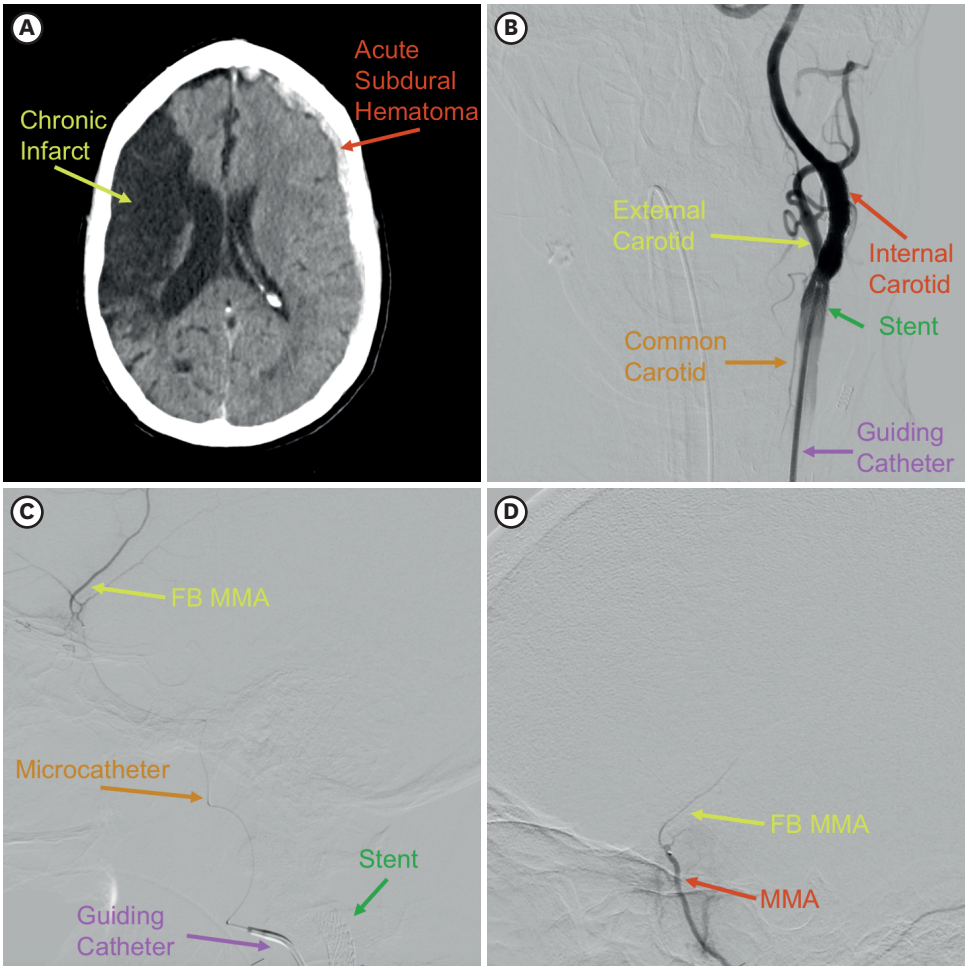

FIGURE 2. Computed tomography noting subdural hematoma (A) and angiography (B-D) showing the process of middle meningeal artery embolization. Upon re-presentation to the hospital with right upper extremity weakness and headache, computed tomography was taken of the head and displayed in axial view in (A). Left hemisphere acute subdural hematoma was noted (red arrow). Chronic infarct from the patient's stroke approximately 4 months prior noted on right hemisphere (yellow arrow). Left middle meningeal artery (MMA) embolization was done to stop the hematoma and is depicted in digital subtraction angiography in (B-D). (B) shows the stent (green arrow) which was previously placed in the common carotid artery (orange arrow) and into the internal carotid artery (red arrow). This has jailed the external carotid artery (yellow arrow). To access the external carotid artery, the guiding catheter (purple arrow) is puncturing through the stent. The arteries appear black from nonionic contrast during angiogram. In (C), the guiding catheter (purple arrow) has successfully punctured through the stent and has accessed the external carotid artery. The stent incased by the internal carotid artery is noted as well (green arrow). The microcatheter has been advanced up to the frontal branch of the middle meningeal artery (FB MMA) (yellow arrow). (D) shows a closer view of the MMA (red arrow) as it extends into the FB MMA (yellow arrow) demonstrating an obliterated frontal branch. Other branches of the middle meningeal artery, which were obliterated (parietal, and petrosquamosal) are not shown.
MMA: middle meningeal artery, FB MMA: frontal branch of the middle meningeal artery.

done four weeks after embolization (**FIGURE 3C**). The next subsequent CT head study was done ten weeks after embolization and noted resolution of the hematoma (**FIGURE 3D**). He was then restarted on aspirin.

Between case commentary

While case 1 is the only case that demonstrates intra-wall access, cases 2–5 will further illustrate MMA embolization for acute SDH. For brevity, we show CT scans to provide insight into the course of the SDH, but do not show digital subtraction angiography from the MMA embolizations as 1) the technique was similar in all cases and 2) the primary purpose of showing digital subtraction angiography from the MMA embolization in case 1 was to highlight the technical details of the intra-wall technique.

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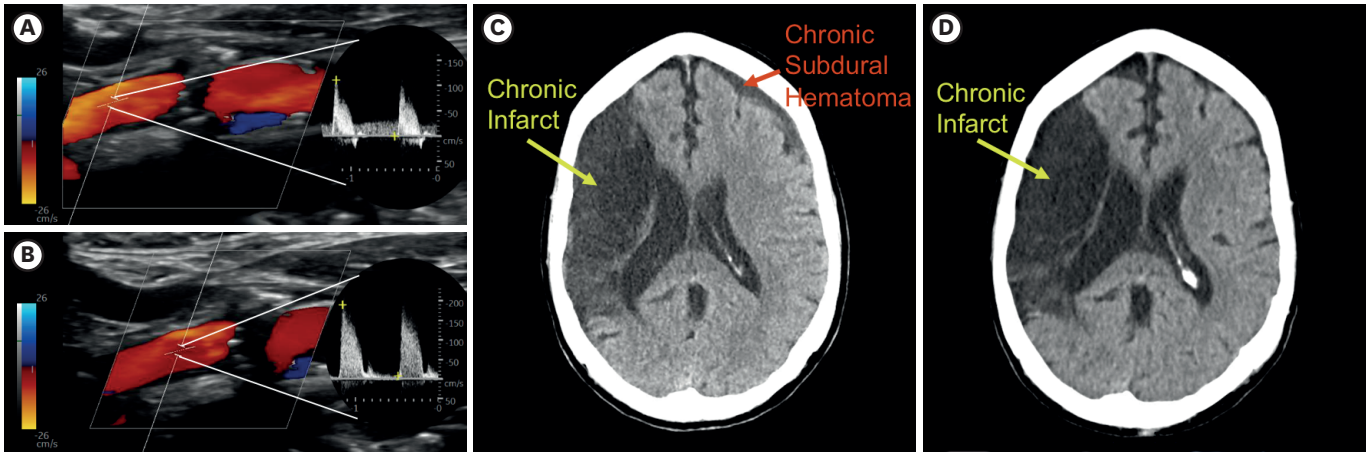

FIGURE 3. Post middle meningeal artery embolization surveillance imaging.
Spectral Doppler of the left external carotid artery are shown 8 days (A) and 5 months (B) after the MMA embolization. In both figures, normal flow and waveform pattern without stenosis was noted. This demonstrates that intra-wall access of the stent at the site of the external carotid artery origin did not compromise blood flow. In the color Dopplers, anterograde blood flow is depicted in yellow/red and retrograde blood flow is depicted in blue. Encircled to the right of each color Doppler image is a sample of the waveform with time in seconds on the x-axis and the flow velocity in centimeters/second in the y-axis.
Four weeks after MMA embolization, (C) depicts the last surveillance computed tomography (CT) of the head in axial view to show remaining hematoma in left hemisphere (red arrow). Chronic infarct from the patient's stroke approximately 4 months prior noted on right hemisphere (yellow arrow). (D) depicts the first surveillance CT head to show resolution of hematoma was done ten weeks after MMA embolization. The chronic right hemisphere infarct is still noted (yellow arrow).
CT: computed tomography, MMA: middle meningeal artery.

Case 2

The patient is a 72-year-old female with a past medical history significant for hyperlipidemia and a subarachnoid hemorrhage secondary to a ruptured aneurysm. Six year prior, the patient experienced a ruptured aneurysm of the left anterior communicating artery, which was coiled. One year prior to presentation, further coiling was undergone and an LA1-LA2 Neuroform Atlas™ stent (Stryker, Inc.) was placed after residual filling was noted on surveillance magnetic resonance angiography (MRA). She has since been on dual antiplatelet therapy with aspirin and clopidogrel.

Her presentation related to a fall while getting out of a bathtub. She impacted her head during the fall. Although she did not have an immediate headache, she developed one in subsequent days. Three days after the fall, the patient coincidently had a surveillance MRA for her aneurysm, bilateral acute SDH were noted. Two days later, at a follow-up appointment, the patient was advised to stop her antiplatelet therapy and present to the hospital. A CT of the head conducted upon presentation confirmed the SDH (**FIGURE 4A**), unchanged from what was found on MRA. The patient was hospitalized for monitoring for three days and was discharged. At the time of discharge, her headache had stopped, and no neurological deficits were noted. However, at a follow-up appointment two days later, her husband reported confusion and difficulty finding words. She had also mistaken her hair straightener for toothpaste that morning. Two days after that, the patient underwent bilateral MMA embolization with injection of Onyx™ liquid embolic system (Medtronic, PLC). A follow-up CT of the head three weeks after MMA embolization notes significant decrease in blood (**FIGURE 4B**) and after two months, a follow-up CT of the head noted resolution of the collections (**FIGURE 4C**). Following the two-month follow-up CT, the patient was re-started on aspirin monotherapy.

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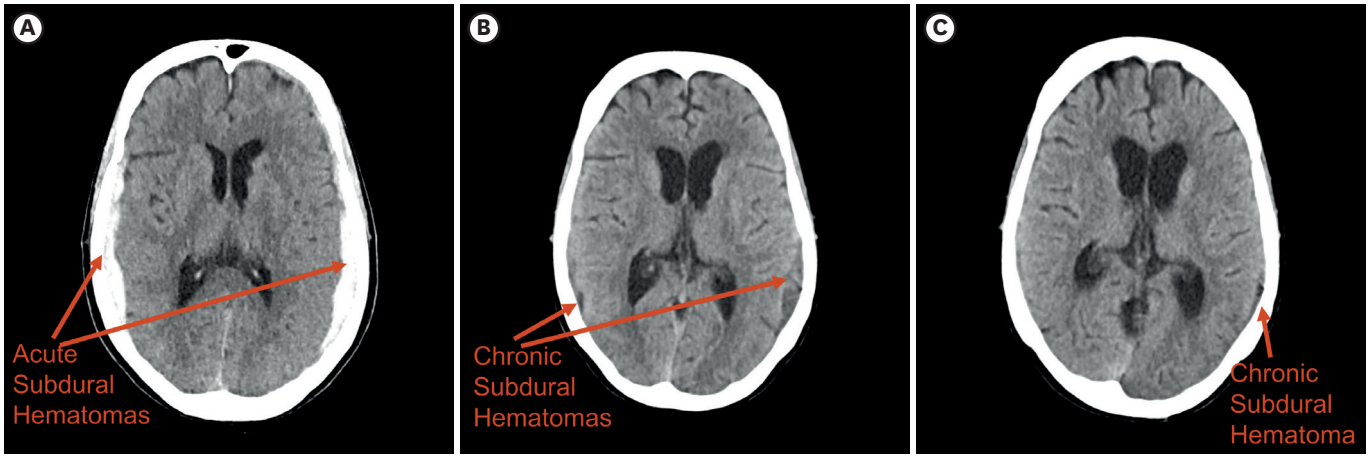

FIGURE 4. Progress of subdural hematomas by CT surveillance.
The initial CT of the head in axial view noted a large right-sided subdural hematoma with elements of both acute and subacute bleeding as well as mass effect and right-to-lift midline shift (A). (B) shows resolving chronic bilateral subdural hematomas reduced to only the temporal areas on CT one month after initial presentation and three weeks after middle meningeal artery embolization. Two months after initial presentation, surveillance CT notes continued progress with trace bilateral temporal subdural hematomas, of which the left side can be seen in (C).
CT: computed tomography.

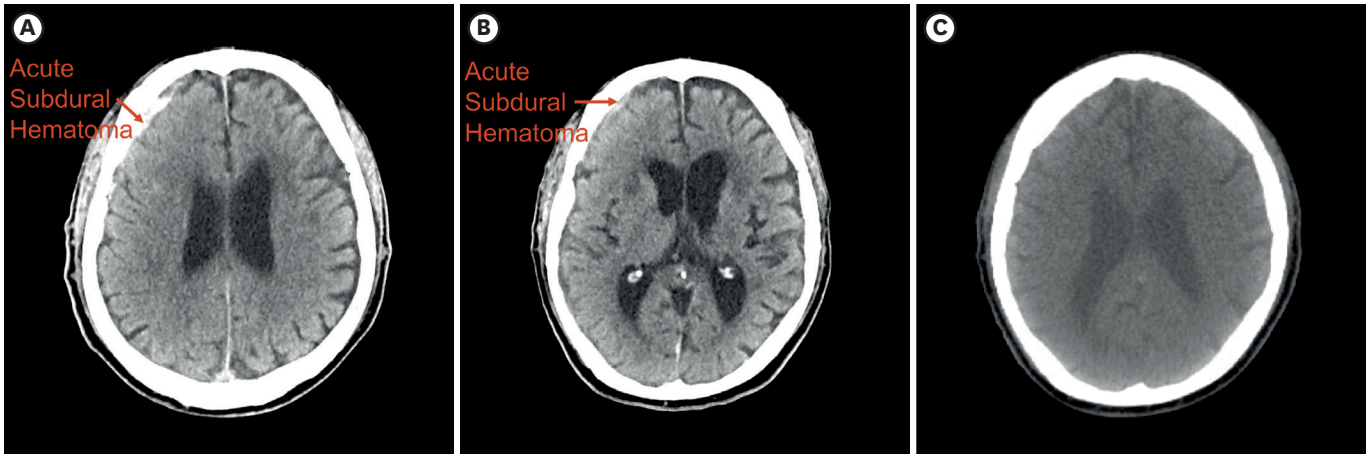

FIGURE 5. Progress of subdural hematoma by CT surveillance.
The initial CT of the head upon presentation was negative for hemorrhage. On day 4 of hospitalization, acute subdural hematoma was noted on magnetic resonance imaging of the brain and a follow-up CT was immediately done. An axial slice of that CT shows acute subdural hematoma in (A). The hemorrhage progressively improved on multiple surveillance CTs during hospitalization, but then a slight re-expansion of the bleed was noted on day 13 and is best shown in (B) with the most pronounced part of the bleed slightly inferior to where it was initially. The patient underwent right-sided MMA embolization on day 19. After discharge, further surveillance CTs were not done, however a CT slice from a positron emission tomography scan done five months after the initial hemorrhage and four months after hospital discharge show resolution (C).
CT: computed tomography.

Case 3

The patient is a 76-year-old male with a past medical history significant for mechanical aortic heart valve on warfarin, end-stage renal disease on hemodialysis, hypertension, hyperlipidemia, prostate cancer, intraparenchymal hemorrhage of the left thalamus and basal ganglia two years prior to presentation, and punctate inferior left cerebellar infarct one year prior to presentation.

The patient had developed a severe headache behind his right eye. Half an hour later, he became unresponsive as witnessed by his wife. Upon arrival in the ER, the patient was responsive only to painful stimuli initially, but then regained consciousness. Initial CT of the

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head in the ER was unremarkable. Electrolyte derangement was not noted, but the patient was due for dialysis the next day, which he received. He continued to report a headache. The patient underwent meningitis work-up which was negative. On day 4 of hospitalization, an MRI of the brain was done which revealed a small acute right SDH and a CT of the head was then done to confirm the acute bleed (**FIGURE 5A**). Warfarin was stopped at that time. The bleed progressively improved and the patient was therefore placed on a heparin drip on day 10 of the hospitalization. The heparin was stopped on day 13 of hospitalization as mild interval increase of blood was noted on CT of the head (**FIGURE 5B**). On day 19 of hospitalization, the patient underwent right-sided MMA embolization. Heparin drip was re-started after the procedure with warfarin re-started 3 days after that. The patient was discharged on day 24 of hospitalization. At outpatient neurological follow-up two months later, his wife reported confusion. Neurological exam was otherwise normal. Four months after discharge, the patient was fully alert at outpatient neurological follow-up. While, no further surveillance CTs were done, the patient underwent A positron emission tomography (PET) scan for a work-up to rule out head or neck cancer five months after the initial hemorrhage and four months after hospital discharge. A CT from that PET scan shows resolution of the bleed (**FIGURE 5C**).

Case 4

The patient is a 74-year-old female with a past medical history of non-Hodgkin's lymphoma status post T-cell transplant, hyperlipidemia, prediabetes, and a right leg deep vein thrombosis (DVT) which was being treated with rivaroxaban.

The patient presented to her primary care physician and reported a one-week history of lethargy and a headache, which she rates as a 6/10 in severity and was located across her forehead. There was associated nausea and the patient had vomited prior to the clinic visit. Ataxic gait was found on exam. She was sent directly to the emergency room from the clinic. In the ER, she was initially awake and alert. A CT of the head noted a mixed density left hemisphere SDH with 1.4 cm leftward midline shift as well as left to right subfalcine herniation and effacement of the suprasellar cisterns (**FIGURE 6A**) with acute and subacute

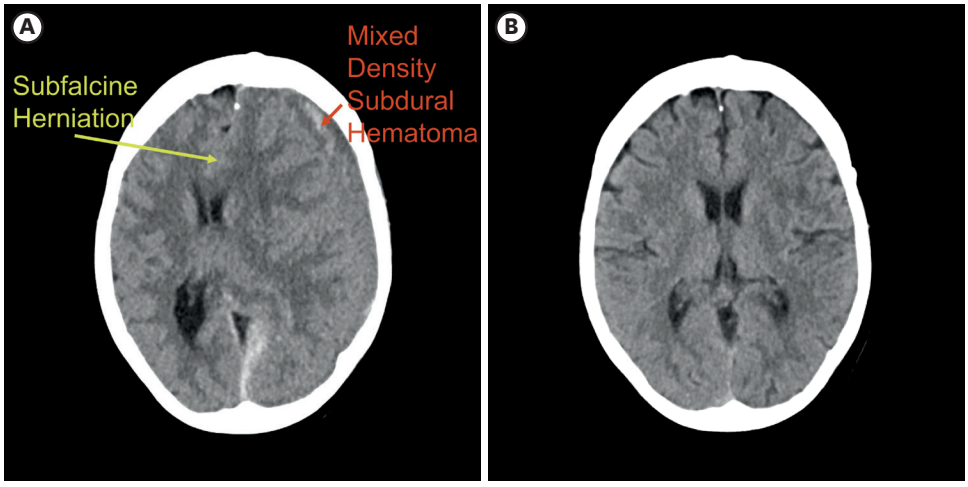

FIGURE 6. Progress of subdural hematoma by CT surveillance.
Initial CT of the head in axial view shows mixed density subdural hematoma with acute and subacute components at the left hemisphere (red arrow) with left-to-right subfalcine herniation (yellow arrow) as shown in (A). Effacement of the suprasellar cisterns was also noted but now shown. In (B), a follow-up CT of the head six weeks status post craniotomy and evacuation with middle meningeal artery embolization shows resolution of the hematoma.
CT: computed tomography.

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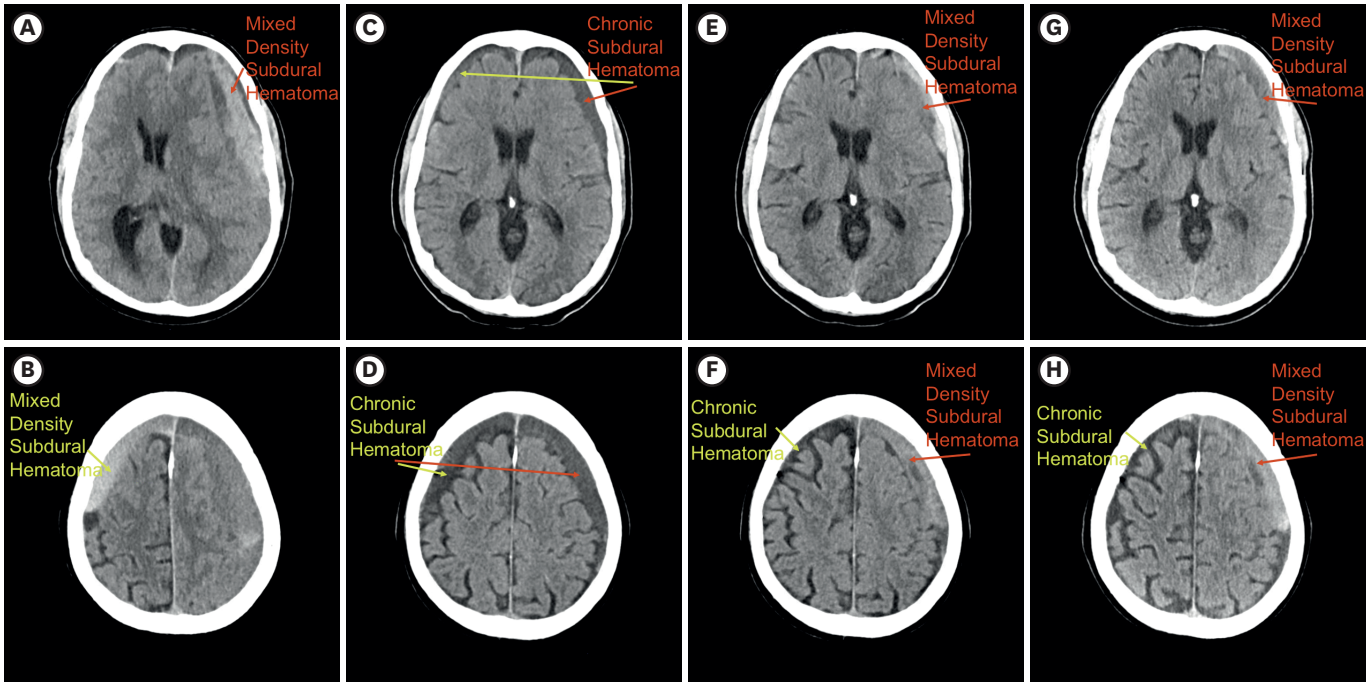

FIGURE 7. Progress of subdural hematomas by CT surveillance.
Initial CT of the head in axial view notes mixed density subdural hematoma with acute and subacute components at the left frontal (A) and right frontal (B) areas. After presentation, the patient underwent left-side craniotomy and evacuation and right-side middle meningeal artery embolization. CT two weeks after embolization noted chronic blood with some decrease of the left-side subdural hematoma (C) and stable size of the right-side subdural hematoma (D). Ten weeks after embolization, acute expansion of the left-side subdural hematomas was noted with a mix of acute and chronic blood (E), however the size of the right-side subdural hematoma continues to decrease and only chronic blood is noted (F). Twelve weeks after embolization, CT of the head noted stable chronic left-sided SDH (G & H), with near resolution of the chronic right-sided SDH (H). All figures show the left side in red and the right side in yellow.
CT: computed tomography.

components. The anticoagulation was reversed with andexanet alfa and the patient was transferred from the ER to another hospital for neurosurgical and neurological assessment that same day. By the time of evaluation at the new hospital, she was not responsive to questions, could not open her eyes and had positive upward Babinski reflex bilaterally. All extremities could withdrawal from nailbed pressure.

On day 1 of the hospitalization, the patient underwent a left craniotomy with evacuation of the SDH. On postoperative exam, the patient was comfortable and interactive with a mild headache. Post-surgical CT noted a leftward midline shift of 7.5 cm. Several hours after the craniotomy and evacuation, the patient underwent left MMA embolization. On day two of the hospitalization, a duplex ultrasound of the lower extremities noted no DVT. The patient was discharged on day three of hospitalization with no neurological deficits. At outpatient follow-up two weeks after the embolization, the patient had no neurological deficits and a CT of the head six weeks after the procedures noted resolution of the hematoma (**FIGURE 6B**). Anticoagulation was not restarted as the patient was planned to discontinue her anticoagulation around that time and the DVT had resolved.

Case 5

The patient is a 70-year-old male with a past medical history significant for hypertension, coronary artery disease, hyperlipidemia, type 2 diabetes, and atrial fibrillation on apixaban. Also, two months prior to presentation, the patient underwent a left-sided tegmen repair for cerebrospinal fluid otorrhea.

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His presentation was related to sudden confusion. Family reported personality changes the night before. On the morning of presentation, the patient was driving from the store in his car with his wife and almost drove off the road twice. By the time he arrived home, the patient was more confused and could not answer basic questions. He was then taken to the emergency room. The neurological exam was intact aside from the confusion. The initial CT of the head noted large mixed density SDH with acute and subacute components of the frontal area with left-to-right midline shift (**FIGURE 7A**) as well as a smaller right-sided mixed density SDH (**FIGURE 7B**). The anticoagulation was reversed with andexanet alfa. On the second day of hospitalization, the patient underwent left-sided craniotomy for evacuation of the hematoma. Follow-up CT after the evacuation noted right-to-left mass effect from the right-sided bleed. On the sixth day of hospitalization, the patient underwent a right-sided MMA embolization with injection of Onyx™ liquid embolic system. A bilateral MMA embolization was originally planned, but the left MMA embolization was not preformed due to the previous tegmen procedure. On day eight, the patient was discharged. Repeat CT two weeks after embolization noted improvement (**FIGURE 7C & D**) and no neurological deficits were noted on exam at one-month follow-up. At follow-up CT ten weeks after embolization, re-expansion of the left-sided SDH with element of both acute and subacute blood were noted (**FIGURE 7E & F**). The right-sided chronic hematoma had improved. No neurological changes were noted. Another CT of the head was done the next day, which was unchanged. Twelve weeks after embolization, CT noted stable chronic left-sided SDH, with near resolution of the chronic right-sided SDH (**FIGURE 7G & H**).

DISCUSSION

MMA embolization is a less invasive method to address SDH than traditional burr hole or craniotomy. Compared to conventional treatment, MMA embolization is associated with fewer SDH recurrences (3.8%–4.3% vs. 21.7%–33.3%)[5,12] and a lower need for re-intervention (0%–12% vs. 8.0%–33.3%)[1,5,12] than conventional treatment for chronic SDH. The surgical complication rate is similar (0%–10% vs. 4.3%–8.7%).[1,5,12] In the cases we present, MMA embolization has been shown to be an alternative to traditional surgery or a supplement to it.

MMA embolization is still a relatively new procedure and the indications and guidelines for this procedure are still actively debated.[10] We encourage further inquiry into expanded indications for acute SDH, as prior studies have only investigated MMA for the treatment of chronic SDH.[9,10] In our search of the literature, the only documented use of MMA embolization in the setting of an acute bleed was to seal off a leaking pseudoaneurysm in a left MMA which had caused an epidural hematoma.[11] The reason for this lack of documented acute SDH cases is likely that MMA embolization does not stop the bleeding from the ruptured bridging veins; it only halts the blood supply to the friable and permeable neovascularizations, which typically occur during the chronic phase, as outlined in the introduction section.

Performing MMA embolization could, however, preemptively neutralize hematoma expansion by preventing the neovascularizations from occurring in the first place. Acute MMA embolization may be a treatment option for patients who are medically unstable and would not be able to tolerate the risk of the hematoma expanding. In this situation, embolizing in the acute setting might be beneficial rather than monitoring the SDH and waiting to embolize in the chronic setting if the SDH expands. This could be especially true

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in patients who need to restart antiplatelet or anticoagulation therapy as soon as possible. If a bleed did expand in the chronic setting, the patient would spend more time off of their medication, which could trigger a bad outcome secondary to that.

In addition to the overall theme in all cases of demonstrating the feasibility of MMA embolization in the acute setting, each individual case has unique aspects.

In case 1, the decision to proceed with MMA embolization through the stent was made to provide the patient MMA embolization over traditional operative methods. In order to do so, a specialized stent-accessing approach had to be done.

A similar method of intra-wall stent access during catheter procedure has been previously reported in the cardiac literature. Caputo et al.[2] noted a procedure to revascularize atherosclerotic blockage of a jailed side branch artery of a stented coronary artery. This involved advancing a guidewire through the stent diamond or articulation site. Once the stent was accessed, a balloon angioplasty was performed with the balloon partially advanced into the side chain artery through the coronary artery.

While subjective and personal clinical experiences may have already acquainted the practicing neurointerventional radiologist with the concept of passing through the side of an existing stent, such as can be done in stent assisted coil embolization of an aneurysm, Y-stenting, MMA embolization as shown in the report, and other procedures, our comprehensive review of the literature does not show that this has been documented in the academic literature. We now present this to assist in the clinical decision-making of the practicing neurointerventional radiologist, providing evidence of the concept in the literature if they encounter a situation that might call for complicated clinical decision-making involving intra-wall access.

We now demonstrate a specialized approach for intra-wall access to an artery jailed by a stent in the neurointerventional setting. This report demonstrates the feasibility of performing this procedure and other neurointerventional procedures in a patient where arterial access is blocked by a previously placed stent.

In case 2, CT shows good progress at one month and near resolution at 2-month follow-up. While it is possible that the bleed may have resolved on it's on without further complications with conservative monitoring, rather than undergoing surgical intervention, the patient had a pre-existing condition with a coiled left anterior communicating artery, which she was taking antiplatelet therapy for. Reducing the risk of hematoma expansion allowed her to re-start antiplatelet therapy as soon as possible, which has been shown to decrease the risk of a thromboembolic event.[3,4,14]

In case 3, we show a case with a pertinent pre-existing condition of atrial fibrillation, which required anticoagulation. The patient had good progressive resolution of the bleed and was able to re-start his warfarin 4 days after the procedure. Reducing the risk of hematoma expansion could reduce the risk of stroke which could have occurred if the bleed had re-expanded in the chronic setting and would have required the patient to stop his warfarin again.

Case 4 is an example of how craniotomy and MMA embolization can be used synergistically. With resolution of the bleed in one month, the recovery was remarkably quick, especially

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considering the severity of the bleed and the associated herniation. This is the first case we show that does not present with purely hyperintense acute blood. The blood is mixed density with a subacute appearance. If we time the start of the hemorrhage to be at or around the onset of symptoms, that would place the initial CT findings at one week after onset, which would be consistent with the clinical portrait. We find the possibility of a silent chronic underlying bleed that later expanded into the acute presentation unlikely as the patient is anticoagulated and if SDH were to occur, which it did, the bleeding would likely continue rapidly from onset until an anticoagulation reversal, which the patient received on presentation. In terms of friable and permeable neovascularization causing the inflammatory process of the bleed, new capillaries have been reported by observation with scanning electron microscopy on the dura matter in a group of SDH patients observed between days 15 and 21, while capillaries and thin-walled sinusoids accompanied by larger diameter blood vessels have been reported in a group of patients on day 40 of SDH.[16] Our timeline places the origination of the bleed to be earlier than 15 days prior to presentation. There is not information on capillary neovascularization earlier than 15 days but we will presume it is minor and will therefore categorize this subacute case still within the preemptive neuralization timeline alongside the acute bleeds as major neovascularizations have not developed and minor neovascularizations are unlikely to have developed at this stage.

In case 5, craniotomy and MMA embolization were also used synergistically. The patient had a bilateral acute SDH and underwent a left-sided craniotomy and evacuation and a right-sided MMA embolization. The left side later re-expanded, but the right side did not. As in case 4, patient 5 presented with mixed density blood. Symptoms occurred as an acute onset the day before presentation. The patient was also anticoagulated. We will therefore treat this case as an early subacute bleed within the preemptive neutralization timeline as we did in case 4.

CONCLUSION

Intra-wall access is a specialized technique to gain neurointerventional access to arteries jailed by a stent which has not been previously published in the neurointerventinal setting. Furthermore, MMA embolization is an established treatment of chronic SDH. We now explore the use of MMA embolization in the acute SDH setting as well.

REFERENCES

1. Ban SP, Hwang G, Byoun HS, Kim T, Lee SU, Bang JS, et al. Middle meningeal artery embolization for chronic subdural hematoma. **Radiology 286:**992-999, 2018 PUBMED | CROSSREF
2. Caputo RP, Chafizadeh ER, Stoler RC, Lopez JJ, Cohen DJ, Kuntz RE, et al. Stent jail: a minimum-security prison. **Am J Cardiol 77:**1226-1229, 1996 PUBMED | CROSSREF
3. Craven CL, Mullins S, Rennie A, Toma AK. Aspirin preventing occlusion after coil migration into the distal anterior cerebral artery. **Cureus 12:**e7797, 2020 PUBMED | CROSSREF
4. Edwards NJ, Jones WH, Sanzgiri A, Corona J, Dannenbaum M, Chen PR. Antiplatelet therapy for the prevention of peri-coiling thromboembolism in high-risk patients with ruptured intracranial aneurysms. **J Neurosurg 127:**1326-1332, 2017 PUBMED | CROSSREF
5. Enriquez-Marulanda A, Gomez-Paz S, Salem MM, Mallick A, Motiei-Langroudi R, Arle JE, et al. Middle meningeal artery embolization versus conventional treatment of chronic subdural hematomas. **Neurosurgery 89:**486-495, 2021 PUBMED | CROSSREF
6. Flaherty ML, Kissela B, Khoury JC, Alwell K, Moomaw CJ, Woo D, et al. Carotid artery stenosis as a cause of stroke. **Neuroepidemiology 40:**36-41, 2013 PUBMED | CROSSREF

https://doi.org/10.13004/kjnt.2024.20.e16

7. Gasecki AP, Eliasziw M, Ferguson GG, Hachinski V, Barnett HJ; North American Symptomatic Carotid Endarterectomy Trial (NASCET) Group. Long-term prognosis and effect of endarterectomy in patients with symptomatic severe carotid stenosis and contralateral carotid stenosis or occlusion: results from NASCET. **J Neurosurg 83:**778-782, 1995 PUBMED | CROSSREF
8. Hohenstein A, Erber R, Schilling L, Weigel R. Increased mRNA expression of VEGF within the hematoma and imbalance of angiopoietin-1 and -2 mRNA within the neomembranes of chronic subdural hematoma. **J Neurotrauma 22:**518-528, 2005 PUBMED | CROSSREF
9. Ironside N, Nguyen C, Do Q, Ugiliweneza B, Chen CJ, Sieg EP, et al. Middle meningeal artery embolization for chronic subdural hematoma: a systematic review and meta-analysis. **J Neurointerv Surg 13:**951-957, 2021 PUBMED | CROSSREF
10. Jumah F, Osama M, Islim AI, Jumah A, Patra DP, Kosty J, et al. Efficacy and safety of middle meningeal artery embolization in the management of refractory or chronic subdural hematomas: a systematic review and meta-analysis. **Acta Neurochir (Wien) 162:**499-507, 2020 PUBMED | CROSSREF
11. Kim DH, Lee JY, Jeon HJ, Cho BM, Park SH, Oh SM. Intraoperative endovascular embolization of middle meningeal artery and a pseudoaneurysm by using N-butyl 2-cyanoacrylate for hemostasis during operation of acute epidural hemorrhage. **Korean J Neurotrauma 11:**167-169, 2015 PUBMED | CROSSREF
12. Kim E. Embolization therapy for refractory hemorrhage in patients with chronic subdural hematomas. **World Neurosurg 101:**520-527, 2017 PUBMED | CROSSREF
13. Link TW, Boddu S, Marcus J, Rapoport BI, Lavi E, Knopman J. Middle meningeal artery embolization as treatment for chronic subdural hematoma: a case series. **Oper Neurosurg (Hagerstown) 14:**556-562, 2018 PUBMED | CROSSREF
14. Mees SMD, van den Bergh WM, Algra A, Rinkel GJE. Antiplatelet therapy for aneurysmal subarachnoid haemorrhage. **Cochrane Database Syst Rev 2007:**CD006184, 2007 PUBMED | CROSSREF
15. Mercado N, Cohen DJ, Spertus JA, Chan PS, House J, Kennedy K, et al. Carotid artery stenting of a contralateral occlusion and in-hospital outcomes: results from the CARE (Carotid Artery Revascularization and Endarterectomy) registry. **JACC Cardiovasc Interv 6:**59-64, 2013 PUBMED | CROSSREF
16. Moskała M, Gościński I, Kałuża J, Polak J, Krupa M, Adamek D, et al. Morphological aspects of the traumatic chronic subdural hematoma capsule: SEM studies. **Microsc Microanal 13:**211-219, 2007 PUBMED | CROSSREF
17. Petty GW, Brown RD Jr, Whisnant JP, Sicks JD, O'Fallon WM, Wiebers DO. Ischemic stroke subtypes: a population-based study of incidence and risk factors. **Stroke 30:**2513-2516, 1999 PUBMED | CROSSREF
18. Schermerhorn ML, Liang P, Eldrup-Jorgensen J, Cronenwett JL, Nolan BW, Kashyap VS, et al. Association of transcarotid artery revascularization vs transfemoral carotid artery stenting with stroke or death among patients with carotid artery stenosis. **JAMA 322:**2313-2322, 2019 PUBMED | CROSSREF
19. Shono T, Inamura T, Morioka T, Matsumoto K, Suzuki SO, Ikezaki K, et al. Vascular endothelial growth factor in chronic subdural haematomas. **J Clin Neurosci 8:**411-415, 2001 PUBMED | CROSSREF
20. Zhu Z, Yu W. Update in the treatment of extracranial atherosclerotic disease for stroke prevention. **Stroke Vasc Neurol 5:**65-70, 2019 PUBMED