Endovascular management of acute epidural hematomas: clinical experience with 80 cases

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OBJECTIVE Small acute epidural hematomas (EDHs) treated conservatively carry a nonmeasurable risk of late enlargement due to middle meningeal artery (MMA) lesions. Patients with EDHs need to stay hospitalized for several days, with neurological supervision and repeated CT scans. In this study, the authors analyzed the safety and efficacy of the embolization of the involved MMA and associated lesions.

METHODS The study group consisted of 80 consecutive patients harboring small- to medium-sized EDHs treated by MMA embolization between January 2010 and December 2014. A literature review cohort was used as a control group.

RESULTS The causes of head injury were falls, traffic-related accidents (including car, motorcycle, and pedestrian vs vehicle accidents), and assaults. The EDH topography was mainly temporal (lateral or pole). Active contrast leaking from the MMA was seen in 57.5%; arteriovenous fistulas between the MMA and diploic veins were seen in 10%; and MMA pseudoaneurysms were found in 13.6% of the cases. Embolizations were performed under local anesthesia in 80% of the cases, with *N*-butyl-2-cyanoacrylate, polyvinyl alcohol particles, or gelatin sponge (or a combination of these), obtaining MMA occlusion and complete resolution of the vascular lesions. All patients underwent follow-up CT scans between 1 and 7 days after the embolization. In the 80 cases in this series, no increase in size of the EDH was observed and the clinical evolution was uneventful, without Glasgow Coma Scale score modification after embolization and with no need for surgical evacuation. In contrast, the control cohort from the literature consisted of 471 patients, 82 (17.4%) of whom shifted from conservative treatment to surgical evacuation.

CONCLUSIONS This study suggests that MMA embolization is a highly effective and safe method to achieve size stabilization in nonsurgically treated acute EDHs.

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KEY WORDS epidural hematoma; traumatic brain injury; middle meningeal artery; pseudoaneurysm; embolization; endovascular treatment; vascular disorders

ACUTE epidural hematoma (EDH) is one of the most dramatic and urgent traumatic brain injuries (TBIs). Patients frequently present with sudden neurological deterioration. In these cases, the most common cause is a tearing of the middle meningeal artery (MMA) near the foramen spinosum. In such circumstances, rapid clinical deterioration requires prompt surgical evacuation before the onset of brain dysfunction.⁴

Small EDHs have not been adequately studied thus far, and their incidence, natural history, clinical relevance,

and ideal management remains to be well established.^{5,7,27} However, late enlargement of previously small hematomas is a well-recognized clinical occurrence.²⁵ One of the most hazardous events in the rupture of intracranial vessels is the relatively slow but continuous expansion of the hematoma and sudden neurological deterioration. Traditionally, these patients need to stay in the hospital for several days or weeks under conservative management, including close neurological supervision and several repeated CT scans.⁹

Recently, endovascular management of intracranial

ABBREVIATIONS AVF = arteriovenous fistula; EDH = epidural hematoma; GCS = Glasgow Coma Scale; MMA = middle meningeal artery; NBCA = *N*-butyl-2-cyanoacrylate; OphA = ophthalmic artery; PVA = polyvinyl alcohol; SAH = subarachnoid hemorrhage; SDH = subdural hematoma; TBI = traumatic brain injury.

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vascular lesions associated with small EDHs has been proposed as a safe and effective treatment option.^{6,20,24,28} In this paper, we describe our experience with the endovascular management of EDHs in 80 consecutive patients (thus far the largest reported series to the best of our knowledge), and compare it with historical cohorts.

Methods

Patient Population

This study included all patients with traumatic EDH consecutively admitted to the Neurotrauma Department of the Hospital das Clínicas da Universidade de São Paulo between January 2010 and December 2014, for whom conservative management was chosen based on standard criteria.⁴ The institutional review board approved the study protocol and all patients provided written informed consent.

We excluded patients who had large hematomas, midline shift, depressed skull fractures, or other injuries related to the EDH; patients without complete imaging studies at the onset (such as those acquired at another institution); patients with coagulopathy; and those without complete clinical data.

Control Group

A historical control group composed of cohorts of patients with small EDHs was used, after a systematic review in the MEDLINE database of papers published between 1986 and 2016. Eligibility criteria included cohort studies of late expansion of previously small EDHs resulting in surgical procedures for evacuation. Only complete text articles written in English were evaluated.

Information Sources

The MEDLINE database and Google Scholar were searched for papers that met the inclusion criteria. An unpublished data quest and a gray search were additionally performed.

The first search in the MEDLINE database was performed as follows: two searches were carried out, one for end-point terms and the other for the patients in the MEDLINE database. These terms were selected by 3 different authors (C.M.A.P., E.G.F., J.G.M.P.C.). Both searches were then additionally matched using the term AND.

Study Selection and Data Collection Process

All the abstracts were analyzed by 3 independent reviewers (C.M.A.P., E.G.F., J.G.M.P.C.). Cohorts were included if they met the eligibility criteria, and the full text version was analyzed. Only patients with the evaluated outcomes (expansion of small EDHs) were included in the final analysis.

Endovascular Procedures

Under local (80% of patients) or general (20% of patients) anesthesia, a femoral sheath was introduced and a 6-Fr guiding catheter was inserted into the external carotid artery on the lesion side. An angiographic study was performed, followed by superselective angiography of the

MMA with a microcatheter, followed by gentle injection of iodine contrast. Careful attention was paid to detect the choroidal blush caused by anastomotic vessels supplying the ophthalmic artery (OphA) via the superior orbital fissure or foramen of Hyrtl and the petrosal branch of the MMA to the territory of the facial nerve.

The microcatheter was then advanced as far as possible in the MMA, stopping just before identifiable contrast extravasation, pseudoaneurysms, arteriovenous fistulas (AVFs), or visible arterial tearing. Next, embolization of the MMA was performed with 20% *N*-butyl-2-cyanoacrylate (NBCA), polyvinyl alcohol (PVA) particles of 250–350 μm or 300–500 μm , gelatin sponge (Gelfoam), or a combination of the 3, until a cessation of the contrast material leakage or stopping of the parent vessel (distal MMA) flow had been observed, or microcatheter reflux occurred. In cases of pseudoaneurysms, the proximal segment, the lumen, and the distal segment were occluded with NBCA (Fig. 1).

After the withdrawal of the microcatheter, an angiographic follow-up series, performed through the guiding catheter, showed complete resolution of the vascular lesion in all but 3 cases. In these cases, a new microcatheter was then used until complete occlusion of any additional abnormal MMA branch occurred.

Results

In the period of the study, 80 patients were treated conservatively and were included. Seventy-two patients were male and 8 were female (age range 12–72 years; mean age 39.8 years, median age 38 years). All patients had small EDHs in regions corresponding to bleeding from branches of the MMA. Most of the patients (76.25%) presented with a score of 14 or 15 on the Glasgow Coma Scale (GCS) on admission (Table 1). No neurological deficits related to the EDHs were observed.

The causes of head injury were mainly falls, traffic-related accidents (including car, motorcycle, and pedestrian vs vehicle accidents), and assaults (Table 2). Cranial fractures were present in 78.7% of the patients. Other associated intracranial injuries seen on CT scans are summarized in Table 3. Fifty-seven (71.25%) of the EDHs were located in the temporal region. The mean distance from the foramen spinosum (recognized by the sharp turn of the MMA along the floor of the middle cranial fossa) and the hematoma was 38 mm. Forty-one (51.2%) were on the left side. The average thickness was 8.65 mm in the lateral temporal region, and it reached 10.5 mm in the temporal pole. Some of the patients in our series had previously undergone urgent craniotomy for contralateral lesions. The presence of contralateral hematoma, controlled with embolization, was also considered in these cases (Fig. 2).

The most frequent site of EDH was the temporal pole or middle cranial fossa. The distal branches of the MMA can rupture and cause hematomas in the frontal or parietal areas. Real-time bleeding may be identified as contrast extravasation on the angiogram (Fig. 3).^{9,15}

Angiographic findings are summarized in Table 4. The active contrast leaking from the MMA was the most important finding. It was the only feature in 46 patients

FIG. 1. Axial CT images of EDH with related fracture (**A and B**), and angiographic studies showing active contrast extravasation (**C**) and AVF (**D**—note the diploic vein). Angiographic view after embolization (**E**), with resolution of both AVF and contrast extravasation.

(57.5%), but it was also found to be associated with most of the other lesions (as with pseudoaneurysms and AVFs). An AVF was seen on angiographic images in 8 patients (10%), manifesting as early filling of diploic veins juxtaposed to the MMA. Pseudoaneurysms of the MMA were seen in 13.6% (Fig. 4).

Embolizations were performed with PVA particles and Gelfoam in 60 patients (75%). In 17 patients (21.2%), PVA alone was used. Combined microcoils, NBCA, and Gelfoam were used in 3 patients. All patients had follow-up CT scans between 1 and 7 days after the embolization. In the 80 cases in our study, no increase in size of the EDH was observed. The clinical evolution of the 80 cases was uneventful, without modification in GCS scores after embolization. Outpatient clinical data and follow-up CT

scans obtained between 1 and 3 months were available in 76 patients, all of them without any clinical or radiological deterioration.

Our literature-based control cohort consisted of a total of 471 patients, 82 (17.4%) of whom were shifted to surgical evacuation. Given that the odds ratio is the ratio of the odds of surgical evacuation indication after embolization divided by the odds with the control group (literature cohort of conservatively treated patients), it was impossible to calculate it because the former value was zero. Table 5 summarizes the control cohort, which includes the larger published series of patients with conservatively managed EDH.^{1,2,5,11,12,14,19,27}

Discussion

Patients with an EDH volume of more than 30 ml, thickness of more than 15 mm, a midline shift greater than

TABLE 1. Summary of clinical data in 80 patients with EDH

Features	Value	%
GCS score on admission		
14–15	61	76.2
12–13	10	12.5
≤11	9	11.2
Total	80	100.0
Hospitalization duration (days)		
Median	7	
Mean	10.5	
Admission to femoral puncture (days)		
Median	4	
Mean	4.6	

TABLE 2. Causes of head injury in 80 patients with EDH

Trauma Mechanism	No.	%
Fall	26	32.5
Vehicle vs pedestrian	15	18.7
Car accidents	12	15.0
Motorcycle accidents	9	11.2
Assault	9	11.2
Other/unknown*	9	11.2
Total	80	100.0

* Buried alive by landslide or trench collapse, cranial collisions while playing football.

TABLE 3. Cranial lesions and topography in 80 patients with EDH

CT Findings	Value (%)
Associated lesions	
Fracture	63 (78.75)
Contusion & fracture	7 (8.75)
Fracture & tSAH	4 (5.0)
Contusion & tSAH	1 (1.2)
Contusion	1 (1.2)
Pneumocephalus	1 (1.2)
No associated lesions	3 (3.7)
EDH topography	
Temporal—lateral	29 (36.2)
Temporal pole	28 (35.0)
Frontal	11 (13.7)
Parietal	9 (11.2)
Frontotemporal	3 (3.7)

tSAH = traumatic SAH.

5 mm, or clinical deterioration are the usual candidates for surgical treatment.⁴

The role of nonoperative treatment of acute EDHs is still not well established in neurosurgical practice. However, patients who present in good clinical condition, without deterioration in GCS score, pupillary dilation, or focal neurological deficits, and who harbor EDHs with no compressive effects generally remain under close neurological observation and undergo repeated CT scans. These patients should be admitted to the intensive care unit and monitored for changes in neurological status and vital signs.¹⁴ This routine, although completely justifiable, raises considerably the economic costs and radiation exposure and carries a nonmeasured risk of sudden neurological decompensation.

There is no consensus in the literature regarding the required length of expectant management. In a series of 252 consecutive patients with traumatic EDH in which 160 patients were initially treated conservatively, enlargement of the hematoma occurred in 37 patients (23%), with a mean enlargement of 7 mm and a mean time to enlargement of 8 hours after the trauma.²⁷ In another series, 64.9% of the

FIG. 3. Angiograms obtained with microcatheterization of the MMA showing contrast extravasation in 2 cases.

patients had enlargement of their EDHs.²⁵ Enlargement of an EDH does not necessarily indicate surgery; nonetheless, in a prospective study of patients who had a small EDH diagnosed within 24 hours of trauma and whose injuries were managed expectantly, 32% subsequently required craniotomy for evacuation of the hematoma within 1–10 days after the trauma.¹⁴ In another series of 125 patients with EDH treated conservatively, 11.2% required delayed surgery.²

Although the deterioration can be the result of progressive brain swelling and ischemia, rebleeding or continued hemorrhage remains a concern in nonoperative management. In our series, we demonstrated that this issue may be reduced or even eliminated with endovascular embolization. Laboratory investigations with electron microscopy have shown that the dura mater has 5 layers. The MMA runs in the vascular layer of the dura, which is covered outside by the outer dural border layer, which is 2 μ m thick, and inside by the dural border cell layer, which is 8 μ m thick.²³ This may be the reason why the most common presentation of tearing of the MMA is bleeding into the epidural space caused by a skull fracture, resulting in the separation of dura mater and bone. Acceleration-induced shear may explain the occurrence of EDH without visible skull fracture. Nevertheless, it is possible to find subdu-

TABLE 4. Angiographic findings in 80 patients with EDH

Angiographic Finding	Value (%)
Active contrast extravasation	46 (57.5)
MMA wall irregularities	8 (10.0)
Acute contrast extravasation & pseudoaneurysm	7 (8.7)
Acute contrast extravasation & AVF	5 (6.2)
Pseudoaneurysm	3 (3.7)
AVF	2 (2.5)
Pseudoaneurysm & AVF	1 (1.2)
Choroidal blush	1 (1.2)
MMA wall irregularities & ICA aneurysm	1 (1.2)
Normal	6 (7.5)
Total	80

ICA = internal carotid artery.

FIG. 2. Left: Axial CT showing bilateral trauma, surgically treated on the right side, with laminar EDH on the left side. Right: Angiogram obtained with microcatheterization of the MMA showing acute contrast extravasation related to the left-sided EDH.

FIG. 4. Angiogram showing MMA pseudoaneurysm.

ral hematomas (SDHs) or even subarachnoid hemorrhage (SAH) and intracerebral hematomas.^{22,32}

Rupture of the MMA may lead to the formation of traumatic aneurysms, most of them believed to be pseudoaneurysms, whose walls are histologically composed of fibrous connective tissue of the surrounding anatomical structures. Normal arterial layers are absent in these instances.³ Angiographically, these “false” aneurysms usually fill late in the arterial phase, opacify less, and empty more slowly than saccular aneurysms. “True” aneurysms do occur in the MMA, with the same histological characteristics as aneurysms of cerebral arteries, both in the context of trauma and in diseases that put increased hemodynamic stress on the wall of the artery, such as Paget’s disease, dural arteriovenous malformations, moyamoya disease, and meningiomas.¹⁸

Pseudoaneurysms of the MMA, although rare, may produce abrupt neurological deterioration due to rapid enlargement of an EDH, after a 3- to 30-day interval.^{14,30} Nowadays, these vascular lesions may be missed due to the widespread use of CT scans in the evaluation of trauma. A hypodense image within an EDH associated with

a basal skull fracture, or a hypodense nodule with strong and homogeneous enhancement may raise the suspicion of a pseudoaneurysm.³¹

Although there is no justification to perform routine cerebral angiograms, neurosurgeons should be aware of possible pseudoaneurysms developing late in the context of skull fractures crossing the MMA. One may consider 3D CT angiography in selected cases.^{21,31} Suspect lesions should be confirmed with angiography, followed by treatment with embolization. We don’t see any reason to postpone treatment waiting for spontaneous resolution²⁶ in the context of an MMA pseudoaneurysm. Interestingly, the first reports of MMA embolization related to traumatic lesions concern the treatment of refractory chronic SDHs.^{10,16,29}

The first published series of endovascular treatment of acute EDH was described by Suzuki et al. in 2004; they reported successful embolization in 9 patients with EDHs and associated lesions.²⁸ The EDHs were followed conservatively without surgical intervention, and eventually disappeared within an average of 18 days. Surgical intervention was performed in 3 patients with other lesions (contusions and SDHs). As noted by Ross,²⁴ this first study was published in a radiological journal. This and the fact that anticoagulation was used in interventional procedures in the setting of TBI are perhaps the reasons for the lack of attention to the endovascular approach.

Misaki et al.¹⁷ described a case of a 44-year-old woman with intractable otorrhagia and coexisting EDH. The treatment was aimed at the ear bleeding, but stabilization and further resolution of the EDH was observed on serial CT scans. Ross²⁴ treated 1 postoperative EDH with MMA embolization. The patient originally had an SDH and developed an EDH (which was drained in a second operation) but rebled and was finally treated successfully with embolization. Ohshima et al.²⁰ described the combined treatment of a large EDH with bur hole endoscopic evacuation and MMA embolization in a 74-year-old patient in poor medical condition. Kim et al.¹³ described the usefulness of intraoperative embolization of an MMA pseudoaneurysm for hemostasis as a desperate measure to control acute, life-threatening, uncontrollable bleeding.

De Andrade et al.⁶ described the largest series so far, with 24 patients harboring small EDHs, all associated

TABLE 5. Literature series of 82 patients with acute EDH who initially received conservative management and eventually needed craniotomy

Authors & Year	No. of Patients	No. (%) w/ Shift to Surgical Evacuation	Mean Age (yrs)	EDH Avg Thickness (mm)
Knuckey et al., 1989	22	7 (32)	27	NA
Chen et al., 1993	74	14 (19)	24.3	15
Sullivan et al., 1999	160	37 (23)	23	10
Offner et al., 2006	54	7 (13)	27	10.1
Balmer et al., 2006	13	1 (7.7)	9.3	19
Jamous et al., 2009	6	0 (0)	11	16
Khan et al., 2014	17	2 (11.8)	9.8	16
Basamh et al., 2016	125	14 (11.2)	39.1	10.2

Avg = average; NA = not available.

FIG. 5. Supraselective MMA angiography showing the choroidal blush and contrast extravasation with pseudoaneurysm distally (A). The microcatheter was gently navigated to a position distal to the anastomosis (B), and safe occlusion of the MMA was performed with gentle Gelfoam injection (no reflux was permitted) (C).

with cranial fractures. They noted active contrast extravasation in 70.9%, pseudoaneurysms in 29.1%, and 2 patients (8.3%) with AVF.

The embolization procedure, although generally fast and straightforward, must be conducted with extreme caution when dealing with the proximal MMA. In cases in which the meningo-OphA (a remnant of the embryonal stapodial artery) is present, the entire supply to the distal OphA, including the central retinal artery, is the MMA. In this setting, even proximal occlusion of the MMA is dangerous. One may suspect this variation when the OphA is not visualized from the internal carotid artery injection and a choroidal blush is seen on the external carotid artery injection.⁸ In 1 case in our series (Fig. 5), the choroidal blush precluded safe embolization with PVA particles. The strategy in this case was to place the microcatheter as far as possible into the MMA, and the use of Gelfoam as the embolization agent. The final result shows the preservation of the choroidal blush and complete occlusion of the torn distal meningeal artery.

Although we had no control cohort in our study, we performed a review of the literature for historical comparison, and found an incidence of 17.4% of delayed surgical procedures in 471 patients initially admitted to conservative treatment, compared with no need for surgical evacuation in our series. Given that AVFs were seen in 8 of our 80 cases, another theoretical benefit of the embolization would be the prevention of a rare but possible symptomatic osteodural AVF in the future in these patients.

Limitations of the Study

This study presents some limitations. First, it does not address the late effects of radiation required to embolize the lesions. However, the radiation required to obtain multiple CT scans in the conservatively managed group may be higher than that in the embolized one. Second, economic issues were not studied. Indeed, health costs are not homogeneous among different countries, and what is cost-effective in one country may not be in others. Third, the selection of a historical cohort as a control group may introduce selection and outcome bias, whose effects are difficult to predict and discuss. Publication bias is another issue associated with historical controls from the literature. To better clarify these issues, a randomized trial is under

way in our institution to compare these groups of patients directly and to identify differences in the economic issues. Nonetheless, this study has demonstrated that endovascular embolization of MMA and its branches in patients with small EDHs is a simple, safe, and effective procedure to prevent late hematoma expansion.

Conclusions

Epidural hematomas in patients with cranial fractures that tear dural arteries distal to the foramen spinosum are more prone to develop late rebleeding causing EDH enlargement. Such intracranial vessel wall injury may cause unstable vascular injuries. This may warrant the embolization of any abnormality found on angiographic studies in these patients, provided that open clot evacuation is unnecessary.

To the best of our knowledge, this is the largest series of endovascular treatment of nonsurgically managed EDHs so far in the literature. It has demonstrated that embolization is safe, with no method-related complications, and highly effective to prevent mortality and morbidity associated with late expansion of small EDHs, when compared with a historical cohort.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Peres, Caldas. Acquisition of data: Peres, Caldas, Puglia, da Silva. Analysis and interpretation of data: Peres, Caldas, Figueiredo. Drafting the article: Peres, Figueiredo. Critically revising the article: Caldas, Puglia, Figueiredo. Reviewed submitted version of manuscript: Peres, de Andrade. Approved the final version of the manuscript on behalf of all authors: Peres. Statistical analysis: Peres. Administrative/technical/material support: Caldas, de Andrade, Teixeira. Study supervision: Caldas, de Andrade, Teixeira, Figueiredo.

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