

Review

Clinical importance of the middle meningeal artery: A review of the literature

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Abstract

The middle meningeal artery (MMA) is a very important artery in neurosurgery. Many diseases, including dural arteriovenous fistula (DAVF), pseudoaneurysm, true aneurysm, traumatic arteriovenous fistula (AVF), moyamoya disease (MMD), recurrent chronic subdural hematoma (CSDH), migraine and meningioma, can involve the MMA. In these diseases, the lesions occur in either the MMA itself and treatment is necessary, or the MMA is used as the pathway to treat the lesions; therefore, the MMA is very important to the development and treatment of a variety of neurosurgical diseases. However, no systematic review describing the importance of MMA has been published. In this study, we used the PUBMED database to perform a review of the literature on the MMA to increase our understanding of its role in neurosurgery. After performing this review, we found that the MMA was commonly used to access DAVFs and meningiomas. Pseudoaneurysms and true aneurysms in the MMA can be effectively treated via endovascular or surgical removal. In MMD, the MMA plays a very important role in the development of collateral circulation and indirect revascularization. For recurrent CSDHs, after burr hole irrigation and drainage have failed, MMA embolization may be attempted. The MMA can also contribute to the occurrence and treatment of migraines. Because the ophthalmic artery can ectopically originate from the MMA, caution must be taken to avoid causing damage to the MMA during operations.

Key words: Middle meningeal artery; Clinical importance; Review

1. Introduction

The middle meningeal artery (MMA) is a very peculiar branch of the external carotid artery. The MMA enters the dura, is embedded in the groove of the inner skull face and follows a straight and fixed course [1]; therefore, the MMA is frequently used as a pathway for endovascular embolization for conditions such as dural arteriovenous fistula (DAVF) and meningioma [2]. Moreover, false and true aneurysms and traumatic arteriovenous fistula (AVF) can occur in the MMA [3, 4]. The MMA also plays an important role in the development of and treatments for moyamoya disease (MMD) and recurrent chronic subdural hematoma (CSDH) [2, 5]. Additionally, migraines have a relationship with the MMA, and sometimes during cranial surgery, the MMA needs to be protected to avoid visual loss resulting from

damage to an anastomosis between the MMA and ophthalmic artery [6]. Hence, the MMA is a very important vessel in neurosurgery. However, there is currently no overall systematic review of the importance of the MMA. In this paper, we present a review to increase the understanding of the role of the MMA in neurosurgery.

2. Pathway for the embolization of DAVFs

DAVFs are abnormal connections within the dura. The arterial suppliers of DAVFs are usually the branches of dural arteries [7-9]. The MMA is the most commonly involved feeding artery for DAVFs because it is the largest meningeal feeder [10].

Moreover, the MMA has a unique characteristic in that it is straight and fixed between the dura. The MMA is therefore commonly used as the means to access a DAVF [11]. All MMA branches can be used to access a DAVF. When a DAVF occurs, it often becomes thicker than normal as a result of hemodynamic stress [12]. The intracranial branches include the petrous branch, petrosquamosal branch, temporal branch, parietal branch, frontal branch, sphenoid branch, orbital branch, and cavernous branch. The branches of the MMA supply the dura over the middle and anterior fossa [13, 14]. The MMA branch that is involved in a DAVF can vary according to the location of the DAVF.

For instance, DAVFs of the anterior cranial fossa are supplied by the frontal branch of the MMA [15], while DAVFs of the middle cranial fossa and cavernous sinus are supplied by the sphenoid branch, cavernous branch, or temporal branch of the MMA [16, 17]. DAVFs of the superior sagittal sinus are supplied by the temporal branch, parietal branch, and frontal branch [18]. DAVFs of the transverse, sigmoid sinus and tentorium are supplied by the petrosquamosal branch, temporal branch, or parietal branch [19, 20]. Sometimes, when the branches of the MMA that are involved in the DAVF are not the main feeding arteries, the MMA may still be chosen as the route to access the DAVF [21].

The outcome of DAVFs treated using a transarterial approach via the MMA is satisfactory. For example, in 2016, Kim et al. reported that in sixty-eight Onyx embolizations that were performed in 55 patients with non-cavernous DAVFs, the MMA was the arterial pedicle that was most frequently used for embolization (in 58 cases), and the overall favorable treatment outcome was 76.4% [22]. Additionally, in 2016, Griessenauer et al. reported 19 patients who underwent transarterial embolization through the MMA after embolization of other arterial feeders had failed, with a success rate of 92.9% [10].

Sometimes, a transarterial treatment can be restricted by tortuous access to the MMA. In these cases, direct access to the MMA and embolization remain feasible. For instance, in 2015, Lin et al. reported a Borden III DAVF in which attempts to access the endovascular site using conventional transvenous and transarterial routes were unsuccessful, and the major MMA feeder was subsequently accessed directly after a temporal craniotomy was performed. Onyx embolization was performed, and complete occlusion was achieved [23]. In another instance, in 2015, Oh et al. reported on a DAVF that involved the superior sagittal sinus. First, an Onyx embolization performed through a tortuous MMA was not successful. The second procedure was

performed through the MMA, which was accessed via a direct puncture following a craniotomy. A microcatheter was then inserted near the fistula, and complete obliteration was achieved [24]. These results show that a combined surgical-endovascular technique can be an effective treatment option for DAVFs that are complicated by a lack of accessibility to an MMA approach.

The MMA shares wide anastomoses with other external carotid artery branches, which are referred to as “dangerous anastomoses” [25, 26]. For instance, the petrous branch of the MMA supplies cranial nerve VII and has anastomoses with the ascending pharyngeal artery. Its sphenoid branch can enter the orbit via the superior orbital fissure to form an anastomosis with a recurrent branch of the ophthalmic artery. The cavernous sinus branch can form an anastomosis with the interior lateral trunk of the internal carotid artery, and the terminal territory of the frontal branch that supplies the anterior falx can anastomose with the anterior ethmoidal artery [25, 27]. Hence, when DAVFs are embolized using an MMA approach, some complications can occur as a result of such “dangerous anastomoses”.

For instance, in 2013, Nyberg et al. reported two patients who were treated for a DAVF and an AVM near the skull base that received a heavily parasitized supply from branches of the external carotid artery. Transarterial embolization resulted in transient cranial neuropathies, including lower facial nerve palsy in two and trigeminal nerve mandibular segment neuralgia in one of the cases. The MMA and internal maxillary arteries are common pathways that are used, and these “dangerous anastomoses” may have been the cause of cranial nerve defects in these patients [28]. In another instance, in 2000, Wang et al. reported a patient with a DAVF in the cavernous sinus that was supplied by the MMA. A choroidal infarction was observed after an embolization of the DAVF was performed through the MMA, and it was caused by the migration of the embolization agent from the MMA to the ophthalmic artery [29]. Hence, more caution should be taken when evaluating these variant collaterals, and careful angiographic monitoring and slow injection of embolization materials may help to prevent these complications.

3. AVFs of MMA

AVFs of the MMA are uncommon lesions. In these cases, the fistula can communicate between the MMA and the accompanying middle meningeal vein, diploic vein, cavernous, or sphenoparietal, or greater petrosal dural venous sinuses, or with a cortical vein [30-33]. Many causes can lead to an AVF developing in the MMA, with trauma being the most common

one. Anatomically, the MMA runs along the outer surface of the dura and is accompanied by paired veins. A tear in the arterial wall resulting from a skull fracture can cause a traumatic AVF in the MMA [34]. Craniotomy is another important iatrogenic cause because it results in the separation of the dura mater and bone. For instance, in 1990, Tsutsumi et al. reported a case of a postoperative AVF of the MMA that occurred after a craniotomy was performed during an aneurysmal surgery in which the pterional approach was used [35]. In another instance, in 1984, Inagawa et al. reported an unusual AVF of the MMA that resulted from a three-point fixation with a skull clamp that was applied to stabilize the head during surgery for an anterior communicating artery aneurysm. In this case, the reason was that the skull clamp penetrated the skull and caused the dura and skull to separate [36].

In addition to trauma and craniotomy, endovascular injury during the interventional manipulation is also an iatrogenic factor. For instance, in 1997, Terada et al. reported a 73-year-old female who developed an AVF of the MMA during embolization of a falx meningioma. The cause of this complication was thought to be a perforation in the sharp bend of the sphenoidal portion of the MMA by the microwire during catheterization [37]. Histologically, medial defects in the MMA have been observed at its branching points, similar to other cerebral arteries. Pathological processes, such as atherosclerosis, may also decrease the elasticity of the MMA and predispose it to the formation of an AVF. Hence, AVFs of the MMA are likely to form as a result of interactions between congenital and acquired predispositions [38, 39]. In addition, some etiologies of AVFs of the MMA have not been explained. For instance, in 2009, Takeuchi et al. reported a case of a traumatic AVF in the MMA on the side of the head that was opposite to the injury [40].

Because the angioarchitectures of AVFs in the MMA are complex, in 1981, Freckmann et al. classified AVFs of the MMA based on venous drainage, as viewed on angiography, into six types: I. cases showing drainage via the middle meningeal veins to the pterygoid plexus that are characterized by a tramtrack appearance of the meningeal vessels, II. cases showing drainage via the sphenoparietal sinus or other meningeal veins into the superior sagittal sinus, III. cases showing drainage via the sphenoparietal sinus into the cavernous sinus, IV. cases showing drainage via the middle meningeal veins and superior petrosal sinus into the cavernous sinus/basilar plexus, V. cases showing drainage via the diploic veins, and VI. cases showing drainage via a bridging (cortical) vein into the superior sagittal

sinus [32]. The presentation of patients with each type of AVF of the MMA differed according to discrepancies that were visible in the angioarchitecture of the AVF.

Some patients with AVFs of the MMA without cortical vein drainage or with drainage to the cavernous sinus, which can include type I, II, and V patients, may have no symptoms [35, 36]. After conservative treatment, these types of AVFs of the MMA may disappear [35]. However, most AVFs of the MMA have clinical symptoms. For instance, in type V cases, retrograde leptomeningeal drainage towards the cortical veins can cause intracranial hemorrhage in that the high-flow fistula can lead to venous hypertension in the superficial middle cerebral vein, resulting in intracranial hemorrhage [41]. In type III and IV AVFs of the MMA, cavernous sinus syndrome is often observed. For instance, in 2009, Unterhofer et al. reported a traumatic AVF between the MMA and the sphenoparietal sinus that drained into the cavernous sinus in a patient who presented with pulsating exophthalmos and chemosis [30].

In most cases, interventions should be considered for AVFs of the MMA. Although some cases of spontaneous closure have been reported, this occurs primarily in cases that suffered minor head injury, resulting in AVFs of the MMA that are low-flow lesions or that are secondary to thrombosis at the site of the fistula [42-44]. Treatments for AVFs of the MMA include surgical resection and endovascular embolization, and good outcomes are often obtained. For instance, in 2008, Rennert et al. reported a traumatic high flow AVF that involved the MMA and facial veins in which complete endovascular embolization was performed using a transarterial approach with microparticles and an electrolytically detachable coil, resulting in a good prognosis [45]. When an AVF of the MMA is superficial and easily exposed, especially in cases associated with intracranial hemorrhage, surgical removal remains a good option [41].

Some patients experience a failed endovascular embolization, and in these patients, surgical resection is the last resort. For instance, in 2009, Sakata et al. reported a 48-year-old woman who suffered head trauma and presented with an acute epidural hematoma caused by a linear fracture of the right temporal bone across the middle meningeal groove. After 15 years, the patient developed an AVF of the MMA. The case was classified as AVF type VI, and the feeding artery of the AVF of the MMA was first embolized using coils. However, the patient hemorrhaged, and an emergent decompressive craniectomy and evacuation of the hematoma was

therefore performed. The dilated superficial sylvian vein was removed with the ruptured venous aneurysm [46].

4. Aneurysms of the MMA

Aneurysms of the MMA are uncommon and can be divided into pseudoaneurysms and true aneurysms [47]. Pseudoaneurysms of the MMA are usually associated with a skull fracture in the temporal region that causes a small tear in the arterial wall, which is then blocked by a clot during the acute phase before recanalizing to form a false lumen. However, pseudoaneurysms can be located in the weakest part of the vessel wall and they may not necessarily lie beneath the fracture line [48]. In addition to the trauma, iatrogenic injury is also an important factor. For instance, in 2015, Grandhi et al. reported an iatrogenic pseudoaneurysm of the MMA that occurred after external ventricular drain placement [3]. Pseudoaneurysms often show an absence of a neck and an irregular shape, which result in delayed and very slow filling and emptying on angiogram [49].

A true aneurysm in the MMA resembles a normal cerebral aneurysm, often originates from its branches, and is usually associated with increased hemodynamic stress or a pathological condition in the MMA [50]. Many diseases, such as dural AVFs, can induce such an increase in blood flow and hemodynamic stress in a dural AVF, which can cause multiple aneurysms in the MMA [51]. In MMD, collateral circulation develops in the MMA, and the hemodynamic stress subsequently increases to higher than normal levels, potentially resulting in a MMA aneurysm [52]. Some studies found the MMA shared similar pathological changes with the intracranial arteries in MMD, and perhaps this is the reason for MMA true aneurysm [53, 54]. In meningioma, the MMA supplies higher than normal blood flow, increasing the risk of an aneurysm [55, 56]. In addition to hemodynamic stress, other pathological conditions of the MMA can also result in aneurysms. These include Paget's disease, hypertension [57], and type 2 neurofibromatosis [58]. Moreover, histologically, medial defects can also occur in the MMA [38, 39].

These pseudoaneurysms tend to gradually enlarge, resulting in a delayed rupture, clinical deterioration, and acute or delayed epidural hematoma; however, they are also occasionally associated with subdural or intracerebral hemorrhage [59]. In addition to extradural hematoma, other puzzling hemorrhage patterns have been observed. One of these involves subdural hemorrhage. In 1992, Aoki et al. reported on a rare case that presented with recurrent acute subdural hematoma that developed 29

days after head trauma. A second operation revealed a large aneurysm-like mass lesion in the subdural space at the base of the middle cranial fossa. This pseudoaneurysm was considered to originate from the MMA [60]. When pseudoaneurysms embed in the brain parenchyma, a rupture in the pseudoaneurysm can cause an intracerebral hematoma [61]. For instance, Jussen et al. [62], Paiva et al. [63] and Singh et al. [64] reported patients who presented with this type of pathology. True MMA aneurysms have manifestations that are similar to those of traumatic pseudoaneurysms. In 2001, Kobata et al. reviewed the literature to identify all published cases of true MMA aneurysms before 2001. The authors found that these aneurysms presented with incidental unruptured aneurysms and epidural, subdural, intracranial and intraventricular hemorrhage [65].

Because there is a risk of secondary rupture, most pseudoaneurysms and true ruptured aneurysms require treatment, although some cases of spontaneous thrombosis have been reported [47, 66]. The therapeutic methods used to treat these conditions include endovascular embolization and surgical resection, assisted by hematoma evacuation when necessary. For instance, in 2014, Paiva et al. studied 11 patients with epidural hematoma, 3 of which had pseudoaneurysms. After embolization, a good outcome was achieved in these patients [67]. In another instance, in 2012, Jussen et al. reported two such cases that underwent endovascular treatment. The authors reviewed the published cases and found that most obtained a good outcome [62]. Moreover, surgically resecting a MMA aneurysm with hematoma evacuation can result in a good recovery [68, 69]. For instance, in 2001, Kobata et al. reported a 77-year-old woman with a large subcortical hematoma that was associated with subarachnoid hemorrhage; an emergent surgery confirmed that the hematoma resulted from a ruptured true MMA aneurysm. The aneurysm had coagulated, and the hematoma was evacuated, resulting in a satisfying outcome [65].

In addition, true ruptured aneurysms of the MMA have a higher rate of rupture because they originate from increased hemodynamic stress or a pathological condition of the MMA. For instance, in 2010, Park et al. reported a case of MMD that was associated with a subarachnoid hemorrhage and intracerebral hematoma that resulted from the repeated rupture of a MMA aneurysm. The aneurysm had progressively enlarged over a period of 1 month and was treated using middle meningeal artery embolization [70]. Because an MMA aneurysm can re-rupture, in some trauma cases, it not safe to remove only the hematoma. Because these patients could

suffer another hemorrhage, it is necessary to examine the MMA to determine whether the temporal fracture crossed it.

5. MMA Contribution in MMD

MMD is an uncommon disease that is characterized by the progressive occlusion of the terminal portion of the internal carotid artery and its main branches within the circle of Willis. This occlusion results in the formation of a fine vascular network at the base of the brain [71]. In MMD, there is also a simultaneous development of a collateral circulation. MMD cases can be classified as one of three types, including vault MMD, ethmoidal MMD, and MMD of the basal ganglia and thalamus, which are determined by the location of the collateral circulation. The MMA and its dural branches have been shown to contribute to the collateral blood supply in the MMA in both vault and ethmoidal MMD. In vault MMD, the MMA can penetrate the dura to anastomose with pial arteries. In ethmoidal MMD, the frontal branch of the MMA may anastomose with the ethmoidal artery to provide blood to the anterior base of the brain, and the anterior branch of the MMA may provide collateral blood flow to the anterior cerebral artery territory via the falx [72, 73].

When the MMA contributes to MMD, the MMA can become stronger and stronger. For instance, in 2015, Matsukawa et al. found that on brain CT, the foramen spinosum and MMA were larger in MMD, which demonstrated that the MMA is very important to MMD collateral circulation [74]. In 2005, Honda et al. used magnetic resonance angiography to evaluate external carotid artery tributaries in MMD and found MMA showed changes that were similar to those in the Matsukawa et al. study [75]. Theoretically, the collateral circulation of the MMA may develop spontaneously, but more often, the revascularization of the MMA is not enough because the MMA cannot easily penetrate the dura to anastomose with the pial arteries on the brain surface [76]. In these cases, an operation is necessary. The indirect revascularization of the MMA using encephalo-duro-arterio-synangiosis (EDAS) and burr holes resulted in the long-term resolution of ischemic and hemorrhagic manifestations in 95% of adults and children. The MMA appeared to have significantly contributed to revascularization on follow-up angiograms, which showed that it had achieved an increase in size and neovascularity comparable to that of the superior temporal artery [77]. Encephalo-duro-arterio-myosynangiosis (EDAMS) is also a widely used and effective technique [78, 79].

During EDAS and EDAMS, it is necessary to

perform a dural inversion procedure [80]. This technique was described in detail by Dauser et al. in 1997 [81]. Split duro-encephalo-synangiosis is considered to be effective in pediatric cases of MMD [82]. However, a simple encephalo-arterio-synangiosis (EAS) without MMA involvement is not always effective [83]. Although burr holes are simple to apply, they can help the MMA to anastomose with pial arteries. For instance, the burr hole could penetrate the dura, providing an opportunity for the MMA outside the dura to establish collateral circulation. In 2014, McLaughlin et al. found that burr hole surgery is an important tool for surgeons who treat children and adults with MMD because it allows revascularization to be tailored to the patient [84]. Hence, during MMD reconstructive operations, the MMA must often be protected [5].

In a study of MMD revascularization, King et al. found that the contributions of the MMA to revascularization in patients who underwent pial synangiosis for moyamoya syndrome were significant and may have frequently exceeded the contribution of the superior temporal artery when the surgery was performed to preserve the dural vasculature and dural inversion [85]. Recently, some new techniques have also been developed for optimal revascularization from the MMA. For instance, in 2013, McLaughlin et al. emphasized the importance of recognizing the 3 major layers of the dura and described a technique involving dural splitting at the locus minoris resistentiae between the dura mater's vascular (middle) layer and its internal median layer and the application of the dura's vascular layer to the surface of the brain after opening the arachnoid. This technique was designed to optimize surgery for dural-pial synangiosis related to MMA branches [86].

6. MMA Embolization in CSDH

CSDH describes the collection of old blood and the breakdown of its products between the brain surface and the dura. This condition occurs frequently in elderly patients and it is associated with acquired predisposing factors, such as trauma that causes the rupture of the bridging vein, the use of antiplatelets, coagulopathy resulting from liver cirrhosis and chronic alcohol abuse [87, 88]. When CSDH has a mass effect and produces symptoms, treatment should be provided. A single burr-hole surgery with irrigation and drainage is usually an effective curative treatment for CSDH [89]. However, some patients exhibit the persistent recurrence of CSDH, which has a recurrence rate of up to 20% [90]. In patients with recurrent CSDH, many surgical methods, including the removal of the outer membrane via craniotomy, the implantation of a reservoir or a

subdural-peritoneal shunt, repeated burr-hole trephination and endoscopic surgery have been proposed, but the efficacy of these methods remains widely debated, and there is currently no defined set of treatment algorithms for recurrent CSDH [91].

A CSDH has an outer membrane that develops from the dura mater. In 1997, Tanaka et al. used histological examinations to show that CSDH outer membranes contain three types of vessels, including small veins, arteries and capillaries, and these vessels cross the dura mater to connect to the MMA. Capillary formation in the dura mater then contributes to the formation of hemorrhage in the subdural space, which increases hematomas [92]. At this time, the MMA usually appears as enlarged on MRA [93]. This study was the basis for performing MMA embolization in refractory CSDH patients. MMA embolization might inhibit blood influx through the capillaries into the hematoma cavity to prevent the growth of the hematoma. For instance, in 2002, Takahashi et al. treated 3 cases of refractory CSDH using embolization via the MMA; these patients had previously experienced several unsuccessful drainage procedures [94]. In another instance, in 2015, Tempaku et al. reported five cases of recurrent CSDH that were treated using MMA embolization and found that interventional MMA embolization was a useful procedure [95].

However, while MMA embolization may be effective in some cases of recurrent CSDH, many previous reports about distal MMA embolization for CSDH have suggested that diffuse dilatation of the MMA and the visualization of scattered abnormal vascular networks should be the theoretical basis for performing the procedure. These findings become apparent, especially when super selective angiography of the MMA is performed [94, 96]. Thus, if no abnormal vascular staining lesions are observed in the distal MMA branch, the embolization procedure is not recommended. For instance, in 2004, Hirai et al. reported 2 cases of refractory CSDH that received anticoagulant therapy and underwent attempted MMA embolization. Angiography showed abnormal vascular networks along the MMA, and embolization of the MMA prohibited repeated bleeding from the macrocapillaries on the hematoma capsule and was useful for eliminating the blood supply to this structure [97]. When an MMA embolization is performed, many materials can be used, including polyvinyl alcohol (PVA), N-butyl-2-cyanoacrylate (NBCA), coils and gelatin sponges, all of which are associated with the same therapeutic outcomes [98, 99]. However, when embolizing the peripheral regions of the MMA, it may be better to use 20% NBCA mixed with lipiodol [100].

When embolizing the MMA to treat CSDH, caution should be exercised to prevent aberrant flow into a dangerous anastomosis, which can cause complications. The MMA can potentially communicate with the ophthalmic artery, the internal carotid artery via the inferolateral trunk, or via a feeding vessel to the facial nerve. Thus, an embolus injection should be carefully performed, and performing an embolization of the proximal MMA using coils may be a good choice [95].

7. MMA Contributes to Other Diseases

7.1 MMA Contribution in Migraine

Migraine is a common, disabling, multifactorial, neurovascular headache syndrome [101]. The dura mater is a pain-sensitive structure, and mechanically stimulating the MMA can cause a pounding, migraine-like headache. MMA has been implicated in the pathogenesis of migraine headaches [102]. A previous study showed that migraines can arise when the trigeminovascular system becomes activated, resulting in vasodilatation [103]. Many factors can induce a migraine. These include substance P and neurokinin A, which act by dilating the MMA [104]. Calcitonin gene-related peptide (CGRP) is also an important factor that can induce headaches. For instance, in 2010, Asghar et al. performed a double-blind, randomized, placebo-controlled, crossover study that included 18 healthy volunteers. They found that CGRP caused the MMA to dilate, resulting in headaches. They also found that sumatriptan reversed the dilation of the MMA that was caused by CGRP [105]. Hence, because sumatriptan can cause the MMA to constrict, it is effective for treating acute migraines [106].

However, no evidence of MMA dilation has been observed in morphological examinations performed when a migraine attack occurred. For instance, in 2009, Nagata et al. performed magnetic resonance angiography (MRA) during a spontaneous migraine attack in a 42-year-old woman, and the authors did not observe any dramatic changes in the vasodilation of the MMA during the attack [107]. In 2009, Shevel et al. found that migraine pain was not associated with the dilatation of the dural meningeal arteries [104]. Because the MMA enters the dura and embeds in a groove in the skull, it is difficult to determine whether it is dilated using contemporary imaging techniques. However, the MMA does indeed appear to play an important role in migraines, and migraine treatments that target the MMA are effective. Moreover, sumatriptan and surgical methods can also be attempted. For instance, in 2006, Fan et al. ligated the superficial temporal artery and MMA, resulting in the

severance of the greater superficial petrosal nerve, in 10 patients with cases of severe migraine. At a follow-up performed 2 to 18 years later, no recurrences were observed [108]. These data demonstrate that MMA ligation may be useful for treating intractable migraines.

7.2 Pathway for embolization of meningioma

Meningiomas are extra-axial tumors that are derived from arachnoid cells, which are located along the dural lining of the venous sinuses of the brain and skull base. In cerebral convexity, parasagittal, falcine, sphenoid wing, and tentorium meningiomas, the MMA is often the artery that supplies blood to the tumor [109]. Currently, preoperative embolization is applied to reduce intraoperative blood loss and facilitate the microsurgical removal of meningioma tumors [110]. Because the MMA is the best candidate for embolization in these cases, MMA embolization is widely used as a preoperative treatment for meningioma. For instance, in 2015, Ishihara et al. retrospectively assessed the safety and efficacy of preoperative embolization of the MMA in 56 cases of meningiomas with NBCA of 105 cases in which surgery was performed for a meningioma. The results showed that this approach reduced intraoperative blood loss and surgery times [111].

Although embolization is considered as a safe technique in patients with meningiomas, serious neurological complications can occur. For instance, in 2013, Law-ye et al. performed surgeries for 137 intracranial meningeal tumors, and 2 cases experienced neurological complications that were potentially caused by the opening of dangerous anastomoses or uncontrolled reflux [112]. Communication between the MMA and the ophthalmic artery is commonly achieved via collateral vessels. To avoid complications during embolization of the feeding artery for a skull base meningioma, clinicians must be aware of these collaterals, even if external and internal carotid angiograms did not reveal any anastomosis [110]. In rare cases, an anastomosis may appear during the embolization as a result of hemodynamic changes. For instance, in 2006, Ohata et al. reported a 57-year-old man with a cavernous sinus meningioma. During embolization, a transdural anastomosis from the MMA to the superior cerebellar artery suddenly appeared [113]. Hence, not only overt anastomoses but also covert anastomoses should be monitored.

7.3 Protecting the MMA in Cranial Surgery

The MMA is very important during neurosurgery. The MMA communicates with many other branches of the external or internal carotid

arteries, and it can also sometimes be the sole source for some arteries [114]. For instance, the ophthalmic artery ectopically originates from the MMA at a rate of 3.5% [115]. These ectopic sites of origin from the MMA to the ophthalmic artery are associated with visual complications following surgeries that are directed along the sphenoidal wing or embolizations of the MMA. For instance, in 2007, Hayashi et al. reported three cases of skull base meningioma in which the retinal blood supply originated from the MMA. Taking this into account, an appropriate surgical approach that did not involve the MMA was selected to avoid causing visual complications during the cranial base surgery [6].

In addition to a skull base approach, a frontotemporal craniotomy can also be performed for aneurysms involving the MMA. For instance, in 2013, Maekawa et al. reported a 47-year-old woman with an aneurysm in the right paraclinoid internal carotid artery. Cerebral angiography found that the ophthalmic artery was filled from the anterior branch of the MMA. Because surgical clipping was considered to present a risk of damaging the MMA, which can result in visual disturbances, the aneurysm was treated using coil embolization [116]. Hence, it is important to be aware of this variant of ophthalmic artery anatomy and to carefully check the details shown in images when planning an operation that involves a frontotemporal craniotomy.

8. Summary

The MMA is a clinically important structure when treating neurological diseases using surgery. The MMA is the most commonly involved feeding artery in DAVFs. Moreover, the MMA runs along a straight path and is fixed between the dura. The MMA is therefore commonly used to access DAVFs. The MMA is embedded in a groove of the skull, and trauma or iatrogenic factors can result in pseudoaneurysms or AVFs in the MMA, and when hemodynamic stress increases, a true aneurysm can appear. AVFs, pseudoaneurysms and true aneurysms can be effectively treated via endovascular or surgical removal. In MMD, the MMA plays a very important role in the development and compensation of collateral circulation. Additionally, some indirect revascularization procedures, such as EDAMS, EDAS and burr holes, also depend on the MMA. For recurrent CDSHs, when conventional burr-hole surgery is performed when irrigation and drainage have failed, MMA embolization can be attempted. Moreover, the MMA contributes to migraines, and constricting the dilation of the MMA or ligating the MMA trunk can be used to effectively treat migraines. The MMA also provides an effective pathway for

embolization of meningioma. Finally, because the ophthalmic artery may ectopically originate from the

MMA, when a cranial surgery is performed, caution should be taken to avoid damaging the MMA.

Table 1. Outline and key points of importance regarding the MMA

Outline	Key point	Recommended documents
Pathway for embolization of DAVFs	The MMA is the most commonly involved feeding artery for DAVFs, and the MMA runs along a path that is straight and fixed between the dura. Hence, the MMA is commonly used to access DAVFs. When DAVFs are embolized using a MMA approach, some complications can occur as a result of "dangerous anastomoses" between the MMA and other arteries.	[10, 11, 25]
AVFs of the MMA	A tear in the arterial wall can cause a traumatic AVF in the MMA. AVFs of the MMA that are caused by venous drainage can be divided into six types according to their presentation on angiography. Endovascular embolization and surgical resection are the most effective treatment methods.	[32, 34, 46]
Aneurysms of the MMA	Aneurysms of the MMA can be divided into pseudoaneurysms and true aneurysms. Pseudoaneurysms of the MMA are usually associated with trauma and iatrogenic injury, whereas true aneurysms are usually associated with increased hemodynamic stress or a pathological condition of the MMA. Endovascular embolization and surgical resection are the most effective treatment methods for aneurysms of the MMA.	[47, 50, 60, 70]
The MMA Contributes to MMD	The MMA is a very important component of MMD collateral circulation. The MMA appears to significantly contribute to revascularization on follow-up angiograms, in which it is increased in size and neovascularity compared to the superior temporal artery. Indirect revascularization via encephalo-duro-arterio-synangiosis (EDAS) and burr holes are effective treatments for MMD, and these operations mainly depend on the MMA.	[72, 73, 77, 81, 82]
MMA Embolization in CSDH	The vessels in the outer membrane of the CSDH cross the dura mater to connect to the MMA. This becomes the basis for performing MMA embolization. However, MMA embolization was only effective when diffuse dilatation of the MMA and the abnormal vascular networks could be observed. When embolizing the MMA, caution should be exercised to prevent aberrant flow into the dangerous anastomosis, which can cause complications.	[92, 94-96]
The MMA Contributes to Migraines	Treatments aimed at the MMA are effective in migraine patients. In addition to sumatriptan, MMA ligation may be useful for treating intractable migraines. However, in a morphological examination, when migraine attacks occurred, there was no evidence of MMA dilatation.	[107, 108]
Pathway for Embolization to Treat Meningioma	Preoperative embolization has been used to reduce intraoperative blood loss and facilitate microsurgical removal of meningioma tumors. The MMA is an ideal pathway through which to perform an embolization to treat a meningioma. Although embolization of meningeal tumors is considered a safe technique, serious neurological complications can occur. These include opening a dangerous anastomosis or uncontrolled reflux.	[110-112]
Protecting the MMA during Cranial Surgery	Because the ophthalmic artery can ectopically originate from the MMA, when cranial surgery is performed, caution should be taken to avoid damaging the MMA.	[114, 115]

MMA: middle meningeal artery; DAVF: dural arteriovenous fistula; AVF: arteriovenous fistula; MMD: moyamoya disease; CSDH: Chronic Subdural Hematoma

Competing Interests

The authors have declared that no competing interest exists.

References

- Kornieieva M, Hadidy A, Zhuravlova I. Variability of the Middle Meningeal Artery Subject to the Shape of Skull. *J Neurol Surg B Skull Base.* 2015; 76: 451-8.
- Mewada T, Ohshima T, Yamamoto T, Goto S, Kato Y. Usefulness of embolization for iatrogenic dural arteriovenous fistula associated with recurrent chronic subdural hematoma: A case report and literature review. *World Neurosurg.* 2016.
- Grandhi R, Zwagerman NT, Lee P, Jovin T, Okonkwo DO. Iatrogenic pseudoaneurysm of the middle meningeal artery after external ventricular drain placement. *J Neuroimaging.* 2015; 25: 140-1.
- Ko JH, Kim YJ. Traumatic pseudoaneurysm of the middle meningeal artery with an arteriovenous fistula on a non-fractured site. *Interv Neuroradiol.* 2014; 20: 352-6.
- Hori S, Kashiwazaki D, Akioka N, Hayashi T, Hori E, Umemura K, et al. Surgical anatomy and preservation of the middle meningeal artery during bypass surgery for moyamoya disease. *Acta Neurochir (Wien).* 2015; 157: 29-36.
- Hayashi N, Kubo M, Tsuboi Y, Nishimura S, Nishijima M, Ahmed Abdel-Aal M, et al. Impact of anomalous origin of the ophthalmic artery from the middle meningeal artery on selection of surgical approach to skull base meningioma. *Surg Neurol.* 2007; 68: 568-71; discussion 71-2.
- Serulle Y, Miller TR, Gandhi D. Dural Arteriovenous Fistulae: Imaging and Management. *Neuroimaging Clin N Am.* 2016; 26: 247-58.
- Gabrieli J, Clarencon F, Di Maria F, Chiras J, Sourour N. Occipital artery: a not so poor artery for the embolization of lateral sinus dural arteriovenous fistulas with Onyx. *J Neurointerv Surg.* 2015.
- Umeoka K, Takusakawa Y, Kominami S, Kobayashi S, Morita A. The meningeal branches of the superior cerebellar artery: a surgical observation study. *J Neurosurg.* 2016; 124: 244-7.
- Griessenauer CJ, He L, Salem M, Chua M, Ogilvy CS, Thomas AJ. Middle Meningeal Artery: Gateway for effective transarterial Onyx embolization of dural arteriovenous fistulas. *Clin Anat.* 2016.
- Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A, Jr. Microsurgical anatomy of the dural arteries. *Neurosurgery.* 2005; 56: 211-51; discussion 51.
- Baik SK, Kim YW, Lee SW, Choi CH, Park J. A treatment option for nontraumatic adult-type dural arteriovenous fistulas: transarterial venous coil embolization. *World Neurosurg.* 2014; 82: 417-22.
- Kresimir Lukic I, Gluncic V, Marusic A. Extracranial branches of the middle meningeal artery. *Clin Anat.* 2001; 14: 292-4.
- Chmielewski P, Skrzat J, Walocha J. Clinical importance of the middle meningeal artery. *Folia Med Cracov.* 2013; 53: 41-6.
- Deng JP, Li J, Zhang T, Yu J, Zhao ZW, Gao GD. Embolization of dural arteriovenous fistula of the anterior cranial fossa through the middle meningeal artery with Onyx. *Clin Neurol Neurosurg.* 2014; 117: 1-5.
- Osburn JW, Kim LJ, Spetzler RF, McDougall CG. Aberrant venous drainage pattern in a medial sphenoid wing dural arteriovenous fistula: a case report and review of the literature. *World Neurosurg.* 2013; 80: e381-6.
- Elhammady MS, Wolfe SQ, Farhat H, Moftakhar R, Aziz-Sultan MA. Onyx embolization of carotid-cavernous fistulas. *J Neurosurg.* 2010; 112: 589-94.
- Arat A, Inci S. Treatment of a superior sagittal sinus dural arteriovenous fistula with Onyx: technical case report. *Neurosurgery.* 2006; 59: ONSE169-70; discussion ONSE-70.
- Ponomarjova S, Iosif C, Mendes GA, Mounayer C. Endovascular Treatment of Transverse-Sigmoid Sinus Type I Dural Arteriovenous Shunts with Sinus Preservation for Patients with Intolerable Symptoms: Four Case Reports. *Clin Neuroradiol.* 2015; 25: 313-6.
- Wajnberg E, Spilberg G, Rezende MT, Abud DG, Kessler I, Mounayer C, et al. Endovascular treatment of tentorial dural arteriovenous fistulae. *Interv Neuroradiol.* 2012; 18: 60-8.

21. Oh JS, Yoon SM, Oh HJ, Shim JJ, Bae HG, Lee KS. Endovascular Treatment of Dural Arteriovenous Fistulas: Single Center Experience. *J Korean Neurosurg Soc.* 2016; 59: 17-25.
22. Kim B, Jeon P, Kim K, Kim S, Kim H, Byun HS, et al. Predictive Factors for Response of Intracranial Dural Arteriovenous Fistulas to Transarterial Onyx Embolization: Angiographic Subgroup Analysis of Treatment Outcomes. *World Neurosurg.* 2016; 88: 609-18.
23. Lin N, Brouillard AM, Mokin M, Natarajan SK, Snyder KV, Levy EI, et al. Direct access to the middle meningeal artery for embolization of complex dural arteriovenous fistula: a hybrid treatment approach. *J Neurointerv Surg.* 2015; 7: e24.
24. Oh JS, Yoon SM, Shim JJ, Bae HG. Transcranial direct middle meningeal artery puncture for the onyx embolization of dural arteriovenous fistula involving the superior sagittal sinus. *J Korean Neurosurg Soc.* 2015; 57: 54-7.
25. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: vessels the neurointerventionalist needs to know. *AJNR Am J Neuroradiol.* 2009; 30: 1459-68.
26. Tubbs RS, Walker AM, Demerdash A, Matusz P, Loukas M, Cohen-Gadol AA. Skull base connections between the middle meningeal and internal carotid arteries. *Childs Nerv Syst.* 2015; 31: 1515-20.
27. Perrini P, Cardia A, Fraser K, Lanzino G. A microsurgical study of the anatomy and course of the ophthalmic artery and its possibly dangerous anastomoses. *J Neurosurg.* 2007; 106: 142-50.
28. Nyberg EM, Chaudry MI, Turk AS, Turner RD. Transient cranial neuropathies as sequelae of Onyx embolization of arteriovenous shunt lesions near the skull base: possible axonometric traction injuries. *J Neurointerv Surg.* 2013; 5: e21.
29. Wang AG, Liu JH, Hsu WM, Luo CB, Yen MY. Choroidal infarction after embolization of arteriovenous fistula of middle meningeal artery. *Retina.* 2000; 20: 573-5.
30. Unterhofer C, Chemelli A, Waldenberger P, Bauer R, Ortler M. Traumatic fistula between the middle meningeal artery and the sphenoparietal sinus. *Acta Neurochir (Wien).* 2009; 151: 1301-4.
31. Iakovlev SB. [An arteriosinusual anastomosis between the middle meningeal artery and the cavernous sinus. Its surgical treatment using the balloon catheter]. *Zh Vopr Neurokhir Im N N Burdenko.* 1997: 31-3.
32. Freckmann N, Sartor K, Herrmann HD. Traumatic arteriovenous fistulae of the middle meningeal artery and neighbouring veins or dural sinuses. *Acta Neurochir (Wien).* 1981; 55: 273-81.
33. Bitoh S, Hasegawa H, Fujiwara M, Nakata M. Traumatic arteriovenous fistula between the middle meningeal artery and cortical vein. *Surg Neurol.* 1980; 14: 355-8.
34. Vassilyadi M, Mehrotra N, Shamji MF, Michaud J. Pediatric traumatic dural arteriovenous fistula. *Can J Neuro Sci.* 2009; 36: 751-6.
35. Tsutsumi K, Shiokawa Y, Kubota M, Aoki N, Mizutani H. Postoperative arteriovenous fistula between the middle meningeal artery and the sphenoparietal sinus. *Neurosurgery.* 1990; 26: 869-71.
36. Inagawa T, Takeda T, Taguchi H, Kamiya K, Yamada T. Traumatic middle meningeal arteriovenous fistula caused by three-point skull fixation. Case report. *J Neurosurg.* 1984; 60: 853-5.
37. Terada T, Nakai E, Tsumoto T, Itakura T. Iatrogenic arteriovenous fistula of the middle meningeal artery caused during embolization for meningioma--case report. *Neurol Med Chir (Tokyo).* 1997; 37: 677-80.
38. Hassler O. Medial defects in the meningeal arteries. *J Neurosurg.* 1962; 19: 337-40.
39. Billewicz O, Kamraj-Mazurkiewicz K, Pryczkowski J. Case of congenital arteriovenous fistula fed by the middle meningeal artery. *Neuroradiology.* 1971; 2: 234-6.
40. Takeuchi S, Takasato Y, Masaoka H, Hayakawa T, Otani N, Yoshino Y, et al. [A case of traumatic middle meningeal arteriovenous fistula on the side of the head opposite to the injured side]. *No Shinkei Geka.* 2009; 37: 983-6.
41. Pritz MB, Pribram HF. Intracerebral hemorrhage from a middle meningeal arteriovenous fistula with a giant venous varix. *Surg Neurol.* 1992; 37: 460-3.
42. Luciani A, Houdart E, Mounayer C, Saint Maurice JP, Merland JJ. Spontaneous closure of dural arteriovenous fistulas: report of three cases and review of the literature. *AJNR Am J Neuroradiol.* 2001; 22: 992-6.
43. Otake G. [Spontaneous closure of a traumatic middle meningeal arteriovenous fistula accompanied by a sagittal epidural hematoma (author's transl)]. *Neurol Med Chir (Tokyo).* 1981; 21: 1267-73.
44. Chandrashekar HS, Nagarajan K, Srikanth SG, Jayakumar PN, Vasudev MK, Pandey P. Middle meningeal arteriovenous fistula and its spontaneous closure. A case report and review of the literature. *Interv Neuroradiol.* 2007; 13: 173-8.
45. Rennert J, Seiz M, Nimsky C, Doerfler A. Endovascular treatment of traumatic high flow dural arterio-venous fistula involving the middle meningeal artery and facial veins. *Rontgenpraxis.* 2008; 56: 164-8.
46. Sakata H, Nishimura S, Mino M, Hori E, Fujita T, Midorikawa H, et al. Serial angiography of dynamic changes of traumatic middle meningeal arteriovenous fistula: case report. *Neurol Med Chir (Tokyo).* 2009; 49: 462-4.
47. Srinivasan A, Lesiuk H, Goyal M. Spontaneous resolution of posttraumatic middle meningeal artery pseudoaneurysm. *AJNR Am J Neuroradiol.* 2006; 27: 882-3.
48. Salazar Flores J, Vaquero J, Garcia Sola R, Rossi E, Martinez R, Martinez P, et al. Traumatic false aneurysms of the middle meningeal artery. *Neurosurgery.* 1986; 18: 200-3.
49. Kawaguchi T, Kawano T, Kaneko Y, Ooasa T, Ooigawa H, Ogasawara S. Traumatic lesions of the bilateral middle meningeal arteries--case report. *Neurol Med Chir (Tokyo).* 2002; 42: 221-3.
50. Zubkov YN, Matsko DE, Pak VA. Saccular aneurysms of meningeal artery: case report. *Neurosurgery.* 1998; 42: 664-6.
51. Akyuz M, Tuncer R. Multiple middle meningeal artery aneurysms associated with fistulous galenic arteriovenous malformation: a case report. *Turk Neurosurg.* 2010; 20: 544-6.
52. Koebbe CJ, Horowitz MB. A rare case of a ruptured middle meningeal aneurysm causing intracerebral hematoma in a patient with moyamoya disease. *AJNR Am J Neuroradiol.* 2004; 25: 574-6.
53. Li B, Wang CC, Zhao ZZ, Hu Y, Aihara K, Ghazizadeh M, et al. A histological, ultrastructural and immunohistochemical study of superficial temporal arteries and middle meningeal arteries in moyamoya disease. *Acta Pathol Jpn.* 1991; 41: 521-30.
54. Yang SH, Li B, Wang CC, Zhao JZ. Angiographic study of moyamoya disease and histological study in the external carotid artery system. *Clin Neurol Neurosurg.* 1997; 99 Suppl 2: S61-3.
55. O'Neill OR, Barnwell SL, Silver DJ. Middle meningeal artery aneurysm associated with meningioma: case report. *Neurosurgery.* 1995; 36: 396-8.
56. Maekawa H, Tanaka M, Hadeishi H. Middle meningeal artery aneurysm associated with meningioma. *Acta Neurochir (Wien).* 2009; 151: 1167-8.
57. New PF. True aneurysm of the middle meningeal artery, cranial Page's disease and hypertension: a triad. *Clin Radiol.* 1967; 18: 154-7.
58. Lesley WS, Thomas MR, Abdulrauf SI. N-butylcyanoacrylate embolization of a middle meningeal artery aneurysm in a patient with neurofibromatosis type 2. *AJNR Am J Neuroradiol.* 2004; 25: 1414-6.
59. Bruneau M, Gustin T, Zekhnini K, Gilliard C. Traumatic false aneurysm of the middle meningeal artery causing an intracerebral hemorrhage: case report and literature review. *Surg Neurol.* 2002; 57: 174-8; discussion 8.
60. Aoki N, Sakai T, Kaneko M. Traumatic aneurysm of the middle meningeal artery presenting as delayed onset of acute subdural hematoma. *Surg Neurol.* 1992; 37: 59-62.
61. Kumar RJ, Sundaram PK, Gunjkar JD. Traumatic giant pseudoaneurysm of the middle meningeal artery causing intracerebral hematoma. *Neurol India.* 2011; 59: 921-2.
62. Jussen D, Wiener E, Vajkoczy P, Horn P. Traumatic middle meningeal artery pseudoaneurysms: diagnosis and endovascular treatment of two cases and review of the literature. *Neuroradiology.* 2012; 54: 1133-6.
63. Paiva WS, de Andrade AF, Amorim RL, Figueiredo EG, Teixeira MJ. Traumatic pseudoaneurysm of the middle meningeal artery causing an intracerebral hemorrhage. *Case Rep Med.* 2010; 2010: 219572.
64. Singh M, Ahmad FU, Mahapatra AK. Traumatic middle meningeal artery aneurysm causing intracerebral hematoma: a case report and review of literature. *Surg Neurol.* 2006; 66: 321-3; discussion 3.
65. Kobata H, Tanaka H, Tada Y, Nishihara K, Fujiwara A, Kuroiwa T. Intracerebral hematoma due to ruptured nontraumatic middle meningeal artery aneurysm--case report. *Neurol Med Chir (Tokyo).* 2001; 41: 611-4.
66. Namba K, Ban S, Oda Y, Tazumi M, Ogata M. [Case of traumatic false aneurysm of the middle meningeal artery with epidural hematoma--comparison with a case resulting in spontaneous thrombosis]. *Rinsho Shinkeigaku.* 1972; 12: 165-70.
67. Paiva WS, Andrade AF, Amorim RL, Bor-Seng-Shu E, Gattas G, Neville IS, et al. Computed tomography angiography for detection of middle meningeal artery lesions associated with acute epidural hematomas. *Biomed Res Int.* 2014; 2014: 413916.
68. Sandin JA, 3rd, Salamat MS, Baskaya M, Dempsey RJ. Intracerebral hemorrhage caused by the rupture of a nontraumatic middle meningeal artery aneurysm. Case report and review of the literature. *J Neurosurg.* 1999; 90: 951-4.
69. Wang CH, Lee HC, Cho DY. Traumatic pseudoaneurysm of the middle meningeal artery: possible indicators for early diagnosis in the computed tomography era. *Surg Neurol.* 2007; 68: 676-81; discussion 81-2.
70. Park YS, Suk JS, Kwon JT. Repeated rupture of a middle meningeal artery aneurysm in moyamoya disease. Case report. *J Neurosurg.* 2010; 113: 749-52.
71. Zhang L, Xu K, Zhang Y, Wang X, Yu J. Treatment strategies for aneurysms associated with moyamoya disease. *Int J Med Sci.* 2015; 12: 234-42.
72. Suzuki J, Kodama N. Cerebrovascular "Moyamoya" disease. 2. Collateral routes to forebrain via ethmoid sinus and superior nasal meatus. *Angiology.* 1971; 22: 223-36.
73. Kuroda S, Houkin K. Bypass surgery for moyamoya disease: concept and essence of surgical techniques. *Neurol Med Chir (Tokyo).* 2012; 52: 287-94.
74. Matsukawa H, Fujii M, Murakata A, Shinoda M, Takahashi O. Foramen spinosum and middle meningeal artery in moyamoya disease: Preliminary results of a pilot study. *Brain Inj.* 2015: 1-6.
75. Honda M, Kitagawa N, Tsutsumi K, Morikawa M, Nagata I, Kaminogo M. Magnetic resonance angiography evaluation of external carotid artery tributaries in moyamoya disease. *Surg Neurol.* 2005; 64: 325-30.
76. Yamada I, Matsushima Y, Suzuki S. Childhood moyamoya disease before and after encephalo-duro-arterio-synangiosis: an angiographic study. *Neuroradiology.* 1992; 34: 318-22.
77. Dusick JR, Gonzalez NR, Martin NA. Clinical and angiographic outcomes from indirect revascularization surgery for Moyamoya disease in adults and children: a review of 63 procedures. *Neurosurgery.* 2011; 68: 34-43; discussion

78. Kim T, Oh CW, Bang JS, Kim JE, Cho WS. Moyamoya Disease: Treatment and Outcomes. *J Stroke*. 2016; 18: 21-30.
79. Gonzalez NR, Dusick JR, Connolly M, Bounni F, Martin NA, Van de Wiele B, et al. Encephaloduroarteriosynangiosis for adult intracranial arterial steno-occlusive disease: long-term single-center experience with 107 operations. *J Neurosurg*. 2015; 123: 654-61.
80. Zhao X, Wang C, Ji Y, Han C, Wang M. Therapeutic effect of multiple burr hole operation combined with dural inversion and periosteal synangiosis for moyamoya disease. *Br J Neurosurg*. 2015; 29: 811-7.
81. Dauser RC, Tuite GF, McCluggage CW. Dural inversion procedure for moyamoya disease. Technical note. *J Neurosurg*. 1997; 86: 719-23.
82. Kashiwagi S, Kato S, Yamashita K, Takasago T, Akimura T, Okamura S, et al. Revascularization with split duro-encephalo-synangiosis in the pediatric moyamoya disease—surgical result and clinical outcome. *Clin Neurol Neurosurg*. 1997; 99 Suppl 2: S115-7.
83. Houkin K, Kuroda S, Ishikawa T, Abe H. Neovascularization (angiogenesis) after revascularization in moyamoya disease. Which technique is most useful for moyamoya disease? *Acta Neurochir (Wien)*. 2000; 142: 269-76.
84. McLaughlin N, Martin NA. Effectiveness of burr holes for indirect revascularization in patients with moyamoya disease—a review of the literature. *World Neurosurg*. 2014; 81: 91-8.
85. King JA, Armstrong D, Vachrajani S, Dirks PB. Relative contributions of the middle meningeal artery and superficial temporal artery in revascularization surgery for moyamoya syndrome in children: the results of superselective angiography. *J Neurosurg Pediatr*. 2010; 5: 184-9.
86. McLaughlin N, Martin NA. Meningeal management for optimal revascularization from middle meningeal artery. *J Neurosurg*. 2013; 118: 104-8.
87. Abe Y, Maruyama K, Yokoya S, Noguchi A, Sato E, Nagane M, et al. Outcomes of chronic subdural hematoma with preexisting comorbidities causing disturbed consciousness. *J Neurosurg*. 2016; 1-5.
88. Min X, Pin C, Xun Z, Cun-Zu W, Xue-Qiang S, Bo Y. Effects of atorvastatin on conservative and surgical treatments of chronic subdural hematoma in patients. *World Neurosurg*. 2016.
89. Shapey J, Glancz LJ, Brennan PM. Chronic Subdural Haematoma in the Elderly: Is It Time for a New Paradigm in Management? *Curr Geriatr Rep*. 2016; 5: 71-7.
90. Jang KM, Kwon JT, Hwang SN, Park YS, Nam TK. Comparison of the Outcomes and Recurrence with Three Surgical Techniques for Chronic Subdural Hematoma: Single, Double Burr Hole, and Double Burr Hole Drainage with Irrigation. *Korean J Neurotrauma*. 2015; 11: 75-80.
91. Kang J, Whang K, Hong SK, Pyen JS, Cho SM, Kim JY, et al. Middle Meningeal Artery Embolization in Recurrent Chronic Subdural Hematoma Combined with Arachnoid Cyst. *Korean J Neurotrauma*. 2015; 11: 187-90.
92. Tanaka T, Fujimoto S, Saito K, Kaimori M. [Histological study of operated cases of chronic subdural hematoma in adults: relationship between dura mater and outer membrane]. *No Shinkei Geka*. 1997; 25: 701-5.
93. Takizawa K, Sorimachi T, Ishizaka H, Osada T, Srivatanakul K, Momose H, et al. Enlargement of the middle meningeal artery on MR angiography in chronic subdural hematoma. *J Neurosurg*. 2015; 1-5.
94. Takahashi K, Muraoka K, Sugiura T, Maeda Y, Mandai S, Gohda Y, et al. [Middle meningeal artery embolization for refractory chronic subdural hematoma: 3 case reports]. *No Shinkei Geka*. 2002; 30: 535-9.
95. Tempaku A, Yamauchi S, Ikeda H, Tsubota N, Furukawa H, Maeda D, et al. Usefulness of interventional embolization of the middle meningeal artery for recurrent chronic subdural hematoma: Five cases and a review of the literature. *Interv Neuroradiol*. 2015; 21: 366-71.
96. Mandai S, Sakurai M, Matsumoto Y. Middle meningeal artery embolization for refractory chronic subdural hematoma. Case report. *J Neurosurg*. 2000; 93: 686-8.
97. Hirai S, Ono J, Odaki M, Serizawa T, Nagano O. Embolization of the Middle Meningeal Artery for Refractory Chronic Subdural Haematoma. Usefulness for Patients under Anticoagulant Therapy. *Interv Neuroradiol*. 2004; 10 Suppl 2: 101-4.
98. Hashimoto T, Ohashi T, Watanabe D, Koyama S, Namatame H, Izawa H, et al. Usefulness of embolization of the middle meningeal artery for refractory chronic subdural hematomas. *Surg Neurol Int*. 2013; 4: 104.
99. Mino M, Nishimura S, Hori E, Kohama M, Yonezawa S, Midorikawa H, et al. Efficacy of middle meningeal artery embolization in the treatment of refractory chronic subdural hematoma. *Surg Neurol Int*. 2010; 1: 78.
100. Ishihara H, Ishihara S, Kohyama S, Yamane F, Ogawa M, Sato A, et al. Experience in endovascular treatment of recurrent chronic subdural hematoma. *Interv Neuroradiol*. 2007; 13 Suppl 1: 141-4.
101. Ferrari MD. Migraine. *Lancet*. 1998; 351: 1043-51.
102. Sanchez-del-Rio M, Reuter U. Migraine aura: new information on underlying mechanisms. *Curr Opin Neurol*. 2004; 17: 289-93.
103. Hargreaves R. New migraine and pain research. *Headache*. 2007; 47 Suppl 1: S26-43.
104. Shevel E. Middle meningeal artery dilatation in migraine. *Headache*. 2009; 49: 1541-3.
105. Asghar MS, Hansen AE, Kapijimpanga T, van der Geest RJ, van der Koning P, Larsson HB, et al. Dilatation by CGRP of middle meningeal artery and reversal by sumatriptan in normal volunteers. *Neurology*. 2010; 75: 1520-6.
106. Law S, Derry S, Moore RA. Sumatriptan plus naproxen for the treatment of acute migraine attacks in adults. *Cochrane Database Syst Rev*. 2016; 4: CD008541.
107. Nagata E, Moriguchi H, Takizawa S, Horie T, Yanagimachi N, Takagi S. The middle meningeal artery during a migraine attack: 3T magnetic resonance angiography study. *Intern Med*. 2009; 48: 2133-5.
108. Fan Z, Fan Z, Wang H. New surgical approach for migraine. *Otol Neurotol*. 2006; 27: 713-5.
109. Shah A, Choudhri O, Jung H, Li G. Preoperative endovascular embolization of meningiomas: update on therapeutic options. *Neurosurg Focus*. 2015; 38: E7.
110. Meguro T, Tomita Y, Tanabe T, Muraoka K, Terada K, Hirotsune N, et al. [Embolization of the feeding artery of a meningioma with dangerous vascular anastomosis between the middle meningeal artery and the ophthalmic artery]. *No Shinkei Geka*. 2013; 41: 995-9.
111. Ishihara H, Ishihara S, Niimi J, Neki H, Kakehi Y, Uemiya N, et al. The safety and efficacy of preoperative embolization of meningioma with N-butyl cyanoacrylate. *Interv Neuroradiol*. 2015; 21: 624-30.
112. Law-ye B, Clarencon F, Sourour NA, Di Maria F, Jean B, Bonneville F, et al. Risks of presurgical embolization of feeding arteries in 137 intracranial meningeal tumors. *Acta Neurochir (Wien)*. 2013; 155: 707-14.
113. Ohata K, Nishio A, Takami T, Goto T. Sudden appearance of transdural anastomosis from middle meningeal artery to superior cerebellar artery during preoperative embolization of meningioma. *Neurol India*. 2006; 54: 328.
114. Kadooka K, Tanaka M. Ophthalmic systems completely supplied from dural arteries indicate the utility of endovascular treatment of cerebral aneurysms. *Interv Neuroradiol*. 2015; 21: 765-8.
115. Lefkowitz M, Giannotta SL, Hieshima G, Higashida R, Halbach V, Dowd C, et al. Embolization of neurosurgical lesions involving the ophthalmic artery. *Neurosurgery*. 1998; 43: 1298-303.
116. Maekawa H. Anomalous ophthalmic artery arising from middle meningeal artery: potential risk of visual complication in frontotemporal craniotomy. *Clin Neurol Neurosurg*. 2013; 115: 2547-8.