De Novo Formation of Pial Arteriovenous Fistulas: Systematic Review of Acquired Lesions and Their Clinical Differences Compared with Primary Lesions

Tzu-Chin Lin[1], Ting-Wei Chang[1], Yi-Ming Wu[2], Ching-Chang Chen[1], Chun-Ting Chen[1], Tai-Wei Erich Wu[1], Chieh-Tsai Wu[1], Po-Chuan Hsieh[1]

■ OBJECTIVE: **Acquired pial arteriovenous fistula (pAVF) is an extremely rare intracranial vascular malformation, with few case reports in the English literature. This study presents a thorough review and analysis of all acquired pAVF cases from the literature in addition to an illustrated case.**

■ METHODS: **We report a case with de novo development of intracranial pAVF after craniotomy. A medical literature database search between 1975 and 2018, including the Medline, Ovid, and PubMed databases, was performed to identify all reports with possible acquired lesions. Differences between these acquired lesions and previously reported primary lesions were evaluated.**

■ RESULTS: **A total of 8 patients with de novo formation of acquired pAVF were included in this series. Most of these pAVFs were fed and drained via a similar arteriovenous pattern, from distal/cortical branches of the middle cerebral artery (6/8, 75%) to the superficial middle cerebral vein (6/8, 75%). Compared with a previously reported primary pAVF series, acquired pAVF tended to be asymptomatic ($P < 0.0001$) and found essentially in adults ($P = 0.0061$). Fewer venous varices ($P = 0.0049$) and associated intracranial mass effect ($P = 0.0189$) were found in the cases of acquired pAVF. All 4 reported acquired pAVFs that were treated microsurgically resulted in complete angiographic obliteration (4/4, 100%). The overall outcome was good or stable even with observation only (7/8, 87.5%).**

■ CONCLUSIONS: **Acquired pAVF is highly correlated with sentinel neurosurgical procedures or venous occlusion events. These lesions should be regarded as a different disease entity from primary pAVF because of the relatively low-flow shunting and benign clinical course.**

INTRODUCTION

Pial arteriovenous fistula (pAVF) is a rare intracranial vascular malformation with fewer than 120 reported cases in the English literature according to previous reviews.[1] These lesions were once believed to be a subgroup of arteriovenous malformation (AVM) but were later reclassified and viewed as a distinct disease entity.[2] However, more than half of the cases in the literature were found to have significant dilatation of the venous drainage system, which indicated the possibility of a primary or chronic origin of the possible pathomechanism.[3] Acquired pAVF may have developed de novo and might be considered a rarer and more distinct disease entity that has yet to be well studied, based on the lack of published reports.

We report a de novo acquired pAVF formation with pseudoaneurysm occurring after a craniotomy was performed to resect a ruptured deep-seated cerebral AVM. We also review the literature for similarly acquired lesions and discuss the causes, lesion locations, pathophysiology, clinical course, and possible strategies for management. The differences between the acquired and primary lesions were compared and evaluated. This is the first study to analyze all the acquired pAVF from the literature and present a thorough comparison with primary pAVFs.

Key words
- Acquired pial arteriovenous fistula
- Cortical venous injury
- Craniotomy
- Primary pial arteriovenous fistula
- Recanalization
- Venous varix

Abbreviations and Acronyms
AVM: Arteriovenous malformation
CSF: Cerebrospinal fluid
DSA: Digital subtraction angiography
MCA: Middle cerebral artery
pAVF: Pial arteriovenous fistula

From the Departments of [1]Neurosurgery and [2]Medical Imaging and Intervention, Chang Gung Memorial Hospital, Chang Gung University and Medical College, Linkou, Taiwan

To whom correspondence should be addressed: Po-Chuan Hsieh, M.D. [E-mail: pochuan.hsieh@gmail.com]

Tzu-Chin Lin and Ting-Wei Chang contributed equally to this article as co-first authors.

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CASE ILLUSTRATION

A 42-year-old male patient presented to our service emergently because of the sudden onset of an explosive headache associated with drowsiness. A neurologic examination showed left homonymous hemianopia and progressively depressed consciousness. Computed tomography angiography performed in the emergency room found a posterior temporal intracerebral hemorrhage with a susceptible vascular lesion within the hematoma. Digital subtraction angiography (DSA) later confirmed the diagnosis of a ruptured AVM with arterial supply from the posterior cerebral artery and 2 drainage veins into the straight and transverse sinus (Figure 1A and B). After informed consent was acquired, a craniotomy was performed to relieve the mass effect caused by the hematoma, and AVM resection was implemented for bleeder control.

The craniotomy AVM resection was uneventful until the completion of the AVM resection before dura closure. Minor bleeding was found from the subdural space, which was anticipated to have occurred from the possible draining vein over the posterior temporal region. The bleeding event was stopped spontaneously within 5 minutes after normal saline irrigation of the resection cavity and re-establishment of the cerebrospinal fluid (CSF) filling space intracranially. The recovery from surgery was uneventful, except for a minor headache reported by the patient. However, the symptoms subjectively worsened 3–4 days later after transfer from the intensive care unit.

The postoperative follow-up DSA was performed 5 days after the primary procedure. Complete resection and no contrast staining of the nidus were seen on the vertebral artery angiogram (Figure 1C). However, arterial flush and an approximate 8-mm aneurysm sac connected from the distal middle cerebral artery (MCA) branches to the cortical vein, which drained into the transverse sinus, was found during ipsilateral internal carotid injection. De novo pAVF with pseudoaneurysm was diagnosed based on these angiographic

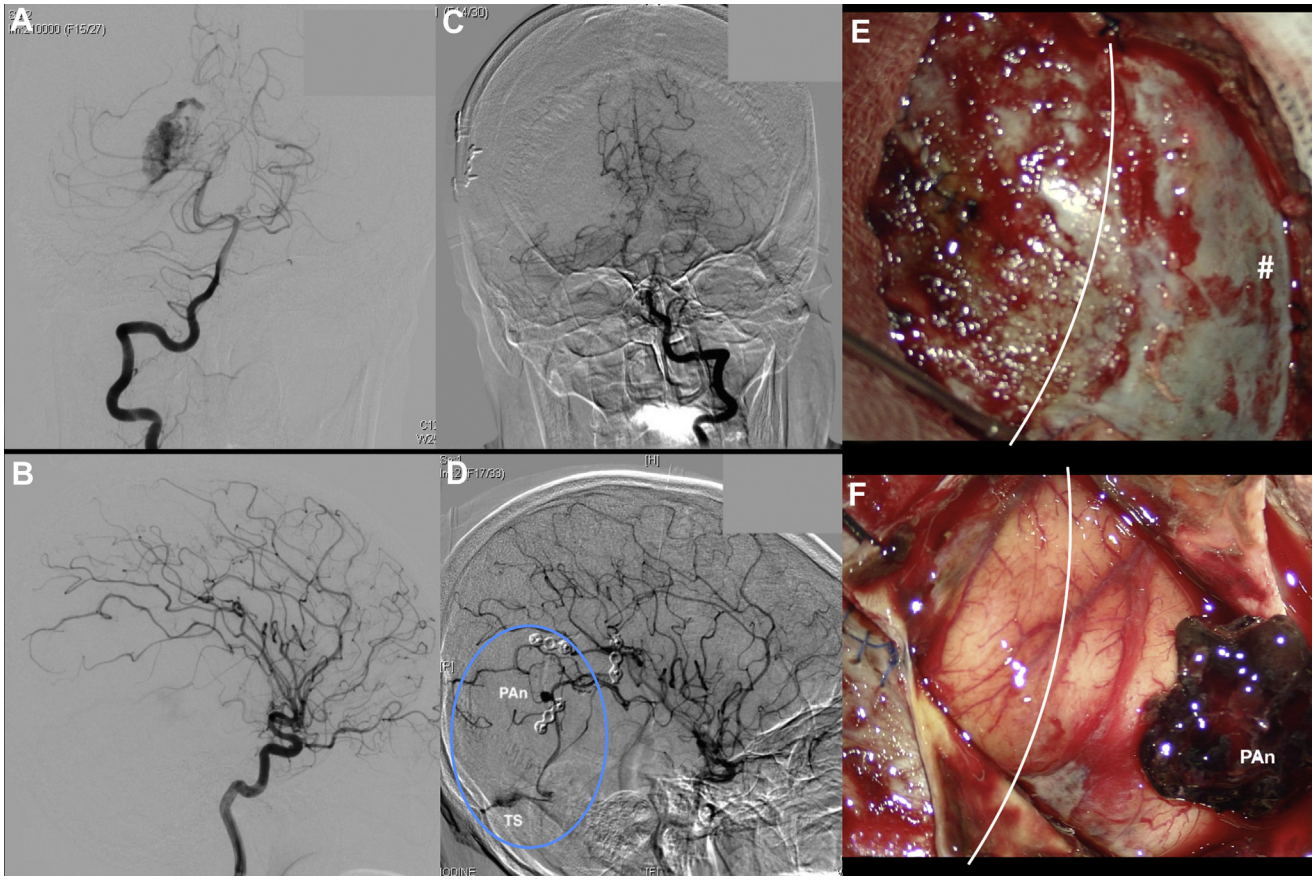

Figure 1. The neuroimaging sets of the ruptured temporal arteriovenous malformation. (**A**) Preoperative vertebral artery angiography showing the arteriovenous malformation nidus perfused by the posterior cerebral artery. (**B**) Preoperative internal carotid artery angiography showing a relative hypovascular mass effect over the posterior temporal region, without obvious vascular abnormality. (**C**) Postoperative 1 week vertebral angiography showing complete resection of the arteriovenous malformation nidus. (**D**) Postoperative 1 week internal carotid angiography showing a pial arteriovenous fistula with pseudoaneurysm formation closely related to the arteriovenous junction (*blue circle*). (**E**) Extended craniotomy for pial arteriovenous fistula. (*White line*, previous craniotomy edge; *#*, estimated pseudoaneurysm location.) (**F**) Intraoperative findings after dura open and subdural removal. PAn, pseudoaneurysm.

Table 1. Summarized Literature Reviews and Clinical Characteristics of Acquired Pial Arteriovenous Fistula

Author	Age (years)/ Sex	Feeding Vessel	Arteriovenous Junction	Final Venous Drainage	Presentation	Bleeding	Venous Varix	Aneurysm/ Pseudoaneurysm	Hematologic Abnormality	Primary Treatment	Diagnosis Time Interval	Treatment	Angiographic Obliteration	Outcome	Possible Cause
Current study, 2018	36/M	MCA	SMCV	IAV	Headache	No	No	Yes	No	Arteriovenous malformation resection	1 week	Microsurgery	Yes	Good	Venous recanalization
Lyons et al., 2017[4]	70/M	MCA	Superior cerebral veins	Superior sagittal sinus	Seizure, status epilepticus	Subarachnoid hemorrhage	No	No	DVT, pulmonary embolism	Spinal surgery (no cerebrospinal fluid leakage)	<1 day	Observation (status epilepticus)	yes	Good (3 months)	Acute loss of cerebrospinal fluid, decreased intracranial pressure
Nomura et al., 2015[5]	61/M	MCA	SMCV	IAV	Seizure, brain edema	No	No	No	No	NA	1 week	Microsurgery	Yes	Good	Trauma
Feroze et al., 2015[6]	51/F	STA	SMCV	SAV	None (FU angiography)	No	No	No	No	Extracranial-intracranial bypass for moyamoya	6 months	Observation	No	Stable	Venous recanalization
Nishiyama et al., 2014[7]	63/F	MCA	SMCV	SAV	None (FU angiography)	No	No	No	No	Aneurysm clipping	2 weeks	Microsurgery	Yes	Good	Direct injury by suture
Schuette et al., 2012[8]	47/F	Anterior cerebral artery	Superior cerebral veins	Superior sagittal sinus	None (FU angiography)	No	No	No	No	External ventricular drain, aneurysm coiling	18 months	Microsurgery	Yes	Good	Direct injury during external ventricular drainage puncture
Kubo et al., 2010[9]	67/F	MCA	SMCV	Sigmoid sinus	None (FU angiography)	No	Yes	No	No	Aneurysm clipping	21 months	Observation	No	Stable(46 months)	Surgicel? venous recanalization?
Phatouros et al., 1999[10]	51/M	MCA	SMCV	IAV	None (FU angiography)	No	No	No	No	Aneurysm coiling	18 months	Observation	No	NA	Venous thrombosis

M, male; MCA, middle cerebral artery; SMCV, superficial middle cerebral veins; IAV, inferior anastomotic vein (vein of Labbé); DVT, deep vein thrombosis; NA, not available; F, female; STA, superficial temporal artery; ISAV, superior anastomotic vein (vein of Trolard); FU, follow-up.

findings (Figure 1D). Craniotomy for pAVF disconnection and resection of the aneurysm/pseudoaneurysm was performed.

We extended the previous craniotomy toward the posterior temporal region based on neuronavigation (previous craniotomy margin: *white curved line* in Figure 1E and F). There was marked subdural hematoma surrounding the susceptible lesion, which was located 1–2 cm posteriorly from the previous craniotomy edge (Figure 1F). Active bleeding was observed from a small torn cortical artery and easily controlled via electrocauterization after removal of the hematoma and pseudoaneurysm sac (Figure 1F). The patient was discharged without new neurologic sequelae after the second craniotomy. Postoperative magnetic resonance angiography showed complete resection of the lesion without any suspicious arterial phase lesions.

METHODS

We searched the Medline, Ovid, and PubMed databases between 1975 and 2018 for possible correlated studies. Keywords included: "pial arteriovenous fistula," "intracranial arteriovenous fistula," and "craniotomy arteriovenous fistula." The possible causes of the AVF were assessed with a focus on the possible pathomechanism that could indicate an acquired lesion, such as surgery, endovascular intervention, or head trauma.

Several clinical characteristics were extracted and analyzed, including feeding artery, superficial and final venous drainage, arteriovenous junction location, initial presentations leading to the diagnosis, spontaneous bleeding, presence of venous varix or pseudoaneurysm, potential hematologic abnormalities leading to fistula formation, sentinel events, diagnosis interval after sentinel events, treatment, outcome, and the authors' hypothesis regarding fistula formation (Table 1). In addition to these factors, comparison of the documented angiographic outcomes of the acquired lesions with a previously reported primary series is summarized (Tables 2 and 3).

Statistical analyses and data processing were performed using SAS software version 9 (SAS Institute Inc., Cary, North Carolina, USA). Descriptive statistics were presented as frequencies and/or percentages. Several clinical variables, including age, presentation, arterial feeder, and venous varix/pseudoaneurysm were compared using a Fisher exact test. A value of $P < 0.05$ was considered statistically significant.

Table 2. Clinical Comparisons Between Acquired Pial Arteriovenous Fistula and Primary Intracranial Single-Channel Pial Arteriovenous Fistula

	Acquired Pial Arteriovenous Fistula (n = 8)	Primary Pial Arteriovenous Fistula (n = 83)	*P* Value (Fisher Exact Test)
Age group			
Adult	8	40	0.0061
Pediatric	0	43	
Presentation			
Symptomatic	3	83	<0.0001
Asymptomatic	5	0	
Mass effect	0	38	0.0189
Hemorrhage	1	27	0.4269
Seizure	2	23	0.9999
Arterial feeder*			
Single	8	64	0.3445
Multiple	0	17	
Venous varix or pseudoaneurysm	2	64	0.0049

*Two unspecified cases in the original report (Yang et al.)[3]

RESULTS

Including the patient we presented, we identified 8 cases of acquired pAVF formation with different possible causes (Table 1) based on our literature review.[4-10] Most (7/8, 87.5%) were related to previous intervention procedures (including open surgery or endovascular surgery). The time interval between the sentinel event related to the formation of pAVF (or suspicious neurosurgical/traumatic events) and the angiographic diagnosis ranged from <1 day to 21 months. More than half of the cases were asymptomatic (5/8, 62.5%) and were found during follow-up DSA of a previously treated vascular lesion. Three patients with symptomatic lesions presented with an intolerable headache, seizures, or even status epilepticus. Only 1 patient presented with subarachnoid hemorrhage (1/8, 12.5%). Although the possibility of posttraumatic seizure as a result of cortical injury does exist, the imaging evidence indicated marked perilesional cerebral edema resulting from the fistula, and this may have contributed significantly to the development of seizures in the case with head trauma instead of previous neurosurgical procedures.[5]

Based on the pattern of the arterial feeders and venous drainage, 1 lesion was fed from distal branches of the anterior cerebral artery and drained into the superior sagittal sinus, which is anatomically related to the possibility of venous injury via insertion of an external ventricular drainage.[8] Most of these lesions (7/8, 87.5%) involved blood flow from the distal branches of the MCA and drained via superficial middle cerebral veins (superficial sylvian vein). The patient we presented had the only lesion found with pseudoaneurysm formation confirmed angiographically.

Four of the 8 lesions were managed conservatively, with the other 4 treated microsurgically. The asymptomatic lesion, which was found at 21 months (longest diagnosis interval in our review series) after craniotomy for aneurysm clipping, was observed for 2 more years after angiographic diagnosis, and no associated adverse event was reported. Another case that was managed conservatively was diagnosed 18 months after an initial aneurysm coiling procedure. However, no data for the observed period, growth of the lesion size, or other associated symptoms were available in that report. Feroze et al. reported an asymptomatic bypass-related pAVF found 6 months after the procedure, which was observed because of small arteriovenous shunting. The last case managed conservatively presented with status epilepticus after spinal surgery, which had to be managed before possible

Table 3. Reported Treatment and Angiographic Evidence of Complete Obliteration

	Acquired Pial Arteriovenous Fistula (N = 8) (n)	Digital Subtraction Angiography Complete Obliteration (n)	Obliteration Rate (%)	Primary Pial Arteriovenous Fistula (N = 91) (n)*	Digital Subtraction Angiography Complete Obliteration (n)	Obliteration Rate (%)
Microsurgery	4	4	100	40	39	97.5
Embolization	0	0	NA	37	32	86.5
Hybrid procedure	0	0	NA	14	12	85.7
Conservative	4	1	25	0	0	NA

NA, not available.
*From Yang et al.[3]

surgical intervention, which prevented the possibility of treating the lesion at an early stage after the diagnosis was made. However, the lesion was shown to have resolved spontaneously in the 2-week follow-up angiography; hence, no neurosurgical procedure was performed. The 3 cases diagnosed in the early phase after primary treatment (<2 weeks) were treated microsurgically. Another patient diagnosed 18 months after primary treatment had received microsurgery for disconnection of the pAVF. More than half of the cases (5/8, 62.5%) were intended to be treated with open microsurgery.

Comparison of several clinical characteristics of the acquired lesions with a previously reported primary series is presented in Table 2. None of the acquired lesions was found in the pediatric group compared with >50% of cases in the primary group ($P = 0.0061$). As previously described, >50% of pAVFs that proved to be acquired were related to neurosurgical or neuroendovascular procedures, and these were diagnosed while the patient was asymptomatic ($P < 0.0001$). Fewer venous varix formations and the associated intracranial mass effect were found in the cases of acquired pAVF ($P = 0.0049$ and $P = 0.0189$, respectively). The reported possible treatment modalities and their obliteration rates are listed in Table 3. Because of the relatively slow shunting, lack of varix formation, and possible asymptomatic clinical behavior, almost half of the acquired lesions were managed conservatively. However, the obliteration rate in the surgical intervened cases was good. The treatment selection among primary lesions was more diverse.

DISCUSSION

Acquired pAVF versus Primary pAVF

Primary pAVF may present in childhood; however, a delayed diagnosis because of a previously asymptomatic course may be the reason why only around 50% of patients in previous reports were identified as pediatrics.[1,3] Primary pAVF was once regarded as an alternative subgroup of AVM, but increasing evidence has indicated that pAVF should be considered a unique disease entity.[2] In this review, we focused on the cases that presented with distinct evidence of de novo formation of pAVF by DSA. However, it is difficult to differentiate the possible mechanism of previously reported pAVF series, and the potential growth of the fistula and varix formation cannot be completely ruled out if the diagnoses are delayed.

One hypothesis includes a possible misstep during embryologic development of cerebrovascular structures that may generate these lesions. Abnormal angiogenesis and associated vascular growth factors and cytokines may confer a higher frequency of venous varix formation in this group of patients.[1] However, in the review series presented here, lack of venous varix formation is observed in these de novo lesions. An insufficient amount of time may lead to this misclassification, yet 3 cases in this review series (3/8, 37.5%) were diagnosed >1 year after possible sentinel events. This finding supports the idea that acquired pAVF tends to be low flow and rarely progresses into varix formation and, thus, should be considered as an alternative category in this rare disease.

Clinical Characteristic Differences Between Acquired pAVF and Primary pAVF

pAVF, when considered primary, can present with various symptoms, such as hemorrhage, seizure, neurologic deficits, headache, bruit, cardiac failure in neonates and infants, symptoms of increased intracranial pressure, and mass effect caused by enlarged venous varices, or as part of other rare syndromes, such as Rendu-Osler-Weber (hereditary hemorrhagic telangiectasia), Klippel-Trenaunay-Weber, or Elher-Danlos syndromes.[1] However, in primary pAVF, the dilation of the venous channel plays a significant role in the signs and symptoms. The venous lesion may cause a mass effect with focal compression, causing different neurologic deficits depending on the neuroanatomic location in which the focal mass effect resides. In acquired pAVF, such phenomena are rarely reported because of the relatively small-caliber venous system.

The natural course and aggressiveness of acquired pAVF are unknown, and the high morbidity and mortality complicate the possible treatment of primary pAVF.[3,11] Yang et al.[3] reported a periprocedural morbidity of 38% during treatment of these symptomatic lesions. Despite an unknown long-term natural history, another study[11] estimated the possible mortality might be as high as 63% after conservative management.

Compared with hemorrhagic lesions with high cortical reflux and venous varices, only 1 of the acquired lesions was reported in conjunction with spontaneous bleeding episodes (subarachnoid

hemorrhage). Three of the lesions in this series were managed conservatively, and despite no imaging evidence, no identifiable adverse events were reported. Spontaneous regression was found in 1 case, which indicated the possible benign course of such lesions.[4] Angiographic diagnoses were delayed >1 year after sentinel events in almost half of the acquired lesions, with the patients remaining asymptomatic. The overall outcome after surgical or conservative management was good (7/8, 87.5%, and 1 unknown follow-up course). It is reasonable to consider such occurrences as relatively benign lesions with low progression rates.

Relationship Between Massive CSF Drainage, Brain Shift, and Cortical Drainage Vein Injury

Four craniotomies (2 for aneurysm clipping, 1 for bypass surgery, and 1 for AVM resection) were complicated with the de novo formation of pAVF. CSF drainage for a wider intracranial space and working angle is anticipated in these procedures. In our case, hematoma removal resulted in a significant increase in surgical space for AVM manipulation; however, the subdural space was enlarged as well. Constant irrigation, avoidance of excessive surgical table and surgical field turning or shifting, and adequate use of a traction device to avoid excessive slacking of the brain may help to decrease the potential for venous injury.

Venous Occlusion, Recanalization, and Recruitment of Regional Arterial Blood Flow after Injury as a Potential Pathophysiologic Mechanism and Its Anatomic Consideration

The correlation between dural sinus thrombosis and the formation of dural arteriovenous fistulas is well established.[12,13] However, such a mechanism does not fully explain the possible formation of primary pAVF. When the fistula is formed as a result of venous occlusion, multiple channels along the affected sinus are anticipated, yet many primary pAVFs are single-channeled. Newton et al.[14] once classified intracranial AVMs into 3 categories based on the feeding arteries: pial, dural, and mixed pial-dural. Because the dural sinus system is the eventual route of the intracerebral and dural venous return, obstruction along this route could result in different anatomic levels of fistula formation.

In our review, many of these lesions were located along the course of superficial middle cerebral veins. This anatomic coincidence may reflect the possible proximity between the distal potential arterial feeders and the superficial venous system. The recanalization of the superficial middle cerebral veins and its related bridging veins or anastomotic veins plays a major role in the formation of these acquired lesions because it covers most of the superficial cortical area.

Despite some focal intraoperative insults (direct suture, external ventricular drainage puncture, or inflammatory processes caused by hemostatic materials) that have been used to explain the possible formation of pAVF, still >50% of acquired pAVF have likely resulted from possible venous injury and/or occlusion. Notwithstanding that various possible mechanisms have been previously proposed (e.g., hemostatic materials used in venous injury, direct suture, trauma, and venous injury during CSF drainage), all these events are linked to a common pathomechanism pathway: "Venous occlusion, recanalization, recruitment of arterial blood, and final fistula formation." This "aberrant vascular reparative response" after vascular injury seems to be crucial in the formation of pAVF; a similar mechanism has been shown to evoke the formation of dural arteriovenous fistulas. However, how frequent these fistulas develop after venous injury is yet to be determined because of the disease rarity and no identifiable risk factor was found in a literature review. Cautious management of patients with possible venous damage in these regions may assist in the early and proper diagnosis of these lesions.

Possible Management Strategies and Indications for Treatment

All the acquired pAVFs were located cortically and drained into the cerebral sinus system via the superficial venous system. No deep venous drainage was found in this review, and the arterial feeders were exclusively from the very distal segment of the MCA or anterior cerebral artery. Considering the possible significance of the feeders, size of the fistula, the simplicity of the fistula structure, and accessibility of the lesions microsurgically or endovascularly, these cases were managed exclusively with microsurgery or conservatively. There is no consensus for the indication of treatment for these lesions; however, a microsurgical approach is reasonable if the lesion was diagnosed perioperatively after craniotomy because the lesion could be easily located via the previous surgical route. Another perisurgical consideration could be the unknown natural course compared with primary pAVF. There are few reports concerning these acquired lesions, and no report regarding the nature history exists that could be used for risk evaluation. Our review found that half of the reported cases were managed conservatively, indicating a relatively benign course that is different from the primary pAVFs.[4,6,9,10] Based on the possible pathophysiologic mechanisms discussed previously, significant reflux to other cortical venous systems, the presence of venous varix, and even pseudoaneurysm formation could be regarded as potential indications for further definite surgical management.[15,16]

CONCLUSIONS

Acquired pAVF is highly correlated with sentinel neurosurgical procedures or venous occlusion events. Whatever these sentinel events, venous occlusion and recanalization seem to be the final pathomechanism, which is similar to the formation of dura AVF. Microsurgically aggressive disconnection of the fistula usually results in a good surgical outcome, yet many of these lesions present with a benign course or even spontaneous occlusion. Pseudoaneurysm formation or other signs of significant cortical venous reflux could be considered as potential surgical indications. The posterior temporal region is found to be an anatomically predominant site for the development of these lesions.

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