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Review

Cortical plasticity in patients with cerebral arteriovenous malformations

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ABSTRACT

The aim of this review is to ascertain the evidence for cortical plasticity in arteriovenous malformation (AVM) patients. Chronic hypoperfusion due to vascular steal from cerebral AVM can result in a translocation of eloquent neurological functions to other brain areas, a phenomenon known as cortical plasticity. We performed a systematic literature review of the studies that have evaluated cortical plasticity in AVM patients. A total of 22 studies from 1996 to 2014 were included for the analyses. The evaluation of cortical plasticity was performed prior to AVM intervention in 109 patients, and during or after AVM intervention in 18. The most commonly assessed neurological functions were motor in 85% and language in 11% of the former cohort, and motor in 78% and language, cognition, and memory each in 39% of the latter cohort. Functional MRI was the most frequently used method for evaluating cortical plasticity, and was performed in 63% of the former and 56% of the latter cohort. In conclusion, cortical plasticity appears to be influenced by both AVM pathogenesis and intervention. Given the limited evidence that is currently available for cortical plasticity in AVM patients, further studies are warranted to determine its incidence and impact on long term clinical outcomes.

1. Introduction

Cerebral arteriovenous malformations (AVM) are rare congenital lesions in which blood flow is shunted from feeding arteries to draining veins through a pathological nidus, rather than through the normal capillary network of the brain [1]. The pathophysiology of AVM is complex and incompletely understood [2–11]. Although there is no functional brain within a nidus, AVM frequently cause neurological symptoms by affecting the surrounding parenchyma via rupture, development of perinidal gliosis, or shunting of the blood supply [1,12,13]. The chronic vascular steal caused by AVM can remodel the surrounding cortex, which may alter its physiology and function [14]. Specifically, AVM located in or adjacent to eloquent brain regions, such as primary motor and somatosensory, speech and language, and visual cortex, can result in the translocation of these eloquent functions to neighboring cortical areas [15]. This transference of neurological function from eloquent to non-eloquent brain regions is related to the phenomenon of cortical plasticity. The goal of this review is to analyze the evidence for cortical plasticity in patients with AVM.

2. Methods

We performed a systematic literature review of the PubMed database using the following search strategy "arteriovenous malformation AND plasticity". All case reports and case series, published prior to June 2015, related to the evaluation of cortical plasticity before, during, or after AVM treatment were included for the analyses. Any non-English language reports were excluded. Additionally, within each case series, patients with intracranial neoplasms and vascular malformations other than AVM, such as cerebral cavernous malformations and dural arteriovenous fistulas, were excluded.

The included studies were then divided into those that demonstrated evidence of AVM-associated cortical plasticity prior to intervention, and those that demonstrated evidence of it during or after the intervention. The following variables were ascertained from each study, when available: the number of patients, AVM location, Spetzler–Martin grade, neurological function of interest, and modality for evaluation of cortical plasticity [16]. For the studies that evaluated cortical plasticity during or after AVM intervention, the type of intervention was also recorded. The continuous data were reported as the mean, median, and range, and categorical data were reported as frequency.

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3. Results

The search strategy yielded a total of 39 articles. One article was excluded for being presented in a language other than English (French), and 16 articles were excluded for a lack of relevance to the topic. A total of 22 studies, published from 1996 to 2014, were included in the analyses.

3.1. Evidence for cortical plasticity prior to AVM intervention

We identified a total of 10 studies, including nine case series and one case report, describing cortical plasticity prior to AVM intervention (Table 1) [17–26]. These studies comprised a total of 109 patients, and the mean number of patients per study was 10.9, with a median of 9.5 (range: 1–23). The location of 31 of the AVM was not reported (28.4%). Of the 78 AVM with reported locations, the most common locations were frontal for 21 (26.9%), frontoparietal for 20 (25.6%), and temporal for 15 AVM (19.2%). The less common locations were parietal for five (6.4%), frontotemporal for four (5.1%), temporoparietal and callosal for three each (3.8%), frontotemporoparietal and parieto-occipital for two each (2.6%), and occipitotemporal, basal ganglia, and ventricular in one AVM each (1.3%).

The Spetzler–Martin grade was not reported in 93 AVM (85.3%). Of the 16 AVM for which the Spetzler–Martin grade could be determined, one was grade I (6.3%), five were grade II (31.3%), eight were grade III (50.0%), two were grade IV (12.5%), and none were grade V. The neurological functions that were assessed for cortical translocation were motor in 93 (85.3%), language in 12 (11.0%), and speech in four (3.7%) patients. The modality for detecting cortical plasticity was functional MRI (fMRI) in 69 (63.3%), diffusion tensor imaging in 23 (21.1%), the Wada test in 13 (11.9%), and transcranial magnetic stimulation in 10 (9.2%) patients. Six patients were evaluated by both fMRI and Wada testing (5.5%).

3.2. Evidence for cortical plasticity during or after intervention

We identified a total of 12 studies, including two case series and 10 case reports, which described cortical plasticity during or after AVM intervention (Table 2) [15,27–37]. These studies comprised a total of 18 patients, and the mean number of patients per study was 1.5, with a median of 1 (range: 1–5). The AVM interventions were surgical resection in 15 (83.3%), embolization in seven (38.9%), and stereotactic radiosurgery (SRS) in three (16.7%) patients. Five patients underwent two interventional modalities (27.8%), including combined preoperative embolization with subsequent surgical resection in four patients, and surgical resection of a residual nidus after initial SRS in one. One patient underwent all three interventional modalities (5.6%).

The AVM locations were frontal in nine (50.0%), parietal in four (22.2%), temporal in three (16.7%), and callosal and cerebellar each in one (5.6%) patient. The Spetzler–Martin grade was not reported for 13 AVM (72.2%). Of the five AVM for which the Spetzler–Martin grade could be determined, three were grade II (60%), one was grade IV (20%) and one grade V (20%). The neurological functions assessed for cortical translocation were motor in 14 (77.8%), language, cognition, and memory each in seven (38.9%), and musical ability in one (5.6%) patient. Four neurological functions were assessed in five patients (27.8%), and two neurological functions were assessed in three (16.7%). The modality for detecting cortical plasticity was fMRI in 10 (55.6%), neuropsychological evaluation in eight (44.4%), direct cortical stimulation (DCS) and MRI (for calculation of tissue volume) each in two (11.1%), and positron emission tomography in one (5.6%) patient. Five patients were evaluated by two modalities (5.6%).

4. Discussion

AVM most frequently present with intracranial hemorrhage, seizures, and focal neurological deficits, with an annual hemorrhage risk of approximately 2–4% [38–42]. Since many AVM present by the third or fourth decades of life, the patients who harbor these lesions are exposed to a significant lifetime risk of hemorrhage [1]. The current treatment options for AVM include surgical resection, SRS, and embolization, either alone or in combination. Surgical resection provides an immediate cure with concomitant elimination of the hemorrhage risk, and is the gold standard of treatment. Surgery yields the best results for small to medium sized compact nidi that are located in non-eloquent brain regions, without deep venous drainage [16,43–46]. SRS is generally the favored treatment for nidi in deep or eloquent cortical locations, although its efficacy decreases with increasing AVM volume [46–70]. Additionally, AVM obliteration typically occurs over a latency interval of 2–3 years, during which the nidus remains at a risk for rupture. Embolization is most commonly used for preoperative devascularization or volume reduction of large AVM prior to surgical resection or SRS, respectively, although it can also be curative in a carefully selected subset of nidi [71–76].

Ruptured AVM clearly require treatment, due to an increased risk of a subsequent hemorrhage after the initial rupture [77,78]. In contrast, the benefit of treatment for unruptured AVM is a subject of debate, given the findings from recently published prospective studies, the randomized trial of unruptured brain AVM (ARUBA) and the Scottish audit of intracranial vascular malformations (SAIVM) AVM study [79–81]. Cortical plasticity may allow for

Table 1
Summary of studies analyzing cortical plasticity before AVM intervention

Study	Patients, n	AVM locations (n)	SM Grade (n)	Neurological functions evaluated (n)	Modality for detecting cortical plasticity (n)
Kato et al. (2014) [17]	10	F (3), FP (3), T (3), PO (1)	I (1), II (3), III (5), IV (1)	Motor (6), speech (4)	TMS
Lee et al. (2013) [18]	22	F (6), NR (16)	NR	Motor	fMRI
Fu et al. (2010) [19]	23	T (8), NR (15)	NR	Motor	DTI
Baciu et al. (2003) [20]	1	F	NR	Motor	fMRI
Ozdoba et al. (2002) [21]	10	BG (1), C (3), F (2), P (2), TO (1), V (1)	NR	Motor	fMRI (all), Wada (6)
Carpentier et al. (2001) [22]	16	F (4), FP (8), FT (2), P (2),	NR	Motor	fMRI
Alkadhi et al. (2000) [23]	9	F (1), FP (8)	NR	Motor	fMRI
Vikingstad (2000) [24]	5	FT (2), T (1), TP (1), FTP (1)	NR	Language	fMRI
Lazar et al. (1997) [25]	7	F (1) T (1), P (1), FP (1), TP (2), PO (1)	II (2), III (3), IV (1), NR (1)	Language	Wada
Maldjian et al. (1996) [26]	6	F (3), T (2), FTP (1)	NR	Motor	fMRI

AVM = arteriovenous malformation, BG = basal ganglia, C = cerebellar, DTI = diffusion tensor imaging, F = frontal, fMRI = functional magnetic resonance imaging, NPE = neuropsychological evaluation, NR = not reported, O = occipital, P = parietal, SM = Spetzler–Martin, T = temporal, TMS = transcranial magnetic stimulation, V = ventricular.