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Color Doppler ultrasonography in the evaluation of compensatory arteries in patients with moyamoya disease: combined with cerebral angiography

H.-W. PAN, L. CHEN, H.-Q. JIANG, Z. YE, Y. WANG, Y. WANG

Department of Ultrasound and Department of Neurosurgery, Huashan Hospital Affiliated to Fudan University, Shanghai, China

Abstract. – OBJECTIVE: To evaluate the hemodynamics of maxillary artery (MA), superficial temporal artery (STA) and ophthalmic artery (OA), and evaluate the hemodynamics prediction capability of these arteries formed compensatory arteries into intracranial using Doppler ultrasonography.

PATIENTS AND METHODS: The evaluation of MA, STA, and OA with transcranial doppler ultrasonography and with cerebral angiography was made in 106 MMD patients (total of 212 hemispheres of the brain), 58 male and 48 female, aged 39.3±12.0 years old. Doppler ultrasonography measured the following blood flow parameters: peak systolic velocity (PSV), end-diastolic velocity (EDV), and resistance index (RI). DSA evaluate whether MA, STA, OA formed compensatory arteries into intracranial. Based on the compensation situation, the patients were divided into two groups: compensatory group and non-compensatory group. The differences of patient's hemodynamic parameters between compensatory and non-compensatory groups were performed using independent two-sample t-tests with equal or non-equal variance as appropriate. Categorical variables were summarized using frequency and percentage and compared using Chi-square tests. We evaluated the prediction ability of each hemodynamic parameters for each artery (combining left and right side) using Receiver Operating Curve. All the analyses were performed using SAS 9.4 (Cary, NC).

RESULTS: Comparing the hemodynamic parameters between the compensatory group and non-compensatory group, all hemodynamic parameters of MA, STA and OA have statistically significant differences between the two groups. Depending on the ROC Curve, EDV (AUC=0.6933±0.0463) for MA, RI (AUC=0.8910±0.0569) for STA, EDV (AUC=0.7863± 0.0330) for OA are better predictors of compensatory growth.

CONCLUSIONS: Color duplex ultrasonography is a reliable, noninvasive and economic tool to assess hemodynamic changes of MA, STA and OA, and has prediction capability of these arteries formed compensatory arteries into intracranial.

Key Words:
Moyamoya disease, Transcranial Doppler ultrasonography, Cerebral angiography, Hemodynamics.

Introduction

Moyamoya disease (MMD) is an uncommon cerebrovascular disorder named by Suzuki and Takaku[1] because "moyamoya" means "puff of smoke" in Japanese. According to the guidelines for the diagnosis of moyamoya disease by the Research Committee on Spontaneous Occlusion of the Circle of Willis[2], moyamoya disease is characterized by stenosis or occlusion at the terminal portions of the ICA or the proximal areas of the anterior or the middle cerebral arteries (ACAs, MCAs) and abnormal vascular networks in the arterial territories near the occlusive or stenotic lesions. According to previous studies[3,4], the incidence rate of MMD is higher in East Asia, the highest known incidence is in Japan. The female to male ratio was shown to be 1.8:1, which means MMD was more prevalent in women than men. The most common clinical manifestations of MMD are ischemic strokes and TIAs, other clinical features include intracerebral hemorrhages, headaches and seizures[5,6].

There are several imaging techniques used to assess cerebral hemodynamics of MMD[6-9], including digital subtraction angiography (DSA), positron emission tomography (PET), single-photon emission computed tomography (SPECT), xenon computed tomography, computed tomography perfusion (CTP), perfusion-weighted magnetic resonance imaging and Doppler ultrasonography. DSA remains the gold standard imaging technique for the diagnosis of moyamoya disease[3,4]. However, Doppler ultrasonography combines both static and dynamic information, repre-

Corresponding Author: Yong Wang, MD; e-mail: dr_wy@189.cn
Yi Wang, MD; e-mail: y_wang1111@hotmail.com

sents a noninvasive, real-time, easily repeatable and economic method[4,5,10-12]. Until now, there are no effective medical therapies for MMD, several types of operative approaches can be used to improve patients' clinical outcome, including direct revascularization such as superficial temporal artery (STA) to superficial branches of the middle cerebral artery (MCA); indirect approaches including encephalo duro arterio synangiosis (EDAS) and encephalo myosin angiosis (EMS) which can facilitate the growth of a new vascular network[13-16]. In our hospital (with the maximum number of MMD patients in Eastern China), we also combine direct and indirect bypass operations for adult MMD. As we know, MMD is characterized by the extensive development of pathognomonic collateral pathways in response to steno-occlusive changes[17]. Among these pathways, "ethmoidal moyamoya" involves dilation of the anterior and posterior ethmoidal arteries, which also function as collateral pathways, mainly from the ophthalmic arteries (OA) to the ACA branches[4,17], extracranial to intracranial compensatory pathways including STA, Supraorbital artery, the circle of Willis and maxillary artery (MA), middle meningeal artery (MMA), the circle of Willis[18,19]. Assessing the development of collateral pathways before the operation is very important for choosing surgical approach[20,21]. But so far no studies focus on the use of Doppler ultrasonography in the hemodynamics of these collateral pathways[2]. Since the location of Supraorbital artery and MMA (supplying temporal muscles) are deeper that ultrasonography can hardly detect them, their superiors vessels MA and STA are easy to examine, the hemodynamics of MA and STA can indirectly reflect the hemodynamics of Supraorbital artery and MMA. Therefore, the purpose of our study was to evaluate the hemodynamics (PSV, EDV, RI) of MA, STA and OA, and evaluate the hemodynamics prediction ability of these arteries formed compensatory arteries into intracranial using Doppler ultrasonography.

Patients and Methods

The study protocol was approved by the Commission on Scientific Research of Human Subjects of Huashan Hospital Affiliated to Fudan University. Written informed consent was obtained from each patient.

Patients

From May 2012 to Jan 2014, 106 MMD patients (total of 212 hemispheres of the brain) admitted by Huashan Hospital, 58 male and 48 female, aged 39.3±12.0 years old were included in this study. The diagnosis of MMD was confirmed by DSA.

Methods

We used a high-end ultrasound device (Philips Healthcare, Andover, MA, USA) with an L9-3 Linear probe (bandwidth, 3-9 MHZ) and a C5-1 abdomen probe (bandwidth, 1-5 MHz) to get Duplex ultrasonography images. Patients were in the dorsal decubitus position. The trunk of STA was measured before it was divided into the frontal and parietal branch. We located the end of STA above the tragus and the initial segment of MA behind the mandibular angle. We also located the end of OA through the ocular window. The following blood flow parameters were measured: peak systolic velocity (PSV), end-diastolic velocity (EDV), and resistance index (RI) (Figure 1). To be consistent and comparable in duplex sono-

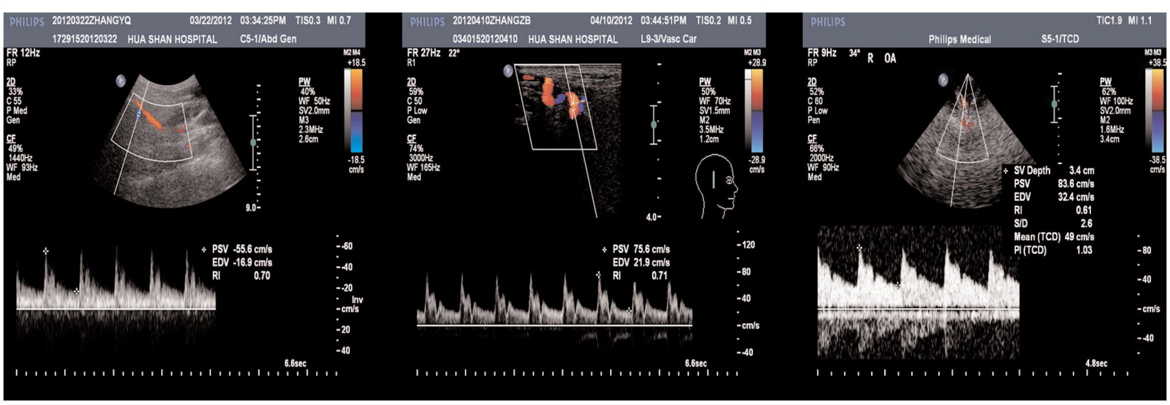

Figure 1. Color Doppler flow imaging, frequency spectrogram, and blood flow parameters. (A) MA. (B) STA. (C) OA.

graphic results, all the tests were done by a single board-certified sonographic radiologist.

We use DSA to evaluate whether or not MA, STA, OA formed compensatory arteries into intracranial. Based on the compensation situation, the patients were divided into two groups: compensatory group and non-compensatory group (Figure 2).

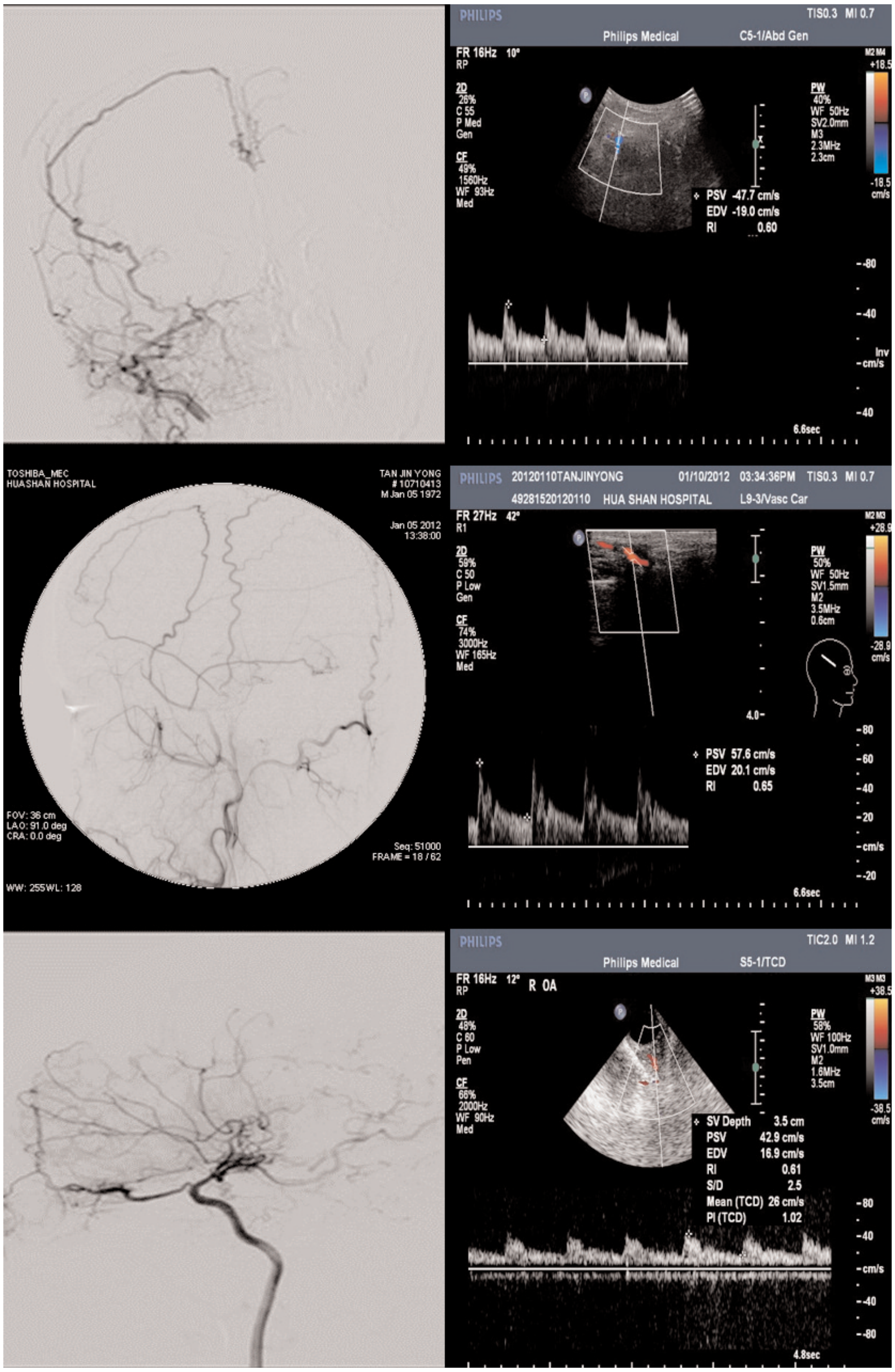

Figure 2. The right side compensated MA in DSA (A, left) and color Doppler flow parameters of the right MA with the same patient (A, right). The right side compensated STA in DSA (B, left) and color Doppler flow parameters of the right STA with the same patient (B, right). The right side compensated OA in DSA (C, left) and color Doppler flow parameters of the right OA with the same patient (C, right).

Table I. Patients characteristics and hemodynamic parameters in the MA.

	Side	Compensatory group (N=47)	Non-Compensatory group (N=165)	*p*-value
Number	Left	24	82	
	Right	23	83	
Age (year)	Left	35.75 (13.60)	40.38 (11.40)	0.098
	Right	37.39 (11.99)	39.87 (12.05)	0.385
Female	Left	13 (54.17%)	35 (42.68%)	0.320
	Right	11 (47.83%)	37 (44.58%)	0.782
PSV (cm/s)	Both	64.68 (18.55)	58.19 (14.91)	0.015
EDV (cm/s)	Both	18.14 (6.40)	14.14 (4.46)	<0.001
RI	Both	0.714 (0.082)	0.754 (0.059)	<0.001

PSV, peak systolic velocity; EDV, end-diastolic velocity; RI, resistance index.

Statistical Analysis

Patient's characteristics and hemodynamic parameters were summarized by the compensatory growth status of each side of each artery. For continuous variables, normal distribution assumption was assessed. Equal variance assumption was assessed. The differences of these characteristics between compensatory and non-compensatory groups were performed using independent two-sample *t*-tests with equal or non-equal variance as appropriate. Categorical variables were summarized using frequency and percentage and compared using Chi-square tests. We evaluated the prediction ability of each hemodynamic parameters for each artery (combining left and right side) using Receiver Operating Curve. We constructed the curve using a Generalized Estimating Equation (GEE) model with the compensatory status as the outcome, each hemodynamic parameter as the predictor. A logistic link was used in the GEE models. Area Under Curve (AUCs) of ROC was calculated and compared between the hemodynamic parameters using Chi-square test. All the analyses were performed using SAS 9.4 (Cary, NC, USA).

Results

Table I depicted the comparison of hemodynamic parameters (PSV, EDV, RI) in the MA between compensatory group (N=47) and non-compensatory group (N=165). Compared with the non-compensatory group, the PSV value (p=0.015) and EDV value (p<0.001) were significantly higher in the compensatory group, the RI value (p<0.001) was significantly lower in the compensatory group. The ROC curve of PSV, EDV and RI are illustrated in Table II and Figure 3. The AUCs of PSV, EDV and RI are 0.5995, 0.6933, and 0.6413. The AUCs of PSV (p=0.5855) and EDV (p=0.2168) compared to the AUC of RI are not statistically significant. EDV (AUC=0.6933±0.0463) is a better predictor of compensatory growth.

Table III depicted the comparison of hemodynamic parameters (PSV, EDV, RI) in the STA between compensatory group (N=16) and non-compensatory group (N=196). Compared with the non-compensatory group, the PSV value (p=0.005) and EDV value (p<0.001) were significantly higher in the compensatory group, the RI

Table II. Prediction of compensatory growth (MA) by PSV, EDV and RI.

	AUC (SD)	95% CI of AUC	*p*-value compared to RI
PSV	0.5995 (0.0492)	(0.5031, 0.6960)	0.5855
EDV	0.6933 (0.0463)	(0.6024, 0.7841)	0.2168
RI	0.6413 (0.0502)	(0.5429, 0.7398)	

PSV, peak systolic velocity; EDV, end-diastolic velocity; RI, resistance index.

Table III. Patients characteristics and hemodynamic parameters in the STA.

	Side	Compensatory group (N=16)	Non-Compensatory group (N=196)	*p*-value
Number	Left	8	98	
	Right	8	98	
Age (year)	Left	41.88 (4.09)	39.12 (12.44)	0.166
	Right	45.38 (4.44)	38.83 (12.32)	0.0043
Female	Left	5 (62.50%)	43 (43.88%)	0.309
	Right	6 (75%)	42 (42.86%)	0.079
PSV (cm/s)	Both	78.75 (26.46)	65.85(16.54)	0.005
EDV (cm/s)	Both	28.69 (9.92)	17.28 (5.00)	<0.001
RI	Both	0.629 (0.629)	0.735 (0.057)	<0.001

PSV, peak systolic velocity; EDV, end-diastolic velocity; RI, resistance index.

Table IV. Prediction of compensatory growth (STA) by PSV, EDV and RI.

	AUC (SD)	95% CI of AUC	*p*-value compared to RI
PSV	0.6316 (0.0867)	(0.4616, 0.8014)	0.0131
EDV	0.8529 (0.0532)	(0.7487, 0.9571)	0.4164
RI	0.8910 (0.0569)	(0.7795, 1.0000)	

PSV, peak systolic velocity; EDV, end-diastolic velocity; RI, resistance index.

value ($p<0.001$) was significantly lower in the compensatory group. The ROC curves of PSV, EDV, RI are illustrated in Table IV and Figure 4. The AUCs of PSV, EDV and RI are 0.6316, 0.8529, and 0.8910. The AUC of PSV compared to the AUC of RI is statistically significant ($p=0.0131$), and the AUC of EDV compared to the AUC of RI are not statistically significant ($p=0.4164$). RI (AUC=0.8910±0.0569) is a better predictor of compensatory growth.

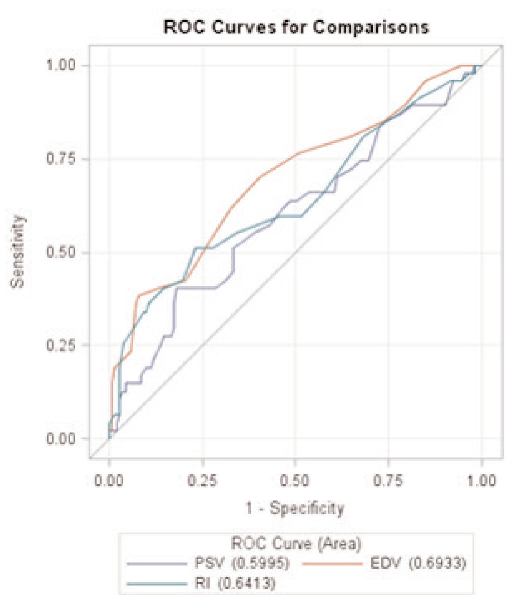

Figure 3. ROC curve using GENMOD (Generalized Estimating Equations) of MA.

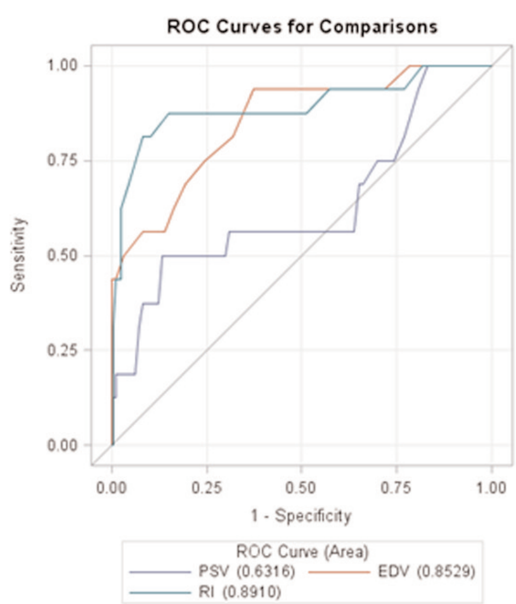

Figure 4. ROC curve using GENMOD (Generalized Estimating Equations) of STA.

Table V. Patients characteristics and hemodynamic parameters in the OA.

	Side	Compensatory group (N=84)	Non-Compensatory group (N=128)	*p*-value
Number	Left	41	65	
	Right	43	63	
Age (year)	Left	36.71 (11.65)	40.98 (12.05)	0.074
	Right	35.81 (11.27)	41.73 (12.02)	0.012
Female	Left	19 (46.34%)	29 (44.62%)	0.862
	Right	21 (48.84%)	27 (42.86%)	0.544
PSV (cm/s)	Both	64.61 (15.90)	51.63 (16.93)	<0.001
EDV (cm/s)	Both	28.86 (10.13)	19.86 (8.32)	<0.001
RI	Both	0.557 (0.091)	0.608 (0.096)	<0.001

PSV, peak systolic velocity; EDV, end-diastolic velocity; RI, resistance index.

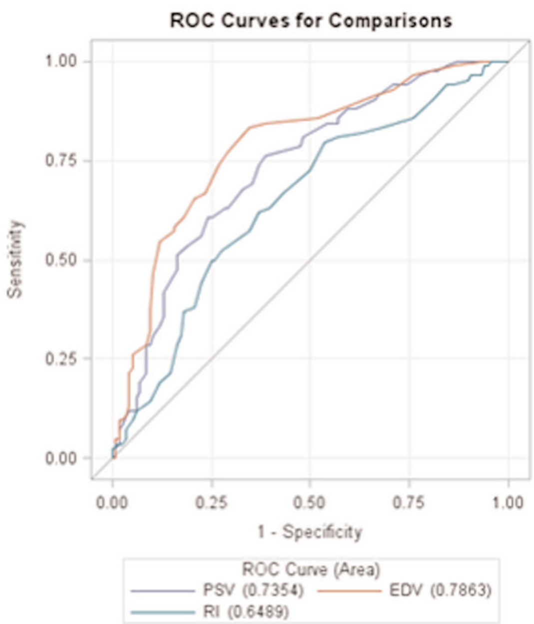

Figure 5. ROC curve using GENMOD (Generalized Estimating Equations) of OA.

Table V depicted the comparison of hemodynamic parameters (PSV, EDV, RI) in OA between compensatory group (N=84) and non-compensatory group (N=128). Compared with the non-compensatory group, the PSV ($p<0.001$) and EDV ($p<0.001$) values were significantly higher in the compensatory group, the RI value were significantly lower ($p<0.001$) in the compensatory group. The ROC curves of PSV, EDV and RI are illustrated in Table VI and Figure 5. The AUCs of PSV, EDV and RI are 0.7354, 0.7863, and 0.6489. The AUC of PSV compared to the AUC of RI is not statistically significant ($p=0.0938$), and the AUC of EDV compared to the AUC of RI is statistically significant ($p<0.001$). EDV (AUC=0.7863± 0.0330) is a better predictor of compensatory growth.

Discussion

Although the incidence rate of moyamoya disease is not high in most global countries, it is an important reason of cerebral stroke in children and adults. Early and prompt diagnosis, and appropriate management are crucial to improve the long-term prognosis of patients[4]. At present, large sample studies on the MMD collateral circulation are quite few in the world.

As we know, it is important to understand the compensatory mechanism of the blood vessel in the patients with MMD disease. When the prima-

Table VI. Prediction of compensatory growth (OA) by PSV, EDV and RI.

	AUC (SD)	95% CI of AUC	*p*-value compared to RI
PSV	0.7354 (0.0335)	(0.6662, 0.8046)	0.0938
EDV	0.7863 (0.0330)	(0.7217, 0.8509)	<0.0010
RI	0.6489 (0.0394)	(0.5717, 0.7260)	

PSV, peak systolic velocity; EDV, end-diastolic velocity; RI, resistance index.

ry cerebral arteries suffer from thrombus formation, thromboembolic events, hemodynamic injury, etc., the body will promote the formation of collateral cerebral circulation in order to save the ischemic penumbra area of cerebral infarction and ease the symptom of chronic cerebral ischemia[22]. In the case of long-term ischemia, MMD may promote the formation of a variety of different types of collateral circulation. Cerebral angiogenesis and formation of collateral circulation penetrate the whole process of MMD, which may influence the prognosis of patients in a certain degree.

Preoperative color Doppler ultrasonography can easily know the collateral compensation situation. We considered MA, STA, OA as important compensatory arteries into intracranial, whether sufficient blood flow of these compensatory arteries can be formed is fundamental to the improvement of cerebral ischemia, and is a key concern in the long-term follow-up. More importantly, if patients need surgical treatment, we should protect the already established compensatory blood vessels no matter what kind of operation should be taken, in order to effectively reduce the complications of blood reconstruction surgery[21,23]. For example, when decide to do bypass (direct) or EDAS (indirect) surgery, to those patients who have STA collateral pathways, we should avoid injuring the frontal and parietal branch of the STA. In addition, Doppler ultrasonography monitoring of STA preoperative may predict the situation of patency of anastomosis in DSA in early postoperative period. So, color Doppler ultrasonography has been recommended in the diagnosis, surgical planning and post-surgical follow-up. However, further studies are necessary to identify the accurate relations between quantitative alteration of the ultrasound parameters and DSA results[24].

In our study, compare the hemodynamic parameters between the compensatory group and non-compensatory group, all hemodynamic parameters of MA, STA and OA have a statistical difference between the two groups, PSV and EDV values were significantly higher in the compensatory group, the RI value were significantly lower in the compensatory group. The reason may be that, as important branches of external carotid artery, STA and MA provide blood supply for the maxillofacial soft tissue, OA as important branch of internal carotid artery provide blood supply for eyeball and organa oculi accessoria after it through the optic nerve tube into orbital cavity, so they all have high resistance type blood flow spectrum. And the intracranial arteries have low resistance type blood flow spectrum because of the abundant anastomosis branches. When STA, MA, OA formed compensatory arteries into intracranial, increasing blood flow occurred since the pressure of the intracranial vessels was lower than that of extracranial vessels, their hemodynamic parameters changed and their blood flow spectrum changed from high resistance to low resistance. Depending on the ROC Curve, We also find that EDV (AUC=0.6933±0.0463) for MA, RI (AUC=0.8910±0.0569) for STA, EDV (AUC=0.7863± 0.0330) for OA are better predictors of compensatory growth.

There are some limitations in our retrospective study. First, the patients in the group came from the single center, a bias was possible in the selection. Second, we did not carry out postoperative ultrasonography long-term follow-up, so we can't observe the hemodynamic changes in these patients after the compensatory vascular surgery. Last, this study did not combine with the brain perfusion.

According to Sakamoto et al[20], ultrasonography was able to reveal alterations of vessels that appear normal on DSA, while it was not all DSA features could be found on ultrasound studies. Ultrasonography cannot provide maps of the extent and degree of cerebrovascular reactivity (CVR) impairment at the tissue level[18,25]. And Ultrasonography cannot diagnose moyamoya disease when it is accompanied by aneurysm[26,27], the reason is that arterial stenosis and occlusion and the abnormal vascular network of moyamoya disease may overlap with aneurysms. So further complementarities of various imaging tools and interpretation of non-overlapping findings are needed to improve the ability to detect the compensatory blood vessels[28]. Therefore, in the future study, we will combine a variety of imaging examinations, and carry out a long-term follow-up to improve the accuracy of quantitative analysis of Doppler Ultrasonography.

Conclusions

Color duplex ultrasonography is a reliable, noninvasive and economic tool to assess hemodynamic changes of MA, STA and OA, and has prediction capability of these arteries formed compensatory arteries into intracranial.

Acknowledgements

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Conflict of Interest

The Authors declare that they have no conflict of interests.

References

1) Suzuki J, Takaku A. Cerebrovascular "moyamoya" disease. Disease showing abnormal net-like vessels in base of brain. Arch Neurol 1969; 20: 288-299.

2) Fukui M. Guidelines for the diagnosis and treatment of spontaneous occlusion of the circle of Willis ('moyamoya' disease). Research Committee on Spontaneous Occlusion of the Circle of Willis (Moyamoya Disease) of the Ministry of Health and Welfare, Japan. Clin Neurol Neurosurg 1997; 99 Suppl 2: S238-240.

3) Burke GM, Burke AM, Sherma AK, Hurley MC, Batjer HH, Bendok BR. Moyamoya disease: a summary. Neurosurg Focus 2009; 26: E11.

4) Kuroda S, Houkin K. Moyamoya disease: current concepts and future perspectives. Lancet Neurol 2008; 7: 1056-1066.

5) Smith ER. Moyamoya arteriopathy. Curr Treat Options Neurol 2012; 14: 549-556.

6) Lee M, Zaharchuk G, Guzman R, Achrol A, Bell-Stephens T, Steinberg GK. Quantitative hemodynamic studies in moyamoya disease: a review. Neurosurg Focus 2009; 26: E5.

7) Tian B, Xu B, Liu Q, Hao Q, Lu J. Adult Moyamoya disease: 320-multidetector row CT for evaluation of revascularization in STA-MCA bypasses surgery. Eur J Radiol 2013; 82: 2342-2347.

8) Ma Y, Li M, Jiao LQ, Zhang HQ, Ling F. Contralateral cerebral hemodynamic changes after unilateral direct revascularization in patients with moyamoya disease. Neurosurg Rev 2011; 34: 347-353; discussion 353-344.

9) Han JH, Park YS, Lee WH, Koong SS, Min KS, Lee MS, Kim YG, Kim DH, Yi KS, Cha SH. Cerebral-perfusion-based single-photon emission computed tomography (SPECT) staging using NeuroGam(R) in patients with moyamoya disease. Childs Nerv Syst 2015 Dec 11. [Epub ahead of print].

10) Research Committee on the P, Treatment of Spontaneous Occlusion of the Circle of W, Health Labour Sciences Research Grant for Research on Measures for Infractable D. Guidelines for diagnosis and treatment of moyamoya disease (spontaneous occlusion of the circle of Willis). Neurol Med Chir (Tokyo) 2012; 52: 245-266.

11) Takanashi J. Moyamoya disease in children. Brain Dev 2011; 33: 229-234.

12) Lee WJ, Jung KH, Lee KJ, Kim JM, Lee ST, Chu K, Lee SK, Roh JK. Sonographic findings associated with stenosis progression and vascular complications in moyamoya disease. J Neurosurg 2015 Dec 11:1-9. [Epub ahead of print].

13) Matsushima T, Inoue T, Katsuta T, Natori Y, Suzuki S, Ikezaki K, Fukui M. An indirect revascularization method in the surgical treatment of moyamoya disease--various kinds of indirect procedures and a multiple combined indirect procedure. Neurol Med Chir (Tokyo) 1998; 38 Suppl: 297-302.

14) Kuroda S, Houkin K, Ishikawa T, Nakayama N, Ikeda J, Ishii N, Kamiyama H, Iwasaki Y. Determinants of intellectual outcome after surgical revascularization in pediatric moyamoya disease: a multivariate analysis. Childs Nerv Syst 2004; 20: 302-308.

15) Yamada S, Oki K, Itoh Y, Kuroda S, Houkin K, Tominaga T, Miyamoto S, Hashimoto N, Suzuki N; Research Committee on Spontaneous Occlusion of the Circle of Willis (Moyamoya Disease). Effects of Yamada S, Oki K, Itoh Y, Kuroda S, Houkin K, Tominaga T, Miyamoto S, Hashimoto N, Suzuki N; Research Committee on Spontaneous Occlusion of the Circle of Willis (Moyamoya Disease). the Registry Study of Research Committee on Moyamoya Disease in Japan. J Stroke Cerebrovasc Dis 2016; 25: 340-349.

16) Blauwblomme T, Lemaitre H, Naggara O, Calmon R, Kossorotof M, Bourgeois M, Mathon B, Puget S, Zerah M, Brunelle F, Sainte-Rose C, Boddaert N. Cerebral blood flow improvement after indirect revascularization for pediatric moyamoya disease: a statistical analysis of arterial spin-labeling MRI. AJNR Am J Neuroradiol 2015 Nov 19. [Epub ahead of print].

17) Liebeskind DS. Collateral circulation. Stroke 2003; 34: 2279-2284.

18) Bacigaluppi S, Dehdashti AR, Agid R, Krings T, Tymianski M, Mikulis DJ. The contribution of imaging in diagnosis, preoperative assessment, and follow-up of moyamoya disease: a review. Neurosurg Focus 2009; 26: E3.

19) Baltsavias G, Khan N, Valavanis A. The collateral circulation in pediatric moyamoya disease. Childs Nerv Syst 2015; 31: 389-398.

20) Sakamoto S, Ohba S, Shibukawa M, Kiura Y, Arita K, Kurisu K. CT perfusion imaging for childhood moyamoya disease before and after surgical revascularization. Acta Neurochir (Wien) 2006; 148: 77-81; discussion 81.

21) Kuroda S, Kashiwazaki D, Hirata K, Shiga T, Houkin K, Tamaki N. Effects of surgical revascularization on cerebral oxygen metabolism in patients with Moyamoya disease: an 15O-gas positron emission tomographic study. Stroke 2014; 45: 2717-2721.

22) Liu W, Zhu S, Wang X, Yue X, Zhou Z, Wang H, Xu G, Zhou C, Liu X. Evaluation of angiographic changes of the anterior choroidal and posterior communicating arteries for predicting cerebrovascular lesions in adult moyamoya disease. J Clin Neurosci 2011; 18: 374-378.

23) Zhu F, Karunanithi K, Qian Y, Mao Y, Xu B, Gu Y, Zhu W, Chen L, Wang Y, Pan H, Liao Y, Morgan M. Assessing surgical treatment outcome following superficial temporal artery to middle cerebral artery bypass based on computational haemodynamic analysis. J Biomech 2015; 48: 4053-4058.

24) Wu M, Huang Z, Zhang D, Wang L, Sun J, Wang S, Zhao Y, Zhao J. Color doppler hemodynamic study of the superficial temporal arteries in superficial temporal artery-middle cerebral artery (STA-MCA) bypass surgery for moyamoya disease. World Neurosurg 2011; 75: 258-263.

25) Ohno T, Kudo K, Zaharchuk G, Fujima N, Shirato H. Evaluation of diagnostic accuracy in CT perfusion analysis in moyamoya disease. Jpn J Radiol 2016; 34: 28-34.

26) Uchino A, Saito N, Takahashi M, Kurita H, Ishihara S. Cerebral arterial variations associated with moyamoya disease diagnosed by MR angiography. Neuroradiol J 2014; 27: 697-701.

27) Hwang K, Hwang G, Kwon OK. Endovascular embolization of a ruptured distal lenticulostriate artery aneurysm in patients with moyamoya disease. J Korean Neurosurg Soc 2014; 56: 492-495.

28) Funaki T, Takahashi JC, Yoshida K, Takagi Y, Fushimi Y, Kikuchi T, Mineharu Y, Okada T, Morimoto T, Miyamoto S. Periventricular anastomosis in moyamoya disease: detecting fragile collateral vessels with MR angiography. J Neurosurg 2015 Nov 27: 1-7. [Epub ahead of print].