PATTERNS OF REGIONAL CEREBRAL BLOOD FLOW IN PATIENTS WITH OCCLUSIVE OR STENOTIC LESIONS OF BOTH THE INTERNAL CAROTID AND VERTEBROBASILAR ARTERIES

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ABSTRACT

For the study of cerebral hemodynamics in patients with multiple occlusive or stenotic lesions of the internal carotid artery (ICA) and vertebral/basilar artery (VA/BA), six male patients (mean aged 62.5 years) were examined by regional cerebral blood flow (rCBF; Fg: intravenous bolus injection of Xe 133). The highest rCBF(73.3 \pm 16.1(SD)m Ω /100g/min) was observed in two patients with stenosis of the VA and bilateral ICAs and the lowest rCBF(47.1 \pm 5.9), in patients with severe stenosis of the BA including unilateral occlusion or stenosis of the VA and ICA. Intermediate rCBF (56.1 \pm 10.0) was found in patients with occlusion of the VA and occlusion or stenosis of the bilateral ICAs. Among them lesions without VA-occlusion and BA-stenosis were important in keeping rCBF high.

Key words: Regional cerebral blood flow, Fast flow in the gray matter, Multiple occlusive lesion, Internal carotid artery, Vertebral artery, Basilar artery

INTRODUCTION

Cerebral blood flow(CBF) is supplied by four main vessels including the internal carotid and vertebral arteries on both sides. There are familiar compensating mechanisms among these vessels showing that a sudden decrease of blood flow in the area of an occluded vessel induces the blood supply from the remaining functioning vessels.¹⁾²⁾³⁾⁴⁾⁵⁾ In such cases the circle of Willis connecting the four vessels takes on an important role.⁶⁾

Although few, some patients have simultaneous multiple lesions of the internal carotid arteries and the vertebral/basilar arteries.⁷⁾ The results of CBF studies on such patients, however, have not been consistent. Therefore, it is important to analyze the cerebral hemodynamics in such patients in relation to the clinical course.⁸⁾⁹⁾ Recently, Bogousslavsky et al. and Leblanc et al. reported on symptoms and long-term follow-up in patients with such multiple lesions in the internal carotid and vertebrobasilar systems.¹⁰⁾¹¹⁾ Although studies of this type are important for the evaluation of regional CBF(rCBF) as well as of the treatment of these patients, reports instructive in terms of treatment are few.¹²⁾¹³⁾

In this study we determined the flow patterns of rCBF in patients with multiple occlusive or stenotic lesions in the carotid and vertebrobasilar arteries. Factors contributing to flow patterns that had a close relation with clinical symptoms were also discussed.

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Received for Publication February 20, 1988

MATERIALS AND METHODS

Symptoms and treatment (Table 1)

Six male patients, mean aged 62.5 years (ranging from 48 to 74 years), were included in this study. Transient ischemic attack (TIA), suggesting the involvement of the vertebrobasilar system,¹⁴⁾¹⁵⁾ was seen in three of the six patients, while TIA caused by the insufficiency of the carotid system¹⁶⁾ was seen in another two patients. One patient, case 5, was free from the symptoms of TIA. Vertigo and cerebellar ataxia were the initial symptoms in cases 1 and 4. Deplopia was seen in cases 1 and 2. Visual loss and hemiparesis with convulsions were found in cases 3 and 6 respectively.

Complicated diseases were disclosed in four cases: systemic arterial hypertension, in case 1; pulmonary tuberculosis, in case 4; pituitary adenoma, in case 5; and Beurger's disease with diabetes mellitus and systemic arterial hypertension in case 6. The period of observation after the CBF examinations has ranged from 2 to 48 months(mean 17.8 months).

Three patients were surgically treated. Episodes of TIA decreased in each of these patients; however, their neurological impairments were little improved. The patient of case 3 was treated in a high pressure oxygen chamber, but his visual acuity did not improve. Transsphenoidal adenomectomy was performed in case 5. Drug administration alone was given in case 6, while surgical treatment plus additional drugs were administered to the other patients.

Angiography

A serial angiographic study of the four vessels was performed in all patients using the selective catheterization method or direct puncture of the vessels. The degree of stenosis of the vessels(%) was determined from the ratio of the diameter of the artery at maximum stenosis to that of the distal artery using the most demonstrable films.

Computed tomography

By X-ray computed tomography(CT) focal lesions related to the neurological signs were evaluated using routine axial slices.

Study of regional cerebral blood flow(rCBF)

At the time of rCBF study, hemiparesis was found in case 2 and case 6. Transient weakness in the legs and right hand were detected in case 1. Cerebellar ataxia was seen in case 4. Periods between the last episode of TIA and rCBF measurements in the six patients were distributed from 2 to 12 months(mean 5.6 months).

Regional CBF study was performed by an intravenous bolus-injection of radioisotope ¹³³Xe (5mCi/ml: Nihon Amasham Yakuhinkôgyô) using our original apparatus. For the computer calculations of rCBF, Obrist's method was used.¹⁷⁾ Topographic flow patterns were estimated in the frontal, temporal, central and parietal regions using eight symmetrically placed scintillation counters(1-inch diameter) on both sides of the scalp, for a total of 16 counters.

For the analysis of rCBF, fast flow in the gray matter (Fg: $m\ell/100g/min$) and mean flow values for 15 min (rCBF mean 15: $m\ell/100g/min$) were obtained. These results were compared with the data for the normal adult in our previous paper.¹⁸⁾ A judgment of an abnormal "decreasing" of rCBF values was determined when the value for the patient was lower than the predicted value (the mean value of the normal adult minus 2 standard deviations(2SD) of the mean) in corresponding regions. The following evaluations are based on the data for Fg. The values of rCBF at a mean of 15 min are also presented.

tCBF ON MULTIPLE LESIONS IN ICA AND BA-VA

Case No.	Age(years) & Sex	Signs & Symptoms		Period(months) between attack		Follow-up (months)	Complicated diseases
		At latest attack	At rCBF measurement	& rCBF measurement	state (→)	(anotabet
1	67 M	Vertigo, diplopia, cerebellar ataxia	Transient hypesthesia in both legs & weakness of r-hand	2	STA-MCA Anast. → no attack	6	Hypertension
2	48 M	Transient diplopia	l-Hemiparesis	12	SAT-MCA Anast. → no attack	9	None
3	63 M	l-Decreased visual acuity & l-optic atrophy	Continued in same degree	6	$OHP \rightarrow$ unchanged	6	None
4	69 M	Vertigo, cerebellar ataxia	Cerebellar ataxia	3	r-CEA → no attack	48	Pulmonary tuberculosis
5	74 M	None	None	-	Pituitary adenoma → Transsphenoic adenomectom		Pituitary adenoma
6	54 M	Convulsions, r-Hemiparesis	r-Hemiparesis	5	Drug admini- stration \rightarrow unchanged	36	Buerger's disease, diabetes mellitus & hypertension

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Abbreviations: M, male; l, left; r, right; rCBF, regional cerebral blood flow; STA, superficial temporal artery; MCA, middle cerebral artery; Anast, Anastomosis; CEA, carotid endoarterectomy; OHP, oxygen High Pressure

RESULTS

Angiographic findings (Table 2)

Two patients showed an occlusion or 70% stenosis of the basilar artery with 80% and 90% stenosis of the intracranial portion of the left vertebral artery, respectively. In addition, a unilateral carotid artery was occluded at the bifurcation (case 2) or stenosed by 90% at the cavernous portion (case 1). On the configuration of the circle of Willis, the left posterior communicating artery in case 1 and the bilateral posterior communicating arteries in case 2, were not visualized.

Two patients showed a unilateral occlusion of the left vertebral artery at the intracranial portion (case 3) and at the level of the right sixth cervical vertebra (case 4). Furthermore, in both of these cases bilateral lesions in the carotid arteries were detected. In case 3 the internal carotid arteries were occluded at their origins on the right side and showed 90% stenosis on the other side. In case 4, both 70% stenosis of the right internal carotid artery and 40% stenosis on the other side were shown at their origins. The right posterior communicating artery was narrowed, and collateral flow via muscle branches of the right vertebral artery were shown to be well developed.

Case No.	Vertebral & Basilar Arteries	Internal Carotid Arteries	Other findings	CT findings	
1	Occlusion of BA 90%-Stenosis of 1-VA at intracranial portion	90%-Stenosis of 1-IC at cavernous portion	1-P com A not visualized A ₁ of 1-IC: narrowed	Normal	
2	70%-Stenosis of BA 80%-Stenosis of 1-VA at intracranial portion	Occlusion of r-IC at bifurcation	bil-P com A not visualized	LDA in pons	
3	Occlusion of l-VA at intracranial portion	Occlusion of l-IC 90%-Stenosis of r-IC at bifurcation	External carotid artery: narrowed	Normal	
4	Occlusion of r-VA 40%-Stenosis of l-IC at C6 70%-Stenosis r-IC at bifurcation		P com A: narrowed Collateral artery from muscle branches	LDA in cerebellar hemisphere	
5	80%-Stenosis of r-VA at extracranial portion	80%-Stenosis of 1-IC 90%-Stenosis of r-IC at bifurcation	r-P com A: narrowed	Pituitary adenoma: enhanced mass in sella	
6	80%-Stenosis of r-VA 60%-Stenosis of bil at extracranial ICs at bifurcation portion		r-P com A: narrowed Collateral arteries from r-Ophthalmic Artery	Normal	

Table 2. Angiographic and CT findings in six patients

Abbreviations: VA, Vertebral Artery; BA, Basilar Artery; IC, Internal Carotid Artery; I, left; r, right; bil, bilateral; Al, Al portion of Anterior Cerebral Artery; P com A, Posterior communicating Artery; LDA, Low Density Area; C6, sixth cervical vertebra.

In cases 5 and 6, 80% stenosis of the extracranial portion of the unilateral vertebral artery was shown. In case 5, 90% and 50% stenoses of the internal carotid arteries were also shown on both sides, respectively. The posterior communicating artery seemed to be hypoplastic and the right internal carotid artery was well communicated with the ophthalmic artery on the same side. In case 6, the bilateral internal carotid arteries were stenosed by 60%. The posterior communicating arteries were well developed.

CT findings (Table 2)

A small low density area in the pons was found in case 2. An infarction of the left cerebellar hemisphere was detected in case 4. In case 5, an abnormally enhanced shadow in the sella was diagnosed as pituitary adenoma. Three of the six cases, cases 1, 3 and 6, showed normal CT findings.

Study of regional CBF (Table 3)

From the amount of decrease in the flow as indicated by Fg values, rCBF was classified into three types: type A, in which rCBF was most remarkably decreased, included cases 1 and 2; type B, in which rCBF was moderately lowered, included cases 3 and 4; and type C, which showed the highest values of rCBF among the three groups, included the remaining two cases, 5 and 6.

The two patients of type A showed a hemispheric flow of Fg distributed from 44.5 to 49.4 $m\ell/100g/min$ with a mean value of 47.1 ± 5.9(SD) $m\ell/100g/min(n=32)$. Mean CFB for 15 min (rCBF mean 15) ranged between 24.1 and 26.7 $m\ell/100g/min$. According to topographic values

in all areas. Fg remained below the normal values.

In type B, the values of Fg were distributed between 54.1 and 59.3 m $\ell/100g/min$ with a mean value of 56.1 ± 10.0(SD)m $\ell/100g/min(n=32)$. These values were found to be intermediate between type A and type C. Topographic values were lower than normal values throughout the area except for the right parietal region in case 4. Mean CBF for 15 min ranged from 28.4 to 35.2 m $\ell/100g/min$, which was below the normal range.

Туре	Fg values (ml/100g/min)	Case	rCBF (Fg)
A	B. 47.1±5.9 (n=32) Rt. 47.3±6.0 (n=16) Lt. 46.9±6.1 (n=16)	1	R L
		2	R L
	B. 56.1±10.0 (n=32) Rt. 56.6±12.7 (n=16) Lt. 55.7±6.8 (n=16)	3	R R
В		4	R L
с	B. 73.7±16.1 (n=32) Rt. 75.2±18.7 (n=16) Lt. 72.2±13.6 (n=16)	5	R L
		6	R L

Table 3. Classification of rCBF(Fg) measurements and topographic findings. Closed circle shows decreased rCBF at each region. Open circle shows normal rCBF at each region.
Abbreviations: B, both cerebral hemisphere; Rt(R), right cerebral hemisphere;

Abbreviations: B, both cerebral hemisphere; Rt(R), right cerebral hemisphere; Lt(L), left cerebral hemisphere.

The patients of type C showed the highest rCBF values of the three groups. Fg values ranged from 70.4 to 75.3 ml/100g/min with a mean value of $73.3 \pm 16.1(\text{SD})\text{ml/100g/min(n=32)}$. However, the mean rCBF for 15 min still remained below normal values, being distributed in the range of 32.8 and 37.1 ml/100g/min. By topographic analysis of this type, less than one half of the area on both cerebral hemispheres was abnormally decreased.

Mean values of Fg between any two of these types were significantly different(p<0.001). In case 6 autoregulation of rCBF was examined by an intravenous infusion of Trimetaphan camsilate. Using this drug, systemic blood pressure fell from 180/100 mmHg to 120/80 mmHg, respectively, when mean Fg values of the hemisphere decreased from $74.5 \pm 8.4(\text{SD})\text{m}^2/100\text{g}/\text{min}(n=16)$ to $66.6 \pm 8.7(\text{SD})\text{m}^2/100\text{g}/\text{min}(n=16)$. The differences between the two values of rCBF were statistically significant(p<0.02). The remaining patients were not evaluated by similar studies. Blood gas analysis values of all the patients were distributed within the normal range.

DISCUSSION

Multiple occlusive or stenotic lesions of the four vessels sometimes cause a sudden decrease in brain blood flow resulting in severe neurological deficits. Patients with such a condition must therefore be carefully followed up. Because such multiple lesions are infrequent, the accumulation of a mass of follow-up data is needed for the establishment of appropriate treatment. In our department these patients amounted to 2% of our cerebrovascular accident patients during the past five years.

There have been reveral reports about the possibility of more frequent attacks in patients with a lesion in the intracranial portion of the vertebral artery than in those with a lesion in the extracranial portion of the artery, $7^{(13)}$ but three out of the six patients in our series did not support this. There may be two factors involved in the development of the ischemic attacks of the posterior fossa. One is hypoperfusion and the other is embolus formation from the occluded or stenotic arteries. In the present series, two patients were treated with superficial temporal artery-middle cerebral artery anastomosis (STA-MCA anastomosis) to increase the blood supply to the supratentorial space. Their attacks decreased after surgery. All three of the patients were also administered anticoagulative drugs. These results suggest some improvement of hypoperfusion.

Neurological symptoms of the patients with lesions in the basilar artery showed vertebrobasilar TIA.¹⁶) In these cases the blood supply to the basilar artery is thought to be supported by the internal carotid artery⁷) including collateral flow via the other routes.

However, it seems curious that the lowest flow state was observed not in patients with bilateral carotid lesions but in patients with a unilateral carotid lesion. Such a difference in the rCBF patterns was in fact detected between type A and the other types. Symon⁵⁾ reported that the ratio of the contribution of cerebral blood flow by the common carotid artery and the vertebral artery are 67:33(%); sixty-seven percent of rCBF is supported by the common carotid artery and the the basilar artery blood flow amounts to 25% of the blood flow of the brain. An increase in the blood flow occurs instantaneously with the sudden occlusion of one of the four vessels.⁸⁾ Most of these results were confirmed in the experiments with monkeys, but the result of CBF study in chronic states is not always supported by the experimental data. Factors contributing to such lowered rCBF as in type A cannot be clearly explained from these data. In fact, a frequent finding in patients with bilateral lesions of the internal carotid artery is that rCBF supported by

the vertebral arteries remained within the normal range.

Our estimated results showed the lowest values of rCBF in type A. From this it appeared that failures in supporting cerebral circulation depend upon stenosis of the basilar artery and a narrowed or hypoplastic posterior communicating artery as stated below. The situation of these vessels was slightly different between cases 1 and 2 of type A. Although the patient of case 1 had a unilateral well-developed posterior communicating artery, blood supply from the internal carotid artery to the posterior fossa did not effectively occur. This was suggested by an episode of vertebrobasilar TIA. An effective perfusion pressure (PP) for the posterior fossa may be slightly different from that for the supratentorial space because of the anatomically short length of the basilar artery with many perforators.

Koga and Austin⁷⁾ reported that "retrograde flow" via the posterior communicating artery improved rCBF and symptoms. However the following findings in our cases were a little different from their results. In type A blood flow from the carotid artery via a posterior communicating artery "retrograde flow" was insufficient to provide any improvement in decreasing attacks of vertebrobasilar TIA, because of the decreasing rCBF at the supratentorial space. The redistribution of blood flow was generally not completed through collateral circulation even in the chronic period. The reason why a redistribution of cerebral blood flow throughout the intracranial space has been prolonged in this type seems, in part, to be due to the hypoplasia of the Al portion (case 1) on the left internal carotid artery as well as of the bilateral posterior communicating arteries (case 2). These constitutional features also seem to influence the impairment of the central neural regulation of blood flow in patients with lesions in the basilar artery, whose neurological state was vertebrobasilar TIA. In contrast, it is interesting that the two patients with the lowest value of CBF showed lesions of the basilar artery. From this point, central neural regulation originating in the brainstem seems to have some relation to the redistribution of CBF. In such cases the procedure to increase the perfusion pressure by externalinternal carotid(EC-IC) by-pass surgery was encouraged from the findings in their postoperative course (Table 1).

In contradiction to these results, four of the six patients with bilateral occlusion or stenosis of the internal carotid artery showed relatively higher values of rCBF than the previous type. Two of the patients with a unilateral occlusion of the vertebral artery (type B) showed lower values of rCBF than two patients with stenosis of the unilateral vertebral artery (type C).

Many previous reports have determined that the volume of the basilar artery flow is only 37% of that delivered by both vertebral arteries.¹⁾³⁾⁴⁾ From these results, even if the perfusion pressure is small in a stenosed vertebral artery, the stenosis of the arteries not the occlusion seems to contribute to the flow through the basilar artery. It is indeed needed to promote blood flow to the brain through the basilar artery by pressure from the bilateral vertebral arteries. A vertebral artery which joins the basilar artery, even if there were stenosis of one of the vertebral arteries on either side, may be able to effectively transmit intravascular pressure through a part of the stenosis into the basilar artery flow. Except for the case with occlusive lesions of the subclavian artery proxymal to the origin of the vertebral artery, the pressure through a part of the stenosis of the vertebral artery seems to be effective as a driving force toward the basilar artery. This fact is supported by Castaingne.²⁰⁾

Although patients showed various angiographic differences with degrees of development of the posterior communicating artery, patients with stenotic lesions seemed to have better circulatory conditions than those with occlusions of the vertebral artery. A higher level of rCBF in patients with bilateral lesions of the internal carotid arteries was shown in type C. Such circulation supplied by stenosed vertebral arteries in the posterior fossa may bring about better conditions in the supratentorial blood flow than that supplied by occluded vessels. This caused a characteristic difference in rCBF patterns between type B and C. However, one patient with type C showed carotid TIA, while the other of the same type remained asymptomatic. It is unclear from our results why the latter is asymptomatic.

Whenever the basilar artery without lesions and a well-developed posterior communicating artery are preserved, blood supply from the basilar artery to the suppratentorial space seems to occur easily. Schrott²¹⁾ reported on the neurological state of vertebrobasilar symptoms, stating that the stroke occurs when CBF is directed to the supratentorial space through the brainstem resulting in relative ischemia of the posterior circulation. In one of our three such patients, case 4, blood flow from the basilar artery to the internal carotid artery produced vertebrobasilar TIA. In this case an infarction of the cerebellar hemisphere was also found. In some of our cases with bilateral lesions in the carotid artery, the development of the posterior communicating artery was poor. In such patients supratentorial blood flow is supposed to be supplied mainly via the posterior communicating artery associated with other collateral flow. However, in our patients the symptoms were different from vertebrobasilar TIA: of our four cases, symptoms agreeing with Schrott's reports were found in only one case. We think that such neurological states, because of the relative ischemia in the posterior circulation, seem to be limited in number.

Koga and Austin⁷⁾ stated that two patients with verterbrobasilar lesions showed lowered rCBF values in spite of a well-developed posterior communicating artery, like our type C. They postulated that cerebral autoregulation is easily impaired in patients with vertebrobasilar disease. This is, as stated earlier, another function that causes neurological states and a decreasing rCBF in patients with multiple lesions. Autoregulation seems to include the central neural regulation of CBF; therefore, all of our patients were suspected of having an impaired autoregulatory mechanism. Autoregulation was impaired in case 6 (type C), and we were able to examine the function. In our opinion such examinations that may cause an acute drop in blood pressure should not be recommended, because they may induce permanent neurological deficits in a patient with multiple lesions.

Briefly, contributing factors analyzed by cerebral hemodynamics in patients with multiple lesions may be important for the treatment of these patients.

ACKNOWLEDGEMENT

The authors would like to thank Dr. Takagi of Minamiseikyö Hospital (Nagoya) and Dr. Noda of Nishio Municipal Hospital (Nishio, Aichi) for presenting clinical materials and treatment.

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