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Time Course of Vasospasm—Its Clinical Significance

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Summary

Serial angiograms, CT scans and clinical courses of 40 patients with vasospasm are analyzed. Angiographic vasospasm has a tendency to develop on arteries adjacent to ruptured aneurysms. The configuration of spasm changes from smooth narrowing into irregular narrowing. The former is observed after the 5th day and the latter after the 9th day of the rupture. Resolution of spasm follows irregular narrowing.

Angiographic vasospasm, neurological symptoms, and low density areas on CT scan develops at intervals of 2 to 3 days.

Optimal timing for direct aneurysmal surgery is discussed in relation to the time course of vasospasm.

Key words: Vasospasm, smooth narrowing, irregular narrowing, low density, cerebral infarction

Introduction

It is well known that vasospasm is one of the most significant prognostic factors for patients with ruptured intracranial aneurysm in the acute stage. There have been many clinical and experimental studies concerning vasospasm, but its exact etiology and pathogenesis are still unknown.

The purpose of this report is to analyze angiographic vasospasm, trace its time course, and to discuss the time relationship in the development of spasm, cerebral infarction, and neurological symptoms in clinical cases. We shall also try to determine the optimal timing for aneurysmal surgery from the viewpoint of the time course of vasospasm.

Materials and Methods

During the past 5 years, 169 patients with ruptured aneurysm 3 weeks after the onset of hemorrhage were admitted to Mihara Memorial Hospital. Eighty-four patients had angiographic evidence of vasospasm.

Of these, 10 conservatively treated cases and 30 operated cases with preoperative vasospasm were selected for analysis to seek the natural course of vasospasm. Special attention was paid to distribution, type, progress and resolution of the spasm. Findings were compared with postoperative cases.

In addition, computed tomography (CT) was repeated in 13 patients with spasm. Time relationship between the development of vasospasm, neurological symptoms, and appearance of low density area on CT scan was examined in these cases.

Angiographic vasospasm was diagnosed when the arterial stenosis was dilated in a subsequent angiogram or when the artery showed marked narrowing compared with vessels of the contralateral side. Patients with stenosis of a mild degree were excluded.

Angiographic vasospasm was classified into two types, smooth and irregular, according to its configuration. The latter type involved the so-called nodular or segmental narrowing (Fig. 1). The aneurysms were located on the anterior communicating artery (ACA) in 17 patients, on the carotid siphon in 9, on the middle cerebral artery (MCA) in 11, and at other sites in 3.

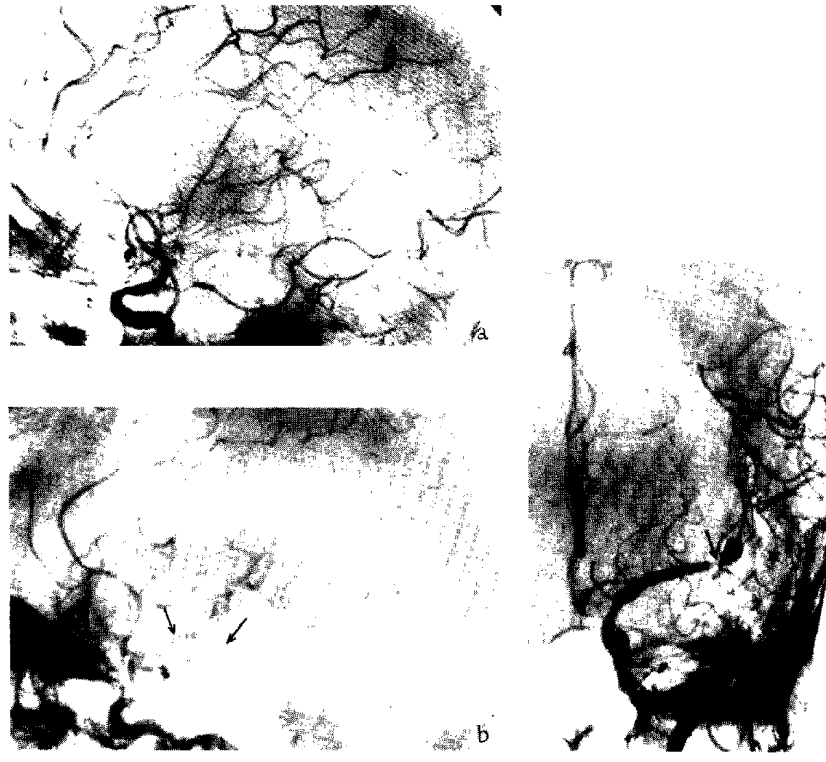

Fig. 1a: Angiogram of a patient with the smooth narrowing type.
1b: Angiogram of a patient with the irregular narrowing type. Insular arteries show nodular contour (arrows).
1c: Angiogram of a patient with the irregular narrowing type. Left Ml shows segmental stenosis (arrow).

Results

Distribution of vasospasm

Table 1 shows the relationship between the distribution of vasospasm and the ruptured aneurysm site. Films which show maximal spasm among repeated angiograms were selected for analysis. Three cases of aneurysm of the vertebrobasilar system or distal anterior cerebral artery were excluded.

Aneurysm of the anterior communicating artery (17 cases)

Vasospasm on the anterior cerebral artery (ACA) was seen most frequently. Spasm was found in the proximal ACA in 14 (81%) and in the distal ACA in 14 (88%) out of 17 and 16 cases, respectively (the distal ACA was not visible on angiogram in one case). Spasm of the middle cerebral artery (MCA) occurred less frequently. Eleven (65%) showed spasm on the insular portion and 12 (71%) cases on the horizontal portion of MCA.

Aneurysm of the middle cerebral artery (11 cases)

Vasospasm was most common on MCA. All 11 cases showed spasm on the insular portion and 7 (64%) also had spasm on the horizontal portion of the MCA. Spasm of the ACA was less common in these cases with 36% in the proximal and 9% in the distal ACA.

Aneurysm of the internal carotid artery (9 cases)

Spasm was often found not only on the MCA, but also on the ACA in these cases. It was observed on the MCA in the insular portion in 9

Table 1 Site of aneurysm and distribution of angiographic vasospasm

Arterial Segment	Site of Aneurysm	Anterior Communicating Artery	Middle Cerebral Artery	Internal Carotid Artery
ACA	Distal	14/16 (88)	2/10 (20)	5/8 (63)
	Proximal	14/17 (82)	4/9 (44)	5/8 (63)
MCA	Insular	11/17 (65)	11/11 (100)	9/9 (100)
	Horizontal	12/17 (71)	10/11 (91)	6/9 (67)
ICA		3/17 (6)	3/11 (27)	3/9 (33)

Spasm (+)/Total (%)
ACA: Anterior Cerebral Artery
MCA: Middle Cerebral Artery
ICA: Internal Carotid Artery

(100%), in the horizontal portion in 6 (67%), in the proximal ACA in 5 (63%), and in the distal ACA in 5 (63%).

Time course of angiographic vasospasm

Figure 2 shows the time course of vasospasm with regards to the location of the aneurysm. Segments examined were insular arteries in the cases of middle cerebral and internal carotid aneurysms, and distal ACA in the cases of anterior communicating aneurysm. No spasm was observed before the 6th day after rupture.

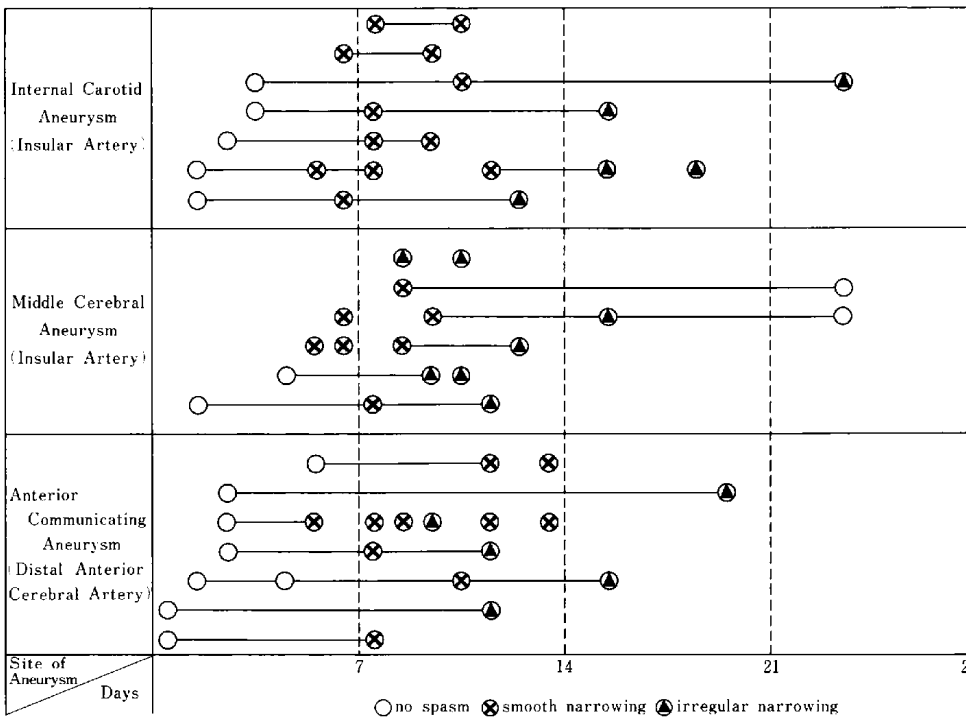

Fig. 2 Time course of angiographic vasospasm.

Spasm observed between the 6th and the 8th days were the smooth narrowing type in all cases. Irregular narrowing was seen exclusively on angiograms obtained after the 9th day. Furthermore, irregular narrowing was found to often follow smooth narrowing on repeated angiograms. Resolution of the spasm was also observed on arteries which had shown irregular narrowing.

The following cases showed typical courses of spasm on serial angiograms.

Case 1. A 45-year-old female admitted on the day of subarachnoid hemorrhage attack. She was drowsy and complained of severe headache. Bilateral carotid angiography performed on the 3rd day disclosed an aneurysm of the ACA. Spasm was not found (Fig. 3a). CT scan showed a high density area in the interhemispheric fissure, suprasellar cistern, and left medial Sylvial fissure. Seven days after admission, the patient was stuporous with progressive right hemiparesis. Repeated angiogram showed marked smooth narrowing on the ACA, the horizontal and proximal insular portions of the MCA (Fig. 3b). On the 12th day, repeated angiography showed irregular narrowing on the arteries in which smooth narrowing had been previously observed (Fig. 3c). The patient showed improvement of consciousness but right hemiparesis and aphasia remained.

Case 2. A 69-year-old female admitted 3 days after becoming suddenly unconscious for about one hour. She was drowsy and complained of headache, but weakness of the extremities was not recognized. Angiogram revealed an aneurysm of the left internal carotid artery without spasm (Fig. 4a). Two days after admission, she developed progressive right hemiparesis and impairment of consciousness. Smooth narrowing was observed on the left insular arteries on angiography performed when this neurological deterioration occurred (Fig. 4b). Smooth nar-

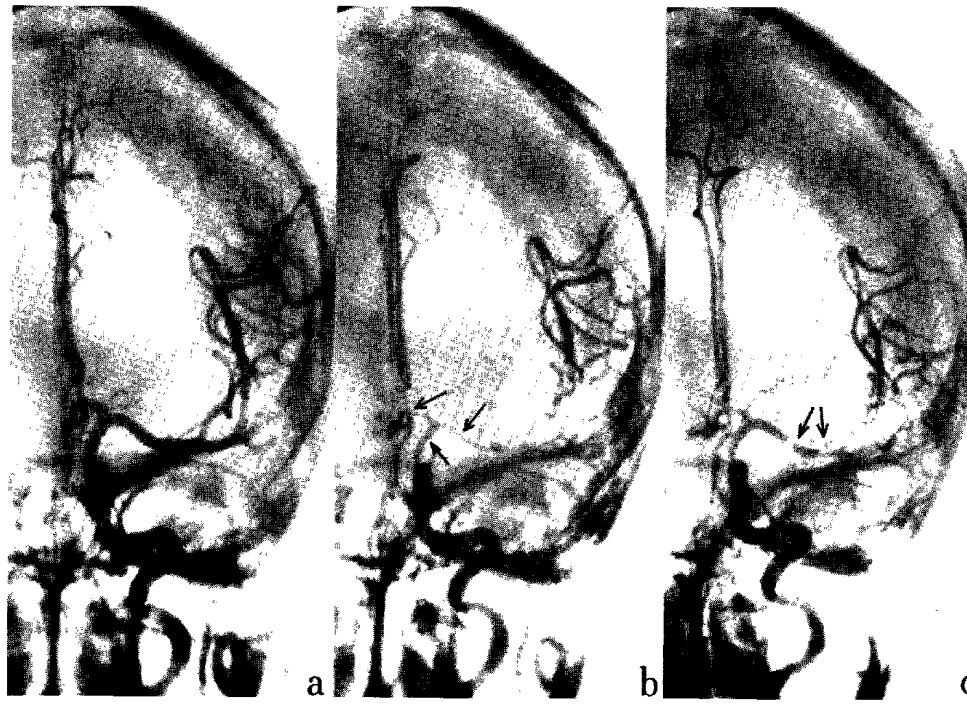

Fig. 3 Left carotid angiogram of Case 1 performed on the 3rd (a), the 8th (b) and the 12th days (c) after the attack. No spasm is seen on the first angiogram. The second shows smooth narrowing on the internal carotid, the middle cerebral and the anterior cerebral arteries (arrows). There is irregular narrowing on the middle cerebral artery (arrows) in the third angiogram.

rowing changed into irregular narrowing on repeated angiogram on the 16th day (Fig. 4c), and the patient did not regain consciousness.

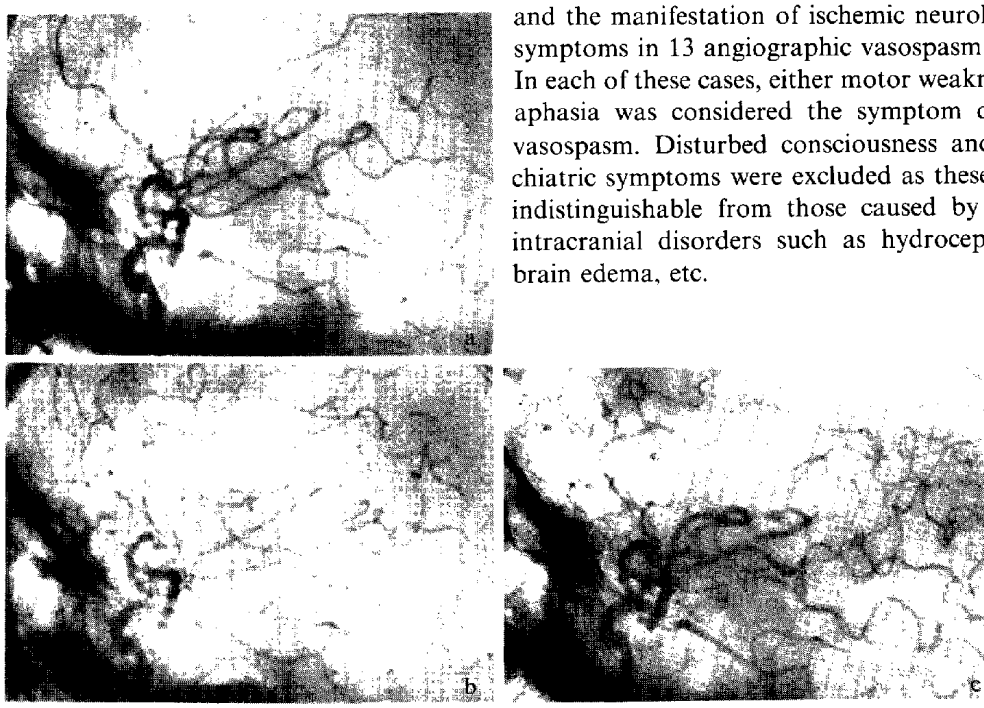

Fig. 4 Left carotid angiogram of Case 2 performed on the 4th (a), the 7th(b) and the 16th days (c), respectively. There is no spasm in the first angiogram, but the second shows marked smooth narrowing on the insular arteries. There is irregular narrowing on the third angiogram.

Neurological symptoms with the development of cerebral infarction

Figure 5 shows the time relationship between the appearance of a low density area on CT scan and the manifestation of ischemic neurological symptoms in 13 angiographic vasospasm cases. In each of these cases, either motor weakness or aphasia was considered the symptom due to vasospasm. Disturbed consciousness and psychiatric symptoms were excluded as these were indistinguishable from those caused by other intracranial disorders such as hydrocephalus, brain edema, etc.

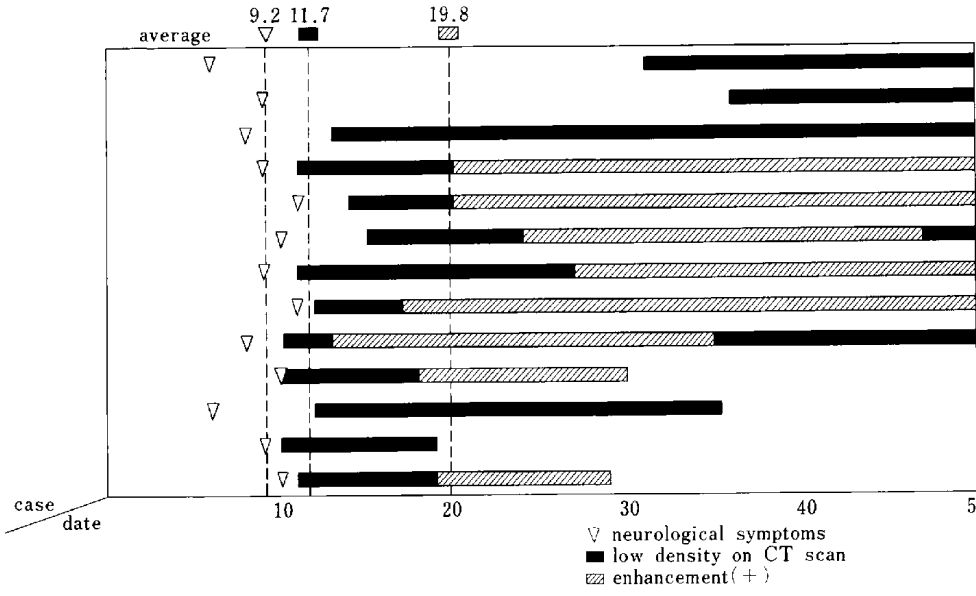

Fig. 5 Development of neurological symptoms and cerebral infarction.

Neurological symptoms caused by vasospasm developed between the 6th and the 11th day after the attack (average 9.2 ± 1.6). Low density areas on CT scan were found in all cases and they coincided with the sites of spastic arteries on the angiograms. They were observed between the 10th and 15 th day (average 11.7 ± 1.7) in 11 out of 13 cases and were enhanced by contrast medium after the 13th day after the attack (average 19.8 ± 4.3).

The two other cases showed low density areas on the 31st and the 36th days. Neurological deficits in these cases were mild and improved markedly.

Figure 6 shows findings of repeated CT scans of Case 1 (serial angiograms illustrated in Fig. 3). CT scan on the 10th day showed low density areas in the ACA and MCA. They became clearer on the 13th and enhanced markedly on the 20th day. After 3 months, sharp margined low density areas were observed in regions corresponding to the enhanced areas.

Discussion

There have been many reports concerning types and distributions of angiographic vasospasms,[2,6,7,8,13] but only a few discuss the natural courses of their development and resolution.[12] In this study, we have found that vasospasms following rupture of intracranial aneurysms have the following characteristic features.

Vasospasm has a marked tendency to develop on arteries adjacent to ruptured aneurysms. This close correlation between the site of aneurysm and the distribution of spasm can be explained by the fact that vasospasm tends to occur on arterial segments which are surrounded by subarachnoid hematomas. The location and extent of these hematomas are determined by the site of the aneurysm. Ruptured anterior communicating aneurysm is particularly likely to produce hematoma in the interhemispheric fissure or the suprasellar cistern. Middle cerebral aneurysm tends to form hematoma in the Sylvian fissure. From ruptured internal carotid aneurysm, blood may track into basal cisterns, the Sylvian fissure and the interhemispheric fissure.[11]

These concepts are supported by our previous report concerning the relationship between the

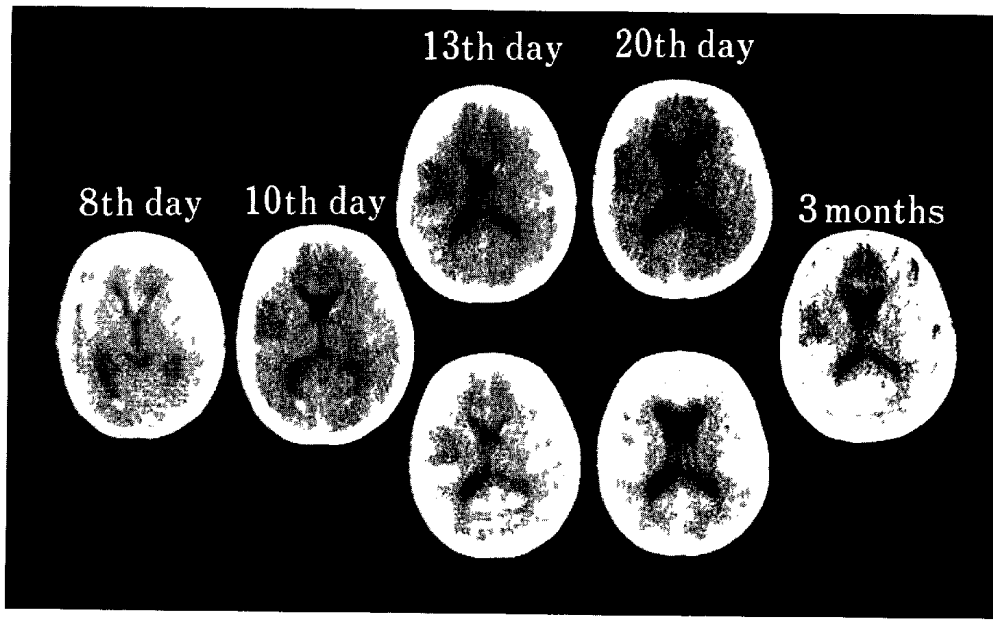

Fig. 6 Repeated CT scans of Case 1. Low density areas can be seen in the territory of ACA and MCA on the 10th day after the attack. They are more clearly defined on the 13th day and slightly enhanced (lower). Marked enhancement is seen on the 20th day (lower). After 3 months, sharp margined low density areas are observed corresponding to the enhanced areas.

location of high density area on CT scan and the distribution of vasospasm.[10]

Data presented in this paper reveal that the intensity and configuration of vasospasm changes from time to time. Smooth narrowing changes into irregular narrowing on angiograms obtained after the 9th day of the attack and resolution of the spasm occurs after irregular narrowing. The time course of vasospasm may be explained from pathological findings of the arterial wall.[3,4,5]

According to our histological study, contraction of the medial smooth muscle cells and the resultant medial necrosis are the main causative factors of angiographic vasospasm and its resolution. In patients with smooth narrowing appearing on angiograms, the media of the spastic arteries was thickened and the internal elastic lamina was markedly corrugated. In cases that had shown resolution of the spasm angiographically, the affected arteries were markedly dilated due to necrosis of the medial smooth muscle cells.

These histological facts and angiographic findings suggest that smooth narrowing is "true spasm," which indicates arterial narrowing caused by contraction of the media. Irregular narrowing is not "true spasm;" but a transitional form of arterial dilatation due to medial necrosis and it is considered to be a sign that the artery has no potentiality of constriction and will resolute or dilate by itself.

The extent of ischemic lesions caused by vasospasm can be exactly recognized by CT scan. Figure 7 is the schema of time relationship between the development of vasospasm, neurological symptoms, and cerebral infarction.

Vasospasm develops after the 5th day of the attack. Neurological symptoms appear one or 2 days later and cerebral infarction develops shortly after the onset of neurological deterioration. This schema suggests that treatment of vasospasm should be carried out in accordance with the time course of vasospasm.

During the first 5 days after subarachnoid hemorrhage, the aim of treatment is to prevent or minimize future development of vasospasm. It may be helpful to remove the subarachnoid hematoma as much as possible by direct operation.[9]

When smooth narrowing is observed on the angiogram, surgical intervention should be delayed because this procedure may worsen or prolong the vasospasm.[1]

Once irregular narrowing is observed, it is a sign of resolution of vasospasm and immediate surgery is indicated. Surgical procedure will not influence the intensity of vasospasm during this period.

When patients with vasospasm show neurological deterioration and low density areas are recognized on CT scan, treatment of cerebral edema and infarction should first be carried out. We have usually used Grycerol in such cases and have obtained good results in some.

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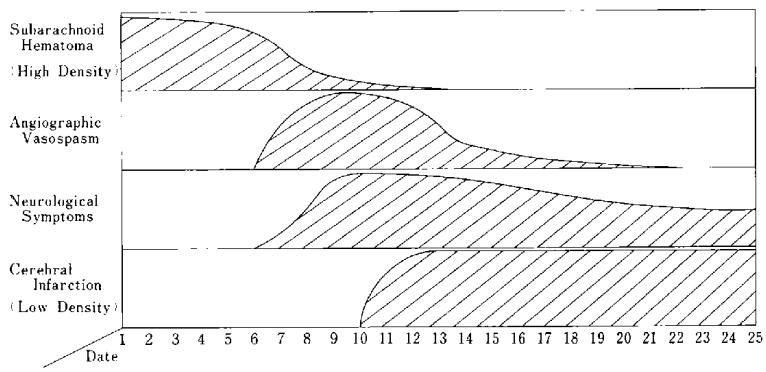

Fig. 7 Time relationship with development of angiographic vasospasm, neurological symptoms and cerebral infarction.

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