

“Misery Collaterals” as Poor Angiographic Findings - Definition, Classification, and Practical Application -

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Objective : Unique intercal carotid artery angiographic findings have been found especially in very poor grade aneurysmal subarachnoid hemorrhage(SAH) patients before and during the endovascular coiling. The author investigates their patterns and classifies them into four subtypes.

Methods : Among Hunt& Hess grade IV, V SAH patients, the author could gather eight patients who showed abnormal intracranial circulation in cerebral catheter-based angiography.

Results : The author introduces new term 'misery collaterals' first and has classified them into four types with the case illustrations. Type 1 is the worst condition defined as almost no intracranial circulation. Type 2 is the condition of little intracranial circulation with contrast filling just only at vessels of brain base, type 3 is of no or little cortical circulation with contrast filling at bilateral large vessels of brain base through circle of Willis channel and type 4 is of visible bilateral cortical circulation but delayed intracranial circulation time. The prognosis of these eight patients showed misery collaterals were disappointed.

Conclusion : These finding can be used as the supportive information in deciding a management plan in poor grade SAH patients.

KEY WORDS : Misery collaterals · Angiographic findings · Poor grade SAH · Aneurysm coiling.

Introduction

Despite the risk of complications, conventional catheter-based angiography still has been considered as the gold standard of diagnosing the intracranial vasculopathy including aneurysms because of its characteristics of showing real time hemodynamics, helping to understand parent artery flow change during procedure as the aneurysm has been obliterated, early detection of untoward flow pattern during and just after the isolation of aneurysm and the accuracy^{5,10,11}. With the increasing application of coiling even on the poor grade ruptured aneurysm patients, very unique angiographic findings could be found in the diagnostic angiography before coiling and also during the procedure. The author would like to name and classify them into four types with case illustrations and suggest treatment priority options for the better outcome.

Materials and Methods

Among grade IV, V Subarachnoid hemorrhage(SAH) patients, the author could gather eight patients showed

abnormal intracranial circulation in cerebral catheter-based angiography. These hemodynamic abnormalities were named 'misery collaterals' and could be classified into four subtypes according to the compromise of cerebral perfusion and the collateral pathway through circle of Willis.

Results

The prognosis of these eight patients with misery collaterals were disappointed. Type 1 is the worst condition defined as almost no intracranial circulation. Type 2 is the

Table 1. Four types of misery collaterals

Type 1	no intracranial circulation
Type 2	little intracranial circulation with contrast filling just only at vessels of brain base(ipsilateral M1, A1, and proximal posterior cerebral artery)
Type 3	no or little cortical circulation with contrast filling at bilateral large vessels of brain base through circle of Willis channel, with or without flow reversal to contralateral internal carotid artery and basilar artery
Type 4	visible bilateral cortical circulation but delayed intracranial circulation time, with or without flow reversal to contralateral internal carotid artery and basilar artery

Contrast regurgitation to ipsilateral ECA is shown in all types of misery collaterals

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condition of little intracranial circulation with contrast filling just only at vessels of brain base, type 3 is of no or little cortical circulation with contrast filling at bilateral large vessels of brain base through circle of Willis channel and type 4 is of visible bilateral cortical circulation but delayed intracranial circulation time. All these types have contrast regurgitation to ipsilateral external carotid artery(ECA) by increased intracranial pressure (Table 1).

Case 1 (Type 1 : No intracranial circulation)

68 year old woman presented with a comatose mentality and sluggish light reflex of pupils bilaterally, and brain CT at emergency room showed SAH and severe brain edema. With the impression of ruptured intracranial aneurysm, the patient was moved to the angiography suite directly together with usual intracranial pressure(ICP) control medically, and right internal carotid artery(ICA) and left vertebral artery angiography were performed. There was no intracranial circulation at all (Fig. 1). The patient was moved to operating theater right after the angiography and extraventricular drainage(EVD) operation with single burr hole at the right Kocher's point was done. The initial ventricular pressure was 40cmH₂O and ICP could be lowered and maintained rapidly to 20cmH₂O by height control of EVD set. Then the patient was moved to the angiographic suite again, and the angiography after EVD was performed.

On post-EVD left vertebral angiography, a faint shadow of aneurysm was found at right vertebral artery just proximal to the vertebrobasilar junction. The right vertebral artery angiography was performed and showed aneurysm clearly. Aneu-

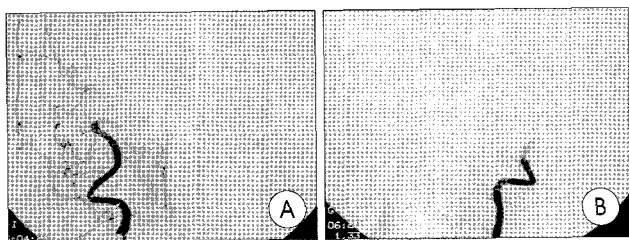


Fig. 1. No intracranial circulation at all on right internal carotid artery (A) & left vertebral angiography (B). On right ICA angiography, external carotid artery branches are shown by contrast regurgitation from intracranially obliterated flow of ICA further.

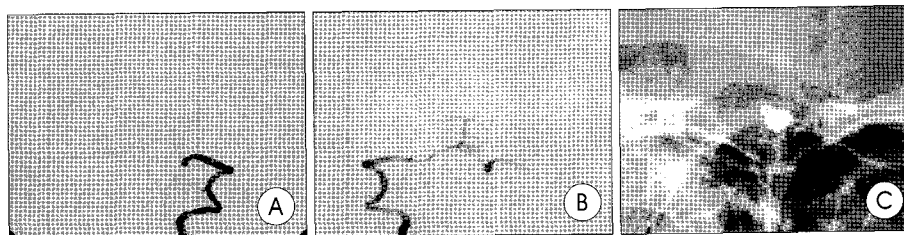


Fig. 2. Post-external ventricular drainage vertebral angiography. A : Left vertebral angiography showing faint aneurysmal dilatation of right vertebral artery. B : and C : Right vertebral angiography showing aneurysm clearly with visible posterior intracranial circulation, and coiling.

rysm coiling was done quickly without any difficulty (Fig. 2). The patient has saved her life but been in vegetative state.

Case 2 (Type 2 : Little intracranial circulation)

A 42-year old male was transferred to emergency room with SAH complaining of comatose mentality. On diagnostic angiography, there was little intracranial circulation just only at the base of brain, so only ipsilateral A1, M1 and proximal posterior cerebral artery(PCA) could be seen with the regurged ECA flow (Fig. 3). On brain CT there was diffuse brain swelling without space occupying lesion and ventricular dilatation, the ICP

control was performed medically only by mannitol dripping. Two days after the SAH onset, the patient was died without follow up angiography for the detection of any vascular lesion including intracranial aneurysm.

Case 3 (Type 3 : Little or no cortical circulation except larger arteries filling at brain base)

This 56 year old woman presented with a ruptured right posterior communicating artery aneurysm for which diagnostic cerebral angiography followed by aneurysm coiling was performed. On diagnostic angiography, no or little cortical circulation except larger arteries at skull base, with flow reversal to contra lateral ICA through circle of Willis channel and basilar artery through posterior communicating artery channel (Fig. 4).

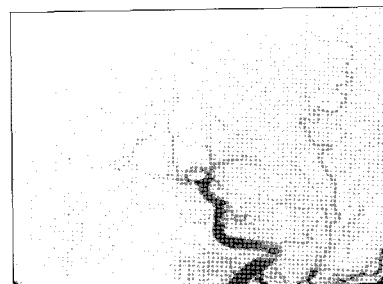


Fig. 3. Left internal carotid artery angiography showing little intracranial circulation just only in the base of brain (ipsilateral M1, A1 and proximal posterior cerebral artery) with contrast regurgitation to ipsilateral external carotid artery branches.

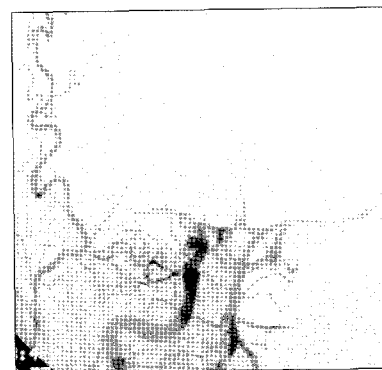


Fig. 4. Right internal carotid artery angiography showing posterior communicating artery aneurysm and no or little cortical circulation except larger arteries at skull base, with flow reversal to contra lateral ICA through circle of Willis channel and basilar artery through posterior communicating artery channel. The external carotid artery branches are also visualized by contrast regurgitation due to increased intracranial pressure.

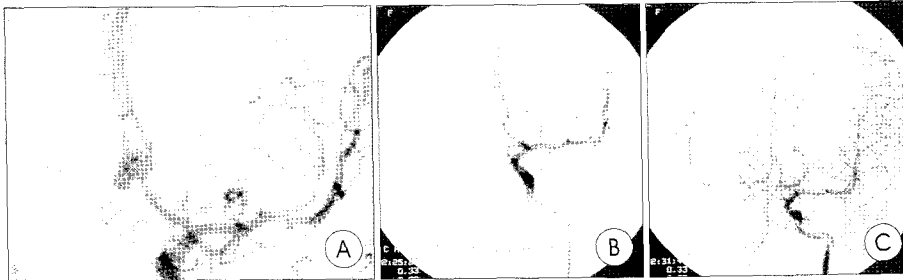


Fig. 5. A : Left internal carotid artery angiography showing anterior communicating artery aneurysm. B : Post-coiling left ICA angiography showing complete obliteration of aneurysm sac with normal intracranial hemodynamics. C : After several minutes, abnormal intracranial collaterals by flow reversal through circle of Willis are visible with posterior communicating artery.

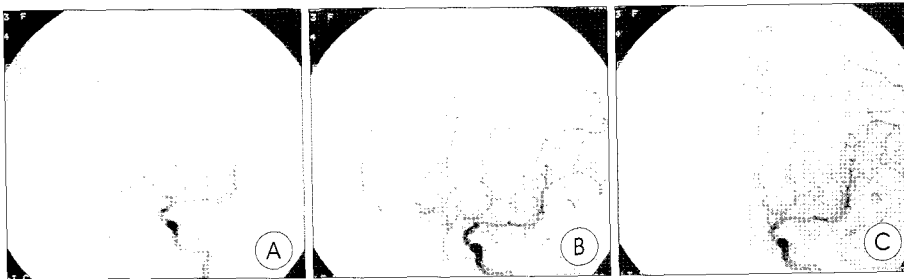


Fig. 6. A : On serial angiography, left distal anterior cerebral artery begins to be obliterated. B : Post-fibrinolysis angiography after 350,000 IU Urokinase intra-arterial infusion shows persisting abnormal intracranial collaterals. C : recanalization of left distal anterior cerebral artery.

Case 4 (Type 4 : Visible bilateral cortical circulation but delayed circulation time definitely)

A 42 year old woman presented with ruptured anterior communicating artery aneurysm with Hunt & Hess grade IV. The endovascular coiling was performed immediately after diagnostic angiography and it was considered as successful coiling (Fig. 5) and expected good outcome. But several minutes after, control angiography showed bilateral cortical delayed circulation with whole intracranial basal arteries visualization through circle of Willis and posterior communicating artery channels (Fig. 5C). And also the left distal anterior cerebral artery (ACA) flow was obliterated by regarding dislodged clot with brain edema. With continuing the ICP control medically the superselective intraarterial fibrinolysis using Urokinase 350,000 U was done and the left distal ACA flow has been recovered on post-fibrinolysis angiography, but still the misery collaterals was shown despite the medical ICP control (Fig. 6). On CT brain scan checked just after the procedure, severe brain edema was found with midline shifting to right side. The urgent decompressive craniectomy was recommended but the relatives refused and the patient was died three days after the coiling.

Discussion

Catheter-based cerebral angiography has long remained the gold standard for the evaluation and diagnosis of

cerebrovascular lesions and has been useful for intraoperative decision making, initiation of endovascular therapy, and postprocedural monitoring. It also can quickly provide a radiographic diagnosis of brain death by absence of flow at the foramen magnum in the posterior circulation and at the petrosal portion of the carotid artery in the anterior circulation⁵⁾. Yoshimoto et al¹⁷⁾ reported arteriovenous transit time calculated from acute phase angiogram in SAH patients reflects increased ICP and is related to prognosis. And extremely delayed intracranial circulation time would indicate irreversible brain damage and would not support interventions such as surgical clipping or endovascular coiling¹⁷⁾. But as most neurosurgeons have experienced, the patients with poor grade SAH

recovered dramatically within a short time after admission or did not recover at all afterward. It means there would be dynamic intracranial circulation changes especially in the acute phase of SAH and the early interventions by coiling or clipping even in the poor grade patients has been accepted recently^{2,4,7,12,14-16)}. With the increasing numbers of the early applications of endovascular coiling for poor grade ruptured aneurysm patients, the author could find unique angiographic findings which could be used as a valuable tool for understanding real time intracranial circulation during the coiling procedure and sign of favorable prognosis.

The original meaning of cerebral collateral circulation refers to the subsidiary network of vascular channels that stabilize cerebral blood flow when principal conduits fail as playing a pivotal role in the pathophysiology of cerebral ischemia^{6,8,9,13)}. And they are commonly divided into primary or secondary collateral pathways. Primary collaterals include the arterial segments of the circle of Willis, whereas the ophthalmic artery and leptomeningeal vessels constitute secondary collaterals. Primary collaterals provide immediate diversion of cerebral blood flow through existing anastomoses. Among the general population, approximately 50% have a complete circle of Willis, where possible variations can include underdeveloped or completely absent blood vessels¹⁾. Interestingly in the cases of acute increasing of the ICP especially in SAH patients, the similar but completely different collaterals through circle of Willis vessels have been showed on the cerebral angiography

with regular speed and amount of contrast injection through ICA. When these findings were appeared before and/or during the aneurysm coiling procedures, the prognosis has been very poor. So the author would like to name these angiographic findings with poor intracranial circulation 'misery collaterals' and classify them into four types according to the cortical, intracranial circulation and the degree of collaterals through circle of Willis vessels (Table 1).

By this time the author has experienced eight cases of misery collaterals, one type 1, two type 2, two type 3 and three type 4, and in all cases the patient's prognoses have been from mild disability state to death. In case 1, the patient was referred to my institute immediately after the onset of hemorrhage, so the author decided to do aggressive ICP control with EVD first even though there was no intracranial circulation at all on the initial diagnostic angiography like the definition of brain death. When the patient was moved to the angiographic suite right after the EVD and was performed angiography again, the misery collaterals type 1 was changed into misery collaterals type 4, and the right vertebral artery aneurysm was detected. The aneurysm coiling could be done without any procedural difficulty. Though the patient has been in severe disability state, it would propose another treatment option in the very poor intracranial circulation case especially in the acute SAH patient. As in case 5, misery collaterals could be appeared during the coiling procedure. In this case the distal ACA flow was obliterated by suggesting dislodged clot with rapidly progressing severe brain edema of uncertain origin and it was recovered by intra-arterial fibrinolysis using Urokinase, but there were still persisting abnormal intracranial collaterals regardless of the distal ACA flow recanalization. The misery collaterals in this case were not thought to be caused by distal ACA flow obliteration. All these findings would be appeared by sudden and tremendous increase of ICP against the common contrast injection pressure and speed. Because of the limited number of cases the solid conclusion for their clinical significance cannot be induced. However if we could support the better hemodynamic condition by any medical or surgical treatment in such kind of conditions, the prognosis may be more favorable. And also if these types are changed in the opposite direction during the coiling procedure, the rapid switchover to the decompressive operation should be needed. So understanding of these misery collaterals staging would be helpful to manage the poor grade aneurysmal SAH patients of hemodynamic disturbance previously.

The author can not explain here the percentage of patients with poor grade SAH that recover dramatically within a short time spontaneously after admission. It is certain the misery collaterals would exist rarely even in Hunt & Hess grade IV,

V patients. Although the reversibility and the prognosis of the poor grade SAH patients were not simply explained by these angiographic findings, it can provide supportive information in deciding a management plan.

Conclusion

The author introduces the term 'misery collaterals' with classification by the degree of persistent intracranial circulation and collaterals through the intracranial basal arteries including circle of Willis.

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Commentary

The authors described the angiographic findings of delayed intracranial circulation in patients with poor grade subarachnoid hemorrhage(SAH). They concluded that such findings named 'misery collaterals' and classified into four types according to the severity.

As the authors describe, the cerebral collateral circulation is a subsidiary network of vascular channels that stabilize cerebral metabolism when main flows obstruct. Yoshimoto et al.⁴⁾ classified such delayed intracranial circulation as arteriovenous transit time calculated from acute-phase of routine angiograms which was reflected increased intracranial pressure and related to prognosis. In my opinion, 'misery collaterals' is not a suitable word to describe delayed intracranial circulation or delayed perfusion. And I think four types of classification is not a different pathophysiology. As Hunt-Hess classification or Fisher grade on SAH patients, it is better to change the term as 'grade'.

Till now, we neurosurgeon made a decision whether to treat

immediately or observe by clinical grade and enhanced CT/CT angiogram in patients with poor grade SAH. Neurosurgeons can expect poor outcome which has an extremely delayed circulation in a comatose patients with no contrast dye on the enhanced CT scan. Nowadays, we can imagine the exact status and prognosis by CT angiogram and perfusion CT scan with easy¹⁻³⁾.

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