Treatment for Intracranial Cerebral Artery Stenosis

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Summary

Symptomatic intracranial cerebral artery stenosis is largely resistant to drug treatment. Regardless of their locations, lesions may cause cerebral infarction with a frequency of 7-10% in a year, but the natural history of asymptomatic intracranial cerebral artery stenosis remains unclear. Revascularization is indicated for symptomatic lesions which show resistance to drug treatment, while bypass surgery is the accepted therapeutic indication for haemodynamic ischemia. Endovascular treatment is effective in haemodynamic ischemia, and is also expected to be effective against embolic symptoms.

Bypass surgery for anterior circulation cases is safe because of its low incidence of complications, whereas bypass surgery for posterior circulation cases is technically difficult and has a high associated complication rate. Hence, endovascular treatment is currently favored for posterior circulation cases, and has also been introduced for anterior circulation cases.

Endovascular treatment has become a widespread modality for intracranial cerebral artery stenosis, but there are many unsolved problems associated with complications, technology and devices. Therefore, in practice, endovascular treatment should be used only with a strict indication, and should be performed only after considerable thought and with appropriate informed consent.

Introduction

Intracranial cerebral artery stenosis can be treated with drugs, but bypass surgery has been performed for cases refractory to such drug treatment. Since the extracranial-intracranial (EC/IC) bypass study suggested a lack of efficacy of bypass surgery¹, such surgery has rarely been performed, but now evaluation of CBF has become generally available, the usefulness of bypass surgery for haemodynamic ischemia is being recognized. On the other hand, in the endovascular treatment area, access to an intracranial cerebral artery has become possible by improvement of the performance of various devices, such as percutaneous transluminal angioplasty (PTA) balloons. As a result, PTA for intracranial cerebral artery stenosis is now performed, and recent improvements in stenting have produced further rapid advances in endovascular treatment. In this report, we describe the natural history and endovascular treatment of intracranial cerebral artery stenosis.

Natural history of intracranial cerebral artery stenosis

There are few reports of the natural history of intracranial cerebral artery stenosis. In cases of symptomatic intracranial internal carotid artery stenosis, the incidence of a shift to cerebral infarction is reported to be 7.6% within a

year, even if drug treatment is performed², while in symptomatic middle cerebral artery stenosis, symptoms recur within a year with a probability of 9.05%³. Moreover, it is reported that cerebral infarction occurs within a year at rates of 7.8% and 10.7% for symptomatic vertebral artery stenosis and basilar artery stenosis, respectively⁴. Based upon these data, it is thought that most symptomatic intracranial cerebral artery stenoses are resistant to drug treatment, and that lesions at all sites cause cerebral infarction with a frequency of 7-10% within a year. No reports of asymptomatic intracranial cerebral artery stenosis are available, and so the natural history is not clear. Therefore, for symptomatic intracranial cerebral artery stenosis, bypass surgery or active revascularization by endovascular treatment may be required, in addition to drug treatment.

Indications of revascularization for intracranial cerebral artery stenosis

Anterior circulation

Lesions of the intracranial internal carotid and middle cerebral artery (M1 portion) are the main therapeutic targets in the anterior circulation. Algorithm for the management of intracranial cerebral artery stenosis is shown in figure 1. The existence of symptoms is the most important factor in treatment decisions. In addition, since assessment of the cerebral blood flow is possible in the anterior circulation, acetazolamide hyporeactivity, as a measure of degradation of the residual cerebral circulation function, is the most important index. For symptomatic cases in which there is deterioration of the residual cerebral circulation function, bypass surgery or endovascular treatment is performed in association with antiplatelet drug therapy. In cases where no deterioration of the residual cerebral circulation function occurs, even if the case is symptomatic, and when the symptoms are due to an embolism from a stenotic segment, priority is first given to treatment with antiplatelet drugs, with subsequent consideration of endovascular treatment if the drugs are ineffective. Although bypass surgery theoretically has no effect for symptoms caused by an embolism from a stenotic segment origin, endovascular treatment seems to be effective in such cases. In addition, for symptomatic cases of thrombotic perforating branch ischemia

in middle cerebral artery stenosis, there is no indication for either bypass surgery or endovascular treatment, and endovascular treatment is particularly unlikely to be performed because the risk of perforating branch occlusion by plaque shift is high. For asymptomatic cases, drug treatment is indicated in principle, but in cases of residual cerebral circulation function degradation, bypass surgery or endovascular treatment could be considered.

Posterior circulation

In the posterior circulation, stenoses of the intracranial vertebral artery and basilar artery become therapeutic targets. The presence of symptoms greatly influences decisions regarding the treatment plan, same as for stenoses in the anterior circulation. However, the situation differs from that for the anterior circulation in that the parameters used to determine the appropriate treatment are poorly established, except for the presence of symptoms, because evaluation of cerebral circulation function in the posterior circulation is quite difficult.

Therefore, drug treatment, mainly with antiplatelet drugs, is the first choice as a general rule, without concern regarding the asymptomatic or symptomatic nature of the condition. Bypass surgery or endovascular treatment is performed for cases which present with haemodynamic symptoms and are resistant to medical treatment. Since the degree of difficulty of bypass surgery for the posterior circulation is generally high, endovascular treatment has recently tended to be the preferred method. Furthermore, endovascular treatment is chosen when the symptoms are resistant to drug treatment and also caused by embolism derived from a stenotic segment, similarly to anterior circulation stenoses. Bypass surgery is not used in the case of thrombotic perforating branch ischemia of the stenotic segment, and endovascular treatment is not performed in such cases, due to the danger of perforating branch occlusion.

Revascularization methods of intracranial cerebral artery stenosis

Surgery

If patients show haemodynamic ischemia, an indication of bypass surgery is considered. Superficial temporal artery-middle cerebral arte-

Figure 1 Algorithm for the management of intracranial cerebral artery stenosis.

ry (STA-MCA) bypass is performed in anterior circulation cases, while in posterior circulation cases the operative method is determined by the lesion site, and superficial temporal artery-superior cerebellar artery (STA-SCA) bypass is often performed in such cases. Because the operative field is shallow, STA-MCA bypass is easy to perform. However, for bypass surgery for the posterior circulation, such as STA-SCA bypass, the limited operative field makes the procedure technically difficult.

The frequency of perioperative complications during STA-MCA bypass is low, and the mortality and morbidity rates are reported to be 0.6% and 2.5%, respectively¹. In contrast, in bypass surgery for posterior circulation cases, mortality and morbidity rates are reported to be 8.4% and 13.3%, respectively⁵.

The stenotic segment often shifts to occlusion after bypass surgery, and as a result an infarct of the perforating branch area develops. In the EC/IC bypass study, it was proved that a 14% change to middle cerebral artery occlusion occurred after bypass surgery for middle cerebral artery stenosis¹.

Endovascular treatment

Endovascular treatment is a good indication for haemodynamic ischemia. Furthermore, when lesions arise from an embolic source, eli-

mination of the symptoms by vessel dilatation is anticipated, and this point differentiates the procedure from bypass surgery. However, endovascular treatment is difficult or dangerous in subjects with the following types of lesions: strongly curved lesions, long lesions, lesions involving perforators or branches, and lesions extending to the bifurcation. In strongly curved lesions, access is difficult and vessel dissection is common after dilatation. Acute occlusion and restenosis frequently occur in long lesions. In lesions including perforators or branches, the risk of occlusion by plaque shift is high, and acute occlusion and vessel dissection easily occur following dilatation in lesions extending to the bifurcation.

Regarding of the use of PTA or stenting, PTA usually seems to be the first choice. We consider stenting when dilatation is insufficient with PTA, or when vessel dissection occurs. Since it is likely that the stent cannot pass the carotid siphon in cases of stenosis of the internal carotid artery or the middle cerebral artery distal to the carotid siphon, the use of PTA is necessary, although when the carotid siphon is open, delivery of the stent to the distal vessel is sometimes possible. There are many reports that have discussed treatment outcome following PTA or stenting. For example, in 25 cases of stenosis of the cavernous and petrous portion

of the internal carotid artery, in which either PTA or stenting were used, morbidity was reported to be 4.2%, mortality was 0%, and the technical success rate was 90%⁶. Furthermore, stenting in ten cases of symptomatic basilar artery stenosis had an associated morbidity of 30%, a mortality of 0%, and a technical success rate of 100%⁷.

Problems such as distal embolism, perforating branch occlusion, vessel dissection, vessel rupture, hyperperfusion syndrome, and restenosis have been reported with the above procedures. Unlike cervical carotid artery stenosis, no device is available for distal protection. However, the frequency of distal embolism is lower than might be expected, probably because the plaque volume is small; one report suggests that symptomatic distal embolism occurs in only 10% of cases of basilar artery stenting⁷. Perforator occlusion occurs due to plaque shift or embolic debris, and is often accompanied by vessel dissection. Since endovascular treatment is based on vessel expansion, which is accompanied by plaque extension, perforator occlusion cannot be avoided, and a recent report has suggested that this problem occurs in 20% of cases of

basilar artery stenting⁷. Hyperperfusion syndrome arises in the case of extensive blood flow degradation, and it often tends to be lethal when cerebral haemorrhage occurs. Hence, blood pressure control following PTA or stenting is extremely important to avoid hyperperfusion syndrome. The frequency of restenosis is unclear because of the lack of availability of long-term follow-up studies. However the vessel diameter of the intracranial cerebral artery is so small that the rate of restenosis is likely to be high. A drug-eluting stent has been developed recently to prevent restenosis; good results have been reported in the coronary vessel area⁸ and this type of stent might be applied to intracranial cerebral artery stenosis.

Conclusions

Endovascular treatment has become a useful modality for intracranial cerebral artery stenosis, but many problems associated with complications, technology and devices remained unsolved. Therefore endovascular treatment should be performed with a strict indication and appropriate informed consent.

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