Endovascular Embolization of Unruptured Vertebral Dissection Using Guglielmi Electrolytically Detachable Coils: Case Report

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We present a patient with unruptured vertebral dissection whose angiograph showed further enlargement of the aneurysm, after a transient reduction in the size of the fusiform dilatation. Tolerance of parent artery occlusion was first confirmed by a balloon occlusion test. Both proximal and distal portions of the vertebral artery, including the dissected site, were occluded using Guglielmi electrolytically detachable coils; there were no complications. No new symptoms occurred during a 9-month follow-up period. For cases presenting with angiographic aneurysmal enlargement, embolization of the lesion using electrolytically detachable coils may be effective.

KEY WORDS: UNRUPTURED DISSECTING ANEURYSM; VERTEBRAL ARTERY; EMBOLIZATION; GUGLIELMI ELECTROLYTICALLY DETACHABLE COIL

INTRODUCTION

Spontaneous dissections of the vertebral artery are recognized as a common cause of stroke.¹ There are two clinical features of this condition: haemorrhage and ischaemia. The prognosis of cases of ruptured vertebral dissection associated with haemorrhage is poor, because the subsequent further rupture rate is high.¹⁻³ By contrast, cases of unruptured vertebral dissection that present with ischaemic symptoms have been reported to have a favourable outcome, and 62% of the lesions were successfully cured angiographically.¹ Surgical intervention is, therefore, not required immediately after the diagnosis. Observation by means of serial radiological examinations is recommended. Some studies
have reported, however, that unruptured vertebral dissection with persistent aneurysmal dilatation is an embolic source or causes subarachnoid haemorrhage. For such cases, radical treatment may be needed. We describe here a case of aneurysmal enlargement detected by angiography 3 months after the onset of symptoms. The clinical course and treatment of the unruptured vertebral dissection is discussed.

CASE REPORT

A 50-year-old man with a 1-month history of sensory disturbance of the left lower extremity was first seen at a local hospital. Angiography revealed fusiform dilatation and retention of contrast medium in the aneurysm in the venous phase at the distal site of the posterior inferior cerebellar artery (Fig. 1A). These findings were consistent with a diagnosis of vertebral dissection. He was transferred to our hospital 53 days after the onset of symptoms. Neurological examination revealed hypaesthesia of the left lower extremity. The second angiograph showed that the size of the aneurysm had not changed (Fig. 1B). The third angiograph, 61 days after the onset of symptoms, showed reduction of aneurysmal size (Fig. 1C), but the patient's symptoms did not improve. The patient was discharged 2 days after the third angiograph without any medical treatment (including anticoagulation therapy). The fourth angiograph, 96 days after the onset of symptoms, showed enlargement of the aneurysm (Fig. 1D). During the follow-up period, a transient reduction in size was followed by further enlargement of the aneurysm. This change may have been due to partial thrombolysis of the thrombosed pseudolumen. We consider that this angiographic change may be associated with a risk of rupture or formation of an embolic source. Endovascular treatment of this vertebral dissection was, therefore, planned.

Endovascular treatment was carried out 106 days after the onset of symptoms, taking a bilateral transfemoral approach under local anaesthesia. Systemic anticoagulation was achieved by intravenous administration of heparin. A balloon occlusion test of the affected vertebral artery was initially performed. As a result, temporary balloon occlusion of the left vertebral artery at a site proximal to the posterior inferior cerebellar artery, for 20 min, did not cause any neurological symptoms.

A 6.0-F balloon catheter (Zeppelin balloon guide) was guided to the left vertebral artery via the left femoral artery. This balloon catheter was introduced to control the blood flow of the left vertebral artery when the aneurysm ruptured during the procedure. A 5.0-F catheter (Omni guide) was guided to the right vertebral artery via the right femoral artery. The microcatheters (Rapid Transit-18) were then introduced through both guiding catheters to distal and proximal sites of the dissection (Fig. 2A). Initially, a Guglielmi electrolytically detachable coil (GDC), 5 mm in diameter and 20 cm in length, was placed at the proximal portion of the dissection anterograde. The diameter of the coil was small, however, and the stability was not good, so the coil was withdrawn. Through another microcatheter, placed at the distal side of the dissection, the GDC was repositioned to the dissected site retrograde. A total of 16 GDCs was used for embolization. Postoperative left vertebral angiography revealed complete occlusion of the vertebral artery distal to the posterior inferior cerebellar artery (Fig. 2B). Right vertebral angiography showed contralateral retrograde filling of the left vertebral artery, distal to the dissected site. No complications occurred during or after the procedure. Postoperatively,
intravenous administration of low-molecular-weight dextran (500 ml/day) and algatroban was continued for 5 days. The postoperative course was uneventful and the patient was discharged with no new neurological deficits 7 days after embolization. Within 9 months after the procedure, no new symptoms occurred.

**DISCUSSION**

There are two types of symptoms due to intracranial vertebral dissection, those of ischaemia and those of subarachnoid haemorrhage. Recent studies have shown that cases presenting with ischaemic symptoms are generally benign, whereas patients
presenting with subarachnoid haemorrhage have a poor prognosis.\textsuperscript{1,2} The treatment of these entities should be discussed separately.

We report here the treatment of a patient with unruptured vertebral dissection associated with aneurysmal enlargement on serial angiography. In this patient, although the dissected portion decreased in size about 2 months after the onset of symptoms, it showed enlargement 1 month later. We considered it likely that partial thrombolysis of the thrombosed pseudolumen had occurred. Neither the natural history nor appropriate management of cases presenting with enlargement after transient reduction have previously been reported. Although the prognosis of unruptured vertebral dissection is good, some studies have indicated that persistent aneurysmal dilatation may become an embolic source\textsuperscript{4} or cause subarachnoid haemorrhage.\textsuperscript{5} We therefore recommend radical therapy for cases of angiographic aneurysmal enlargement.

The purpose of treatment for vertebral dissection is to isolate the dissected site from the blood circulation to prevent rupture. Direct surgery has been done in some cases of vertebral dissection, and has involved proximal clipping, trapping or wrapping. Proximal obliteration of the vertebral artery is the most widely accepted approach for cases that tolerate the balloon occlusion test. In the case described above, however, proximal obliteration did not prevent contralateral retrograde blood-flow to the dissected portion. Trapping was therefore the most reasonable treatment. For some patients, however, direct surgery is too invasive.

Endovascular embolization for saccular aneurysms using GDCs has recently been

\textbf{FIGURE 2}

\begin{figure}[h]
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\caption{(A) The microcatheters were introduced through a guiding catheter to the dissected site, both anterograde and retrograde. (B) Post-operative angiography reveals no filling of the aneurysm and complete occlusion of the vertebral artery distal to the posterior inferior cerebellar artery.}
\end{figure}
The GDCs can be detached from the pusher wire with electrocurrents so that, if the initial position of a coil at the lesion is not optimal, the coil can be withdrawn and repositioned. This can prevent coils from migrating or being positioned inappropriately. In dissections involving the vertebral artery distal to the posterior inferior cerebellar artery, occlusion of the vertebral artery must be kept as short as possible due to the presence of perforating arteries arising from the vertebral artery. The GDCs permit occlusion of short segments. We planned to obliterate the dissected site itself in addition to its proximal and distal vertebral artery. We therefore advanced two microcatheters to the dissected site; one was placed anterograde to and the other was placed retrograde from the contralateral vertebral artery. This technique was useful for embolizing the dissected site including the distal and proximal portions of the vertebral artery.

In conclusion, for cases of unruptured vertebral dissection associated with enlargement of fusiform dilatation, radical treatment should be considered. In patients who tolerate temporary balloon occlusion, endovascular coil embolization of the dissection may be an effective therapy. Further clinical investigations with a larger number of cases, and with follow-up studies, are needed.

**REFERENCES**


