CASE REPORT

Surgical recanalisation of thrombosed M2 trunk after coil embolisation of a ruptured middle cerebral artery aneurysm

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Thromboembolic events are known complications of endovascular coiling of intracerebral aneurysms. We report a case of a 50-year-old patient whose ruptured middle cerebral artery aneurysm was treated with endovascular coiling, which was complicated by occlusion of the inferior M2 trunk during the procedure. An emergency craniotomy was performed to enable coil retrieval, evacuation of the thrombus and clipping of the aneurysm. All the middle cerebral artery branches were recanalised and the patient recovered with no neurological deficits. Our experience suggests that an emergency salvage operation for thrombosis after endovascular coiling is a treatment option offering a good clinical outcome.

Case report

A 50-year-old woman with good past health and an unremarkable family history presented with sudden onset of headache and projectile vomiting. Upon admission, her blood pressure was 184/101 mm Hg. Her Glasgow Coma Scale (GCS) score was 15 with no focal neurological deficits. She attended the Accident and Emergency Department on the day that ictus developed and an urgent plain computed tomography (CT) of her brain showed a diffuse subarachnoid haemorrhage. A subsequent CT cerebral angiogram revealed a 4.3-mm inferior pointing aneurysm at the left middle cerebral artery (MCA) bifurcation (Fig a).

The patient was later transferred to the angiographic suite for embolisation using Guglielmi Detachable Coils (GDC; Boston Scientific, Natick [MA], US). The procedure was performed with the patient under general anaesthesia with endotracheal intubation. A F7 arterial sheath was placed in the right femoral artery. Diagnostic digital subtraction angiography with 3-dimensional reconstruction confirmed the aneurysmal anatomy as depicted in the CT angiogram (Figs b, c). A Tracker Excel-14 Micro-catheter with Synchro micro-guidewire (Boston Scientific, Natick [MA], US) was navigated and placed inside the aneurysmal sac. Two GDC coils (Matrix-2 360º 3 mm x 8 mm, Matrix-2 helical 2 mm x 6 mm) were deployed uneventfully and formed a stable basket inside the aneurysmal sac. A check angiogram discovered minimal extravasation of contrast after deployment of a third coil (Matrix-2 helical 2 mm x 4 mm) [Fig d]. A fourth coil (Matrix-2 helical 2 mm x 4 mm) was immediately deployed to completely occlude the aneurysm. There was no more contrast filling of the aneurysm afterwards but the inferior M2 trunk flow was compromised (Figs e, f). A bolus of 2000 units of heparin was given intravenously along with 4 mg intra-arterial abciximab to try to recanalise the inferior M2 trunk. There was some return of flow but contrast stasis was evident in the late arterial phase, signifying that flow was not adequate for sustaining the distal circulation. Multiple attempts were made to navigate the inferior M2 trunk in hopes of stenting the vessel open but all were unsuccessful. The risk of provoking rebleeding from the aneurysm precluded the use of further intra-arterial abciximab.

An emergency left pterional craniectomy was performed. Protamine was given prior to the skin incision in order to correct the effects of the heparin, and the patient was transfused with platelets and fresh frozen plasma to correct any platelet dysfunction and heparin anticoagulation. After creating a left fronto-temporal opening, the left sylvian fissure was split and the MCA bifurcation was exposed. A 4-mm saccular aneurysm was identified at the M1 bifurcation. Temporary clips were applied to the proximal M1 and distally to both M2 branches. The aneurysm sac was opened and all the GDC coils were retrieved. A small piece of soft thrombus was removed from the inferior M2 trunk. The aneurysm was finally clipped with two titanium aneurysm clips. The temporary trapping time was 9 minutes. An intra-operative micro-Doppler ultrasound confirmed that flow was present in both M2 trunks and a prophylactic decompressive craniectomy was performed to minimise the risk of a massive MCA infarct. The time from occlusion of the M2 trunk during coiling to recanalisation after coil removal was 4 hours.

Key words
Aneurysm, ruptured; Embolization, therapeutic; Equipment failure analysis; Thrombosis

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Postoperatively the patient was sedated and was put on a mechanical respirator. She gradually regained consciousness and was extubated 2 days after the operation. Her GCS level improved to E4V4M6. She had mild difficulties with speech expression and a plain CT of her brain showed a small area of hypodensity in the left temporal lobe. Reassessment digital subtraction angiography was performed on postoperative day 15. The inferior M2 trunk was patent and the aneurysm was completely obliterated (Fig g). She was later transferred to a rehabilitation institution and made an uneventful recovery. Her speech returned to normal during the following months. An autologous cranioplasty was performed 7 weeks after the initial haemorrhage (Fig h).

彈簧環栓塞術治療中腦動脈瘤破裂後的M2段閉塞再通

血管內彈簧環治療腦動脈瘤後出現血栓塞是一種已知的併發症。本文報告一名50歲有中腦動脈瘤破裂的病人，接受血管內彈簧環栓塞術時出現下幹M2段閉塞，隨即為病人進行顱骨切開術以取出彈簧環，清除血栓及夾閉動脈瘤。所有中腦動脈分枝閉塞再通，病人完全康復並沒有出現神經系統異常。本病例顯示為血管內彈簧環治療後出現血栓塞的病人進行緊急挽救手術有良好的臨床結果。

Discussion

Thromboembolisms are common complications of endovascular embolisation of aneurysms with reported incidences ranging from 2.5 to 11%.[1,2] Despite the high incidence of this complication, we based our choice of endovascular embolisation over clipping on the International Subarachnoid Aneurysm Trial.[3] This is a multicentre, randomised clinical trial that compared the clinical outcomes of ruptured aneurysms managed with either neurosurgical clipping or endovascular embolisation in patients considered suitable for both treatments. In this trial, endovascular embolisation offered a reduction of relative and absolute risk of dependency or death by 22.6% and 6.3% respectively, compared with clipping. In addition, there was no significant difference in the rebleeding rate between the two modalities.

Various mechanisms have been postulated as the cause of thromboembolism during the procedure. These include: migration of pre-existing thrombus within the aneurysm during the procedure; thrombosis of intraluminal coil through the aneurysmal neck to the parent vessel; migration of the coil into the parent trunk; thrombosis over the surface of the micro catheter and guide wire; slowing of blood flow due to vasospasm secondary to a pre-existing subarachnoid haemorrhage; presence of the micro-catheter and the mass effect of coils on the parent trunk. In order to prevent this complication, it is standard practice to give heparin for anticoagulation.

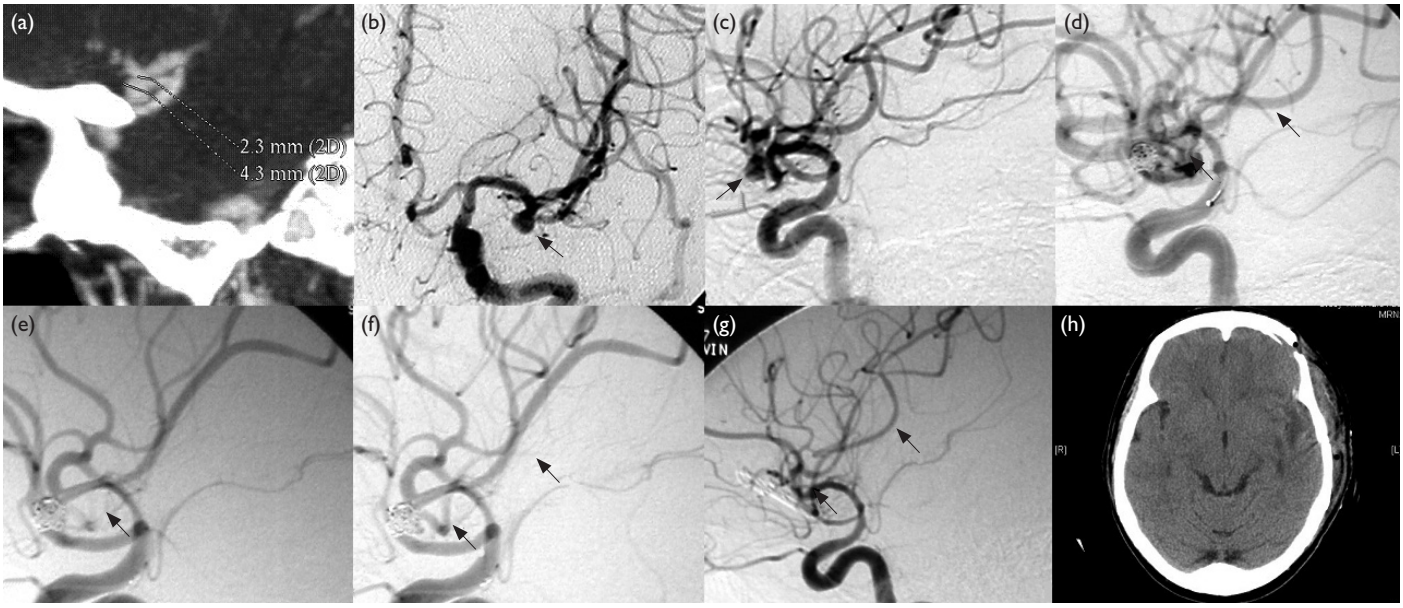

FIG. (a) Computed tomographic (CT) angiogram showing a 4.3-mm middle cerebral artery bifurcation aneurysm and a neck measuring 2.3 mm with a favourable dome-neck ratio for coil embolisation. (b, c) **Pre-embolisation subtraction angiography, anteroposterior and lateral views showing an** aneurysm (arrow) at the M1 bifurcation. (d) **Subtraction angiogram, lateral view, after deployment of three coils; the inferior M2 trunk (arrows) was** patent. (e, f) **Subtraction angiogram after deployment of the fourth coil; the inferior M2 trunk (arrow) was not shown in the early arterial period with** delayed filling in the late arterial phase. (g) **Reassessment cerebral subtraction angiogram after clipping of the aneurysm; no residual aneurysm was found** and the inferior M2 trunk (arrows) was recanalised. (h) **Plain CT of the brain after cranioplasty: postoperative changes in the left perisylvian area with** no major cerebral infarct

The aim is to achieve an activated clotting time (ACT) of two to six times the normal value.[4,5] One study has suggested that use of intravenous acetylsalicylic acid along with intra-operative heparin can decrease the risk of thromboembolism.[6] If thromboembolism does occur, a number of methods of resolving it have been suggested, including the use of a bolus of heparin to further increase ACT, thrombolysis with tissue plasminogen activator or urokinase,[7] glycoprotein IIb/IIIa inhibitor[8] or use of a mechanical retrieval device to remove thrombogenic GDC.[9] The major problem with the use of anticoagulation, antiplatelet or thrombolytic agents is haemorrhage,[7] particularly in patients who have had a recent subarachnoid haemorrhage. The use of thrombolytics may trigger rebleeding of the aneurysm, and studies have shown they achieve complete recanalisation in around 50% of cases only.[8,10] In this report, we describe a patient whose illness was complicated by a thrombosis on a M2 trunk after embolisation of a left MCA bifurcation aneurysm. It was possible to recanalise the MCA trunk by performing an immediate craniotomy and retrieval of the coil and then a thrombectomy and clipping of the aneurysm. The patient recovered with no permanent neurological sequelae.

Emergency surgical operations for complications associated with coil embolisation are uncommon. The usual reason for surgery is a need to retrieve the coil to relieve the mass effect on parent vessels several months after embolisation. Shin et al[11] reported emergency surgical recanalisation of the left A1 segment of the anterior cerebral artery by opening the aneurysm and removing the coil after occlusion by a tangled GDC used to manage a ruptured anterior communicating artery aneurysm. Deshmukh et al[12] reported surgical retrieval of a GDC that had migrated to the M3 segment of the MCA during embolisation of an ophthalmic artery aneurysm that was complicated by extravasation during the endovascular salvage attempt. Thornton et al[13] reported two patients in whom coil retrieval was necessary within 4 days of embolisation due to coil migration from aneurysms of the anterior communicating artery and the posterior communicating artery. Tirakotai et al[14] reported thrombosis of the posterior inferior cerebellar artery (PICA) after an attempt at embolisation of a PICA aneurysm. An emergency opening and revascularisation was performed and the patient recovered well. From the above case reports, it appears most patients recovered well with few or no neurological deficits.

From our previous experience, use of an intravascular stent to keep the coil from protruding into the inferior M2 trunk is effective for recanalising the vessel. In our patient, however, the guide wire could not navigate the inferior M2 trunk and stenting was not successful. Further manipulation with the wire may cause more coil protrusion. Use of a coil retrieval device was a possible alternative but this may have required negotiating through the clot and be associated with complications like perforation.[12] Prior extravasation of contrast during coil packing precluded any attempt to remove the coil. Time—particularly the duration of ischaemia—is also a major concern. The intra-arterial thrombolysis study[15] indicated that the golden period for successful management of intra-arterial thrombolysis in acute stroke is within 6 hours. If this timeline is to be followed, normal blood flow has to be restored no later than 6 hours after an abnormal angiogram. In our patient, the time interval between disturbed blood flow and restoration was 4 hours. General anaesthesia may have a cerebral protecting effect and thus provide extra time. Moreover, the patient suffered from reduced flow, instead of complete occlusion, of a M2 trunk that, theoretically, should permit a window of longer than 6 hours. The surgical techniques used to open the aneurysm and retrieve the coil were no different from standard clipping procedures. The aneurysm was trapped in the usual manner with control of the vessels proximal and distal to the aneurysm. The aneurysmal sac was opened at the fundus and all the coils were removed before clipping. The coil was removed gently to avoid stretching of the platinum coil. Retrieval of the newly placed coil harbouring minimal thrombosis was relatively straightforward compared with procedures performed to remove coils deployed for long periods which have built up dense adhesions.[14] At the same time, it was possible to achieve thrombus removal with complete recanalisation under direct vision.

In summary, we believe open craniotomy was essential to salvage our patient after a complicated embolisation of her left MCA aneurysm. If endovascular salvage, such as intra-arterial thrombolysis, bolus heparin, or stenting of the arterial trunk does not recanalise the vessel, immediate craniotomy and retrieval of the coils is a viable option. Expertise and resources should always be available during coil embolisation as the therapeutic time window for open recanalisation is limited. Good outcomes can be achieved with surgical management of the thromboembolic complications of aneurysm coiling. Previous case reports and our experience with this patient indicate that, contrary to current thinking, use of an emergency operation for revascularising a complicated coil embolisation is an effective salvage manoeuvre. So far, there have been no reported cases of surgical treatment for thrombotic complications after embolisation of MCA aneurysms.

Declaration

The authors do not report any conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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