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Rapid development of a *de novo* intracranial aneurysm following carotid occlusion

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Abstract

Carotid occlusion is a therapeutic intervention for specific intracranial vascular conditions, including giant, traumatic and intracavernous aneurysms. The long-term complications include de novo aneurysm formation at a distant site due to hemodynamic changes in the circle of Willis. The time frame for de novo aneurysm formation has been described in years with a mean of 9.6 years. There is no formalised radiological surveillance program following carotid occlusion. We describe the rapid development of a *de novo* posterior communicating artery aneurysm following contralateral carotid occlusion for a traumatic intracavernous aneurysm and suggest the need for a formalised radiological follow-up program following carotid occlusion.

Keywords: Intracranial aneurysm development; Carotid occlusion; Subarachnoid haemorrhage

1. Introduction

Despite progress in surgical and endovascular techniques, there remains a population of aneurysms that are unable to be treated without sacrifice of the parent feeding artery. Carotid occlusion with coils, glue or balloons is used in certain instances of intracavernous internal carotid artery (ICA) aneurysm or giant aneurysm with beneficial effect. The long-term complications of carotid occlusion include cerebral ischaemia, aneurysm regrowth and delayed haemorrhage from aneurysmal regrowth or formation of de novo cerebral aneurysms distant to the origin aneurysmal site.[1–5] We present a case of rapid development of a contralateral posterior communicating artery (PComA) aneurysm following ipsilateral coil occlusion of the proxi-

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mal ICA for a enlarging traumatic intracavernous ICA aneurysm.

2. Case report

A 19-year-old non-smoker presented following a high-velocity motor vehicle accident. He suffered severe multi-system injuries including a severe head injury with initial Glasgow Coma Scale score (GCS) of 8 and multi-level spinal fractures. His CT brain scan demonstrated diffuse traumatic subarachnoid haemorrhage in the basal cisterns, a left frontal extradural haematoma and fractures involving the sphenoid sinus and clivus (Fig. 1). An emergency craniotomy and evacuation of extradural haematoma was performed and he was transferred to the intensive care unit where inotropic supports were instigated to maintain a normal cerebral perfusion pressure. A 4-vessel cerebral angiogram was obtained the following day revealing an irregular left intracavernous aneurysm consistent with a traumatic false aneurysm measuring $3.5 \times 4.1 \times 4.2$ mm (Fig. 2). This was not thought to be the cause of the diffuse subarachnoid haemorrhage which was deemed traumatic. No other aneurysms were evident. Repeat selective left ICA angiogram was performed 3 days later demonstrating obvious enlargement of the intracavernous irregular aneurysm measuring $6.9 \times 5.6 \times 2.8$ mm (Fig. 2). There was no active extravasation. Coil occlusion of the left ICA was performed along the opening of the pseudoaneurysm and distally along the inferior limb of the carotid siphon. Complete cessation of flow in the left ICA and traumatic aneurysm was confirmed angiographically (Fig. 3). There was evidence of increased flow through the right ICA which was now perfusing the left-sided anterior circulation via the anterior communicating artery (AComA) and the middle cerebral artery (MCA) territory (Fig. 4). The patient remained sedated, intubated and ventilated throughout these events. Follow-up angiogram was performed 1 day post-occlusion which demonstrated complete occlusion of the proximal left ICA and traumatic aneurysm with filling of the intracranial ICA via the remaining intracranial vasculature. A 2.5×2.9 mm contralateral right PComA aneurysm was identified (Fig. 5). This was not present on the initial right-sided cerebral angiogram (Fig. 6), nor on the right ICA angiogram performed immediately following left ICA occlusion 1 day earlier. An emergent craniotomy and clipping of the PComA aneurysm was performed with post-operative angiogram showing complete clipping. At operation the aneurysm was noted to be thin-walled, red and regular in shape consistent with a de novo aneurysm. There was no evidence this was a false aneurysm.

The patient was discharged from intensive care to the ward and made good neurological gains prior to transfer to a rehabilitation facility. Follow-up angiogram performed 6 months post-injury demonstrated persistent complete occlusion of the proximal left ICA, traumatic aneurysm and right PComA aneurysm.

3. Discussion

Intracranial aneurysms have been estimated to affect 5% of the general population.[6–11] The usual presentation of these involves sudden rupture and subarachnoid haemorrhage. Despite recent advances in microsurgical operative technique, endovascular procedures and intensive neurosurgical care, the mortality and morbidity from subarachnoid haemorrhage remains high with a 40–50% mortality rate.[12,13] Outcomes following intervention for cerebral aneurysms are clearly improved in the elective non-ruptured setting. The optimal goal of aneurysm treatment as such is identification of high-risk patients, regular safe surveillance and early intervention in the unruptured state.

The aetiology of cerebral aneurysms remains contentious. Case reports support Gull's hypothesis of a genetic link with the development of cerebral aneurysms with hereditary diseases including Marfan and Ehlers-Danlos

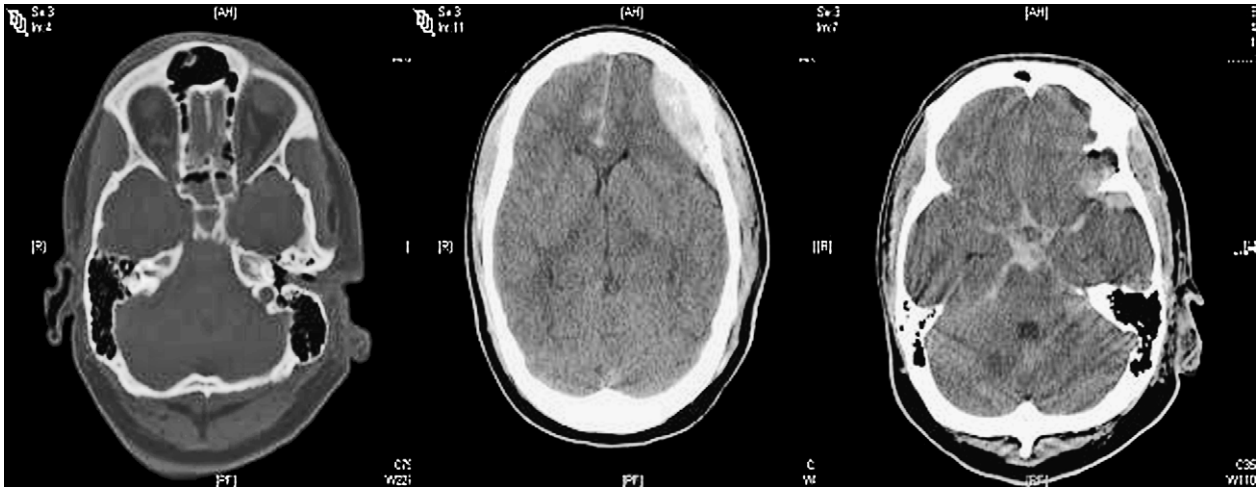

Fig. 1. Initial CT brain scan showing subarachnoid blood in the basal cisterns (right), extradural haematoma (middle) and a fracture of the sphenoid bone (left).