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Review

Cerebral vasospasm and delayed ischaemic deficit following elective aneurysm clipping

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ABSTRACT

Although common after subarachnoid haemorrhage, cerebral vasospasm (CVS) and delayed ischaemic neurological deficit (DIND) rarely occur following elective clipping of unruptured aneurysms. The onset of this complication is variable and its pathophysiology is poorly understood. We report two patients with CVS associated with DIND following unruptured aneurysmal clipping. The literature is reviewed and the potential mechanisms in the context of patient presentations are discussed. A woman aged 53 and a man aged 70 were treated with elective clipping of unruptured middle cerebral artery aneurysms, the older patient also having an anterior communicating artery aneurysm clipped. The operations were uncomplicated with no intra-operative bleeding, no retraction, no contusion, no middle cerebral artery (MCA) temporary clipping, and no intra-operative rupture. Routine post-operative CT scan and CT angiogram showed that in both patients the aneurysms were excluded from the circulation and there was no perioperative subarachnoid blood. Both patients had no neurological deficit post-operatively, but on day 2 developed DIND and vasospasm of the MCA. Both patients had angiographic improvement with intra-arterial verapamil treatment. In one patient, this was done promptly and the patient made a complete recovery, but in the other, the diagnosis was delayed for more than 24 hours and the patient had residual hemiparesis and dysphasia due to MCA territory infarction. CVS and DIND following treatment of unruptured aneurysms is a very rare event. However, clinicians should be vigilant as prompt diagnosis and management is required to minimise the risk of cerebral infarction and poor outcome.

1. Introduction

Cerebral vasospasm (CVS) is commonly seen following subarachnoid haemorrhage (SAH), occurring in 2/3 of patients, beginning around day 3–5 following haemorrhage and resolving around day 14 [1]. It is characterised by arterial narrowing seen on radiological imaging that is not due to atherosclerosis or catheter-induced spasm [2,3]. The incidence of CVS following SAH closely correlates with the amount of blood within the subarachnoid space [4,5]. Approximately one third of CVS patients after SAH develop delayed ischaemic neurological deficits (DIND) [2,6,7].

Previous studies have indicated that CVS following SAH is associated with the breakdown of clot in the subarachnoid space, [8–10] implicating a role for the vasospastic mediators released from lysed red blood cells [11–13]. The onset of DIND also occurs at approximately the time of maximal haemolysis [14]. Based on the widely-held assumption that CVS was the primary cause of DIND, the somewhat misleading term of "clinical vasospasm" was previously used interchangeably with DIND [15,3]. However, because the role of CVS as the sole or primary cause of DIND is currently debated [16,17] we will confine the use of the term CVS to demonstrable arterial narrowing, and DIND to a clinical deficit.

Only 20–40% of SAH vasospasm patients develop DIND [2,6,7]. The severity of CVS also does not always correlate with the development of neurological symptoms [15,3]. Although the pathophysiology behind this discrepancy is not well understood, endothelial dysfunction, loss of autoregulation and microvascular thrombosis have been proposed to play a major role [18]. In addition to CVS, other factors may play a causative role in the development of DIND including early brain injury and cortical spreading depolarisation [19,20].

Although common after SAH, CVS and DIND are extremely rare following elective treatment of unruptured aneurysms. We report two cases, one of a 53-year-old woman and another of a

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70-year-old man developing CVS with DIND on the second post-operative day following elective clipping of unruptured aneurysms. To our knowledge this is the first reported case of angiographically proven CVS and DIND in a male patient following successful clipping of an unruptured aneurysm, and both patients demonstrated onset of CVS and DIND earlier than has been previously reported.

2. Clinical presentation

2.1. Patient 1

A 53-year-old, right handed woman, presented for elective clipping of a 5 mm distal left M1 segment middle cerebral artery (MCA) aneurysm. The aneurysm had been discovered coincidentally on CT scan and MRI for unrelated neck and shoulder pain. There was no history or radiographic evidence to suggest prior bleeding from the aneurysm. The aneurysm was confirmed pre-operatively with digital subtraction angiography (DSA) (Fig. 1A).

The patient underwent a left pterional craniotomy and uneventful exposure of the aneurysm, which arose from the origin of a significant frontal branch of the M1, about 8 mm proximal to the main MCA bifurcation. Miniclip application across the aneurysm neck caused initial compromise of flow to this frontal branch, and the clip was repositioned three times until satisfactory flow could be determined. There was no evidence of any prior SAH and no intraoperative rupture of the aneurysm. No temporary clipping or retractors were used during the operation and no veins or arterial branches were injured or sacrificed. The patient awoke and was extubated immediately post-operative and had no speech, motor or any other neurological deficit, and was walking the day of surgery. Routine post-operative CT scan on the day of surgery showed no blood in the Sylvian fissure or basal cisterns. The patient remained perfectly well until 30 hours after the operation, when she rapidly developed dysphasia and fluctuant right hemiparesis predominantly affecting the upper limb.

The diagnosis of vasospasm was not initially considered, and a presumptive diagnosis at that time was thrombosis of the frontal branch, or possibility subclinical seizure. CT scan showed no intradural haemorrhage and CTA demonstrated that the aneurysm had been excluded from the circulation. EEG was negative for epileptiform activity. MRI was then performed which did not show evidence of acute infarction. The patient was treated with moderate induced hypertension and hypervolaemia in an attempt to optimise collateral circulation, but with no clinical improvement. A cerebral DSA performed on the third post-operative day (more than 24 hours after the onset of symptoms) demonstrated significant left MCA vasospasm (Fig. 1B). Intra-arterial verapamil was given, with good angiographic response (Fig. 1C). However, the patient continued to have significant dysphasia and hemiparesis, and repeat MRI on the 6th post-operative day showed evidence of a left MCA infarct (Fig. 2). The patient was discharged to a rehabilitation facility, and at a 3 month follow-up was independent with minimal dysphasia but persisting right upper limb weakness.

2.2. Patient 2

A 70-year-old right-handed man was admitted for an elective clipping of an anterior communicating artery (ACoA) aneurysm and also two small left MCA aneurysms. These aneurysms had been discovered coincidentally on a previous CT scan following minor trauma. There was no clinical or radiographic evidence of previous SAH. His past medical history was significant for type 2 diabetes mellitus, smoking and essential hypertension. In preoperative discussion conservative management options were emphasised, but the patient requested treatment of the aneurysms.

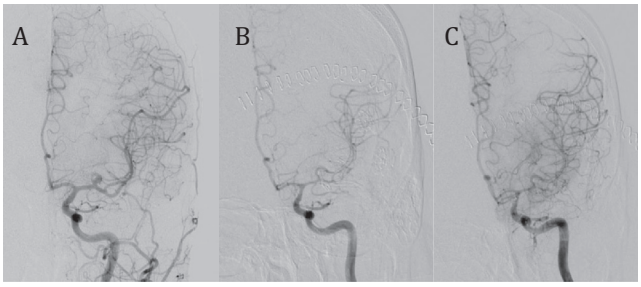

Fig. 1. Digital subtraction angiography of patient 1. (A) Preoperative left internal carotid artery (ICA) injection, anteroposterior projection. (B) Postoperative day 3 left ICA injection, AP projection, showing cerebral vasospasm. (C) Postoperative day 3 left ICA injection, AP projection after intra-arterial 10 mg verapamil infusion with resolution of vasospasm.

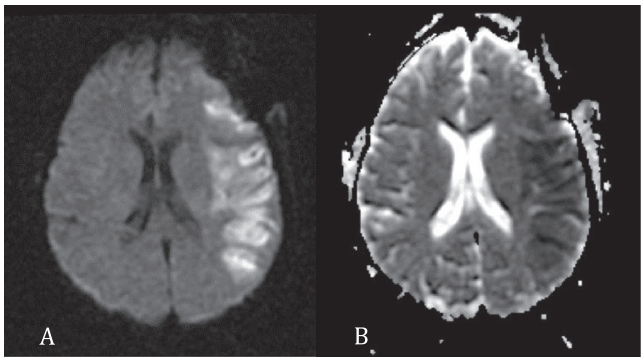

Fig. 2. MRI patient 1, diffusion-weighted imaging protocol on day 6 demonstrating established infarct in the left MCA territory. (A) Restricted diffusion and (B) low apparent diffusion coefficient (ADC) attenuation in the left MCA territory.

Old xanthochromic staining of the arachnoid immediately adjacent to one of the MCA aneurysm was noted intra-operatively, but there was no evidence of any recent SAH. The operative procedure was uncomplicated with no intraoperative aneurysm rupture. The MCA aneurysms were exposed and clipped with no temporary clipping, no retraction, no contusion and no venous or arterial injury. The ACoA aneurysm was also exposed with no retraction, but about 1 cm of gyrus rectus was resected to clearly identify both A2 vessels prior to clipping. Temporary clipping of both A1 segments was used for 4 minutes for final dissection and clipping of the ACoA aneurysm. Intraoperative DSA demonstrated exclusion of all aneurysms from the circulation (Fig. 3A, B) and normal vessel calibre. The patient awoke and was extubated immediately after the operation, and was neurologically intact and walking within a few hours of surgery. Routine post-operative CT scan on the day of surgery showed no blood in the Sylvian fissure or basal cisterns.

On post-operative day 2, the patient deteriorated such that he opened his eyes to pain only, was severely dysphasic, and had a right hemiparesis. Urgent CT scan revealed no intracranial bleed and DSA demonstrated severe arterial vasospasm involving the left M2 and M3 branches (Fig. 3C). During the angiogram, 20 mg of Verapamil was injected into the left M1 in divided doses, with good angiographic response (Fig. 3D) and good clinical response. The patient was then treated with a 21 day course of nimodipine and also moderate induced hypertension and hypervolaemia for 3 days. A day 6 post-operative MRI did not demonstrate an acute infarct and MRA at that time demonstrated normal calibre of left M1 and M2 branches. The patient was discharged to home on day 11 with no residual neurological deficit.