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Brain abscess after endosaccular embolisation of a cerebral aneurysm

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ABSTRACT

Endovascular embolization has become an important treatment option for cerebral aneurysms, along with surgical clipping. But few literatures mentioned infectious complications after coiling of aneurysms. We present a patient with a brain abscess that developed after endosaccular embolization of a left middle cerebral artery aneurysm. The brain abscess was located adjacent to the aneurysm and discovered more than 2 months after embolization. We discuss the clinical implications of this rare complication and review the literature for infections related to the coils used for embolization of aneurysms.

1. Introduction

Endovascular coil embolisation of intracranial aneurysms has increasingly been used during recent years. Many studies have reported complications after the procedure, but seldom mention infectious events as a consequence. We present a patient with a brain abscess that developed after endosaccular embolisation of a left middle cerebral artery aneurysm. The brain abscess was located adjacent to the aneurysm and was discovered more than 2 months after embolisation.

2. Case report

A 54-year-old man presented with a subarachnoid haemorrhage (SAH) (Fig. 1). He had a Hunt-Hess grade of IV at the time of admission. Cerebral angiography showed an aneurysm ($7 \times 6 \times 5$ mm^3 in size) at the bifurcation of the left middle cerebral artery (Fig. 2), and the patient underwent an endovascular coil embolisation.

The femoral area was sterilised with iodine tincture and 75% alcohol. Six detachable coils were deployed into the aneurysm. The first coil was a 7 mm × 15 cm Nexus Tetris coil (ev3 Endovascular, Plymouth, MN, USA), the next two coils were 5 mm × 12 cm Microplex coils (MicroVention, Tustin, CA, USA), and the last three coils were 4 mm × 12 cm Microplex coils (MicroVention). Postembolisation angiograms showed that a small residual neck of the aneurysm remained after the coiling procedure (Fig. 2B, C). The patient showed complete clinical recovery. He did not suffer influenza or any other infectious disease during his hospital stay. He began complaining of severe headaches 11 weeks after he was discharged from the hospital. A head CT scan was performed and showed a $5 \times 4.5 \times 5.5$ cm^3 brain abscess adjacent to the treated aneurysm (Fig. 3A–C). After the abscess was drained externally, the patient complained of severe headaches and had sudden neurological deterioration. A CT scan showed that the aneurysm had ruptured (Fig. 3D), and the patient died 6 hours after the drainage procedure.

3. Discussion

Of the more than 700 patients with aneurysms treated in our medical centre from January 2005 to December 2010, 66.7% were treated using embolisation. Of these, this patient was the only one in which a brain abscess developed after the coiling procedure. Neurointerventional literature regarding the development of a postembolisation brain abscess is very scarce. Falagas et al. reviewed seven cases of infection following intracranial endovascular procedures and concluded that the most common infectious complication directly related to endovascular coil embolisation was abscess formation. Five cases in this review were aneurysm cases.[1]

Regarding the cause of such a brain abscess, one possibility is that the aneurysm might be a mycotic aneurysm, although mycotic aneurysms only account for 2.5–6% of all intracavernous aneurysms.[2] Cloud et al. reported that mycotic aneurysms are usually the result of continuous spread from a cavernous sinus thrombosis and are secondary to infective endocarditis.[3] However, our patient had no previous history of cardiovascular or infectious disease and therefore was at a low risk for developing a mycotic aneurysm.

Hanafy et al. reported that poor clinical Hunt-Hess grades on

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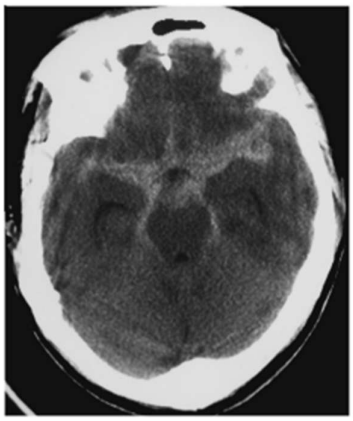

Fig. 1. Axial CT scan showing the subarachnoid haemorrhage with particular accumulation of blood in the left lateral fissure.

response that was initiated at the time of aneurysm rupture.[4] Our patient presented with a Hunt-Hess grade of IV. The genesis of inflammation after SAH is also supported by the literature, as it has been shown that the erythrocytes that accumulate in the subarachnoid space lyse and release inflammatory cytokines.[5] In addition, abscesses have a predilection to form in a region with edema.[6] Thus, the site of a ruptured aneurysm is a location where a brain abscess could quite easily develop. Hence, the mechanisms behind the development of a brain abscess after endosaccular embolisation for the treatment of an aneurysm may be complicated and multiple mechanisms are likely to be involved.

In the literature, the majority of patients with an intracranial abscess were treated with antimicrobial agents. One patient was treated with surgical puncture and external drainage of the abscess.[6,7] Our patient's death could have been caused by the abrupt decompression of the abscess, causing concurrent decompression of the aneurysm, which led to aneurysm rupture. Another possibility is partial treatment of the aneurysm. Finally, the infection could have caused a disruption in the aneurysm wall that made the aneurysm more susceptible to rupture. We regret that we did not perform microbiological examination of the tissue from our patient.

Finally, it is important to remember that abscess is a potential early complication following aneurysm embolisation. When deciding whether to treat such an abscess, the relationships between the abscess, the aneurysm, and the potential for haemorrhagic should be considered.

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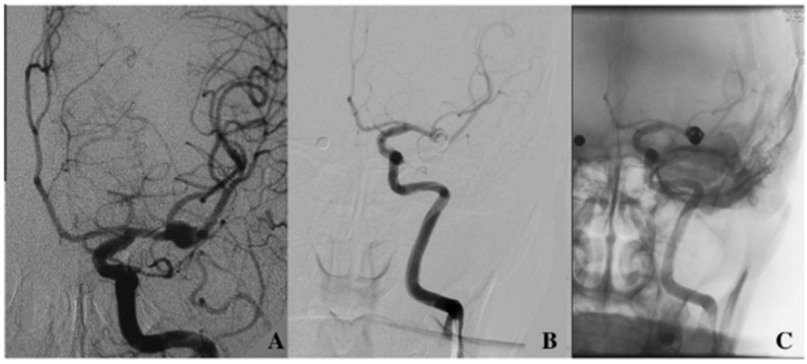

Fig. 2. Digital subtraction angiography of the left intracranial carotid artery. (A) Angiogram (oblique view) performed prior to the surgery showing the aneurysm located at the bifurcation of the left middle cerebral artery and a bleb at the top of the aneurysm. (B, C) Angiograms (anteroposterior view) performed immediately after embolisation of the aneurysm showing a small residual neck of the aneurysm remaining after the procedure.

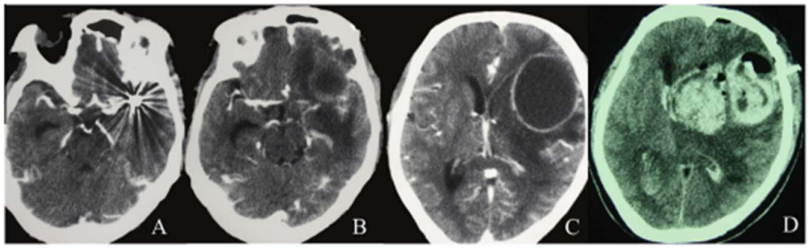

Fig. 3. (A–C) Axial contrast-enhanced CT scan showing the coils and brain abscess adjacent to the aneurysm. (D) Axial CT scan showing the ruptured aneurysm following the external drainage procedure.

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CADASIL presenting with a behavioural variant frontotemporal dementia phenotype

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ABSTRACT

The behavioural variant of frontotemporal dementia (bvFTD) is characterised by personality change with a decline in cognition. We describe two patients with cerebral autosomal dominant arteriopathy with subcortical infarcts and leukencephalopathy (CADASIL) who presented with behavioural phenotypes similar to bvFTD. The first patient presented with progressive personality and behavioural change, had florid white matter hyperintensity, and had a novel missense mutation C366W in exon 7 of the Notch3 gene. The second patient presented with progressive memory impairment and marked personality changes after a transient ischaemic attack. In this second patient, the radiological features were subtle and only the family history of stroke prompted testing for CADASIL using Notch3 genotyping. We present these patients to demonstrate that CADASIL may mimic bvFTD, with little clinical or radiological evidence to distinguish the two. CADASIL may be an under-recognised diagnosis in apparent bvFTD. Screening Notch3 in a substantial and unselected cohort of frontotemporal dementia patients might be appropriate to investigate this possibility.

1. Introduction

The behavioural variant of frontotemporal dementia (bvFTD) is characterised by personality change with a decline in cognition.[1] Here, two patients with cerebral autosomal dominant arteriopathy with subcortical infarcts and leukencephalopathy (CADASIL) – one with a novel missense mutation C366W in exon 7 of the Notch3 gene – are presented, whose behavioural phenotype closely resembled that of bvFTD.

2. Case report

2.1. Patient 1

This 68-year-old woman presented to general neurological services following concern from her family and neighbours about progressive personality and behavioural change over the past 2 years. The patient lived alone and was previously independent. Her family's principal concern related to new problems organising personal finances, hoarding of rubbish causing her house to be unsanitary, and increasing reclusiveness. The patient was relatively insightless as to the extent of her difficulties and extremely resistant to the idea of outside help for housekeeping or self-care. When the possibility of a social work referral was mentioned she became very angry and left the consultation, only persuaded back later by her family. Her autobiographical memory was relatively preserved. She could remember recent novels she had read and discuss their merits, although with a rather fatuous affect. She was distractible and the consultation was interrupted repeatedly by trips to the toilet. There was no past medical history or family history of note, including no history of cerebrovascular disease.

On cognitive examination, the patient's Mini-Mental State Examination score was 24/30 and 65/100 on the Addenbrooke's Cognitive Examination–Revised version, losing points mostly on verbal fluency, and also on memory and semantic knowledge items. MRI demonstrated florid, confluent white matter hyperintensity extending throughout both cerebral hemispheres notably including the rostral temporal lobes (Fig. 1a). Normal investigations included a routine full blood count and biochemistry, anti-nuclear antibodies, anti-neutrophil cytoplasmic antibodies, anti-cardiolipin antibodies, very long-chain fatty acids and white cell enzymes.

Sequencing of Notch3 identified a novel heterozygous missense mutation (C366R) in exon 7, where an arginine was substituted for a conserved cysteine residue. This is a novel mutation for CADASIL, but is in keeping with previously described mutations, which almost invariably involve cysteine mutations in epidermal growth factor repeats.[2,3] These features strongly support the pathogenicity of this dominant mutation. However, testing for osmophilic

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