

CASE REPORTS

Bihemispheric cerebral abscesses from infected anterior communicating artery aneurysmal coil

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Abstract

This is the report of an anterior communicating artery aneurysmal coil as the likely source of septic emboli resulting in bilateral hemispheric abscesses sparing the medial frontal, parietal and occipital lobes and brainstem. Cessation of antegrade anterior cerebral artery flow and resulting turbulent flow into the distal cortical arterial watershed of middle cerebral artery territory bilaterally is the probable mechanism for this unusual pattern.

INTRODUCTION

Multiple bihemispheric cerebral abscesses are an unusual presentation for a single intracranial embolic source. A systemic source such as endocarditis may be suspected but bilateral cerebral involvement with sterile blood cultures and sparing of other systemic organs is not expected.

The following case illustrates an anterior communicating artery aneurysmal coil as the likely source of septic emboli resulting in bilateral hemispheric abscesses sparing the medial frontal, parietal and occipital lobes and brainstem. Cessation of antegrade anterior cerebral artery (ACA) flow and resulting turbulent flow into the distal cortical arterial watershed of middle cerebral artery (MCA) territory bilaterally is the probable mechanism for this unusual pattern.

CASE REPORT

A 59-year old insulin-dependent diabetic underwent anterior communicating artery (AComA) aneurysm coil placement four years ago, complicated by ACA distribution infarction. Her neurologic deficits stabilized several weeks later and she was placed in a skilled nursing home with residual right hemiparesis with impaired cognition. One week prior to admission she had cough without fever or chills. She developed new left-sided weakness over the course of several days and was admitted. General examination was unremarkable aside from poor dentition. Admission CT scan revealed midline coil artifact

with old bilateral ACA distribution infarction, left worse than right, and extensive white matter changes with midline shift. (Figure 1 A, B) Brain MRI revealed six supratentorial ring-enhancing lesions with associated edema. (Figure 1 C, D) Spinal tap was deemed safe and results revealed 11 WBC /mm³, lymphocytic predominant with elevated protein of 112 mg/dL and glucose of 63 mg/dL. Extensive systemic evaluation for possible malignancy and source of infection were unrevealing. Blood cultures, transthoracic and transesophageal echocardiogram results were negative. Despite being placed on broad-spectrum antibiotics and dexamethasone she deteriorated becoming stuporous with worsening right hemispheric mass effect. She underwent diagnostic brain biopsy.

Right frontal craniotomy revealed well-encapsulated purulent abscess, containing a mixture of gram+ cocci and rods, but subsequent cultures for fungal, viral, bacterial organism were unrevealing.

DISCUSSION

Despite increasing use of intracranial coils for mostly aneurysmal obliteration, reported complications are surprisingly scarce. Localized hemorrhage and distal downstream coil embolism may occur at the time of the procedure. Delayed infectious complications are rare, but when it does happen the most common complication appears to be a localized intracranial abscess adjacent to the coils. Distant septic embolism resulting in

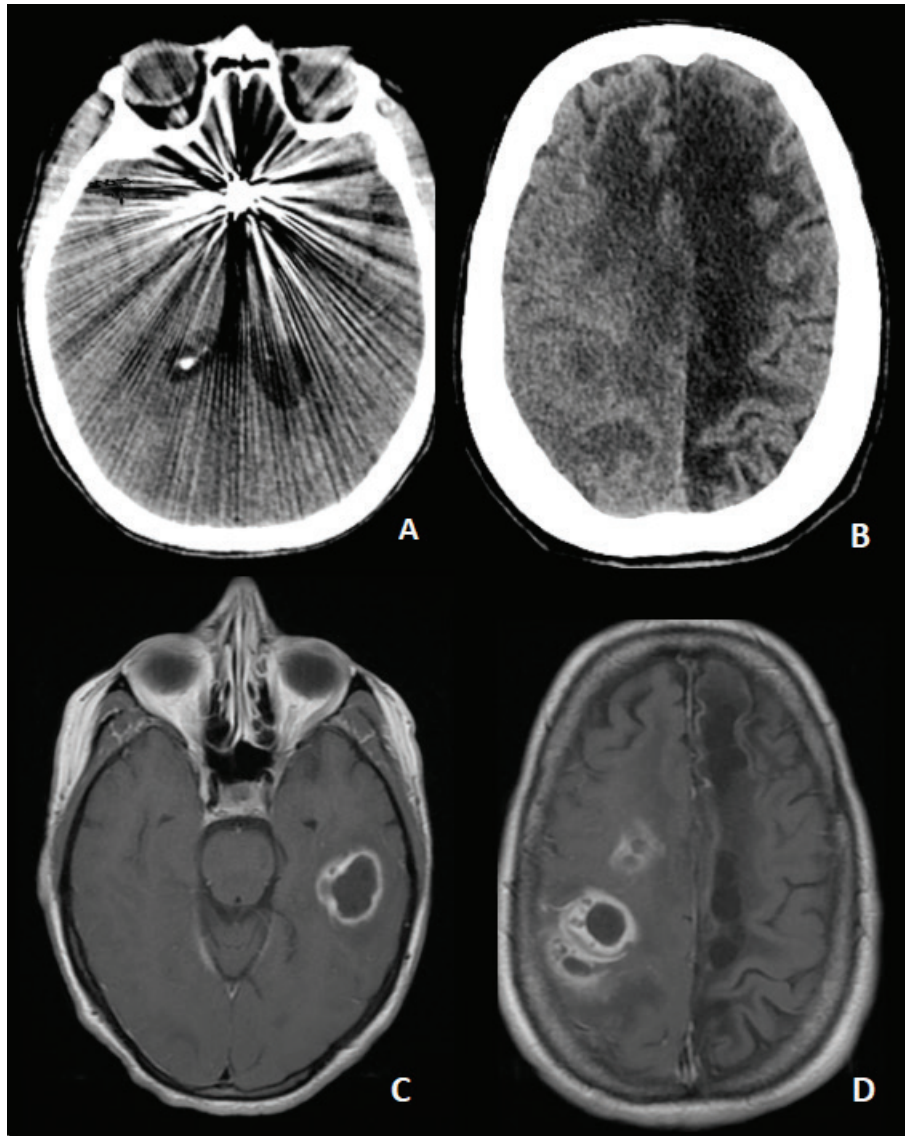


Figure 1. (A) Axial head CT scan slices revealing signal artifact due to anterior communicating artery coils. (B) Encephalomalacia left greater than right from previous anterior cerebral artery territory infarctions. Superimposed right hemisphere swelling causing midline shift to left. (C, D) Axial gadolinium enhanced T-1 weighted MRI scan slices reveal multiple ring enhancing lesions bilaterally with associated edema.

cerebritis and subsequent abscess formation in the absence of localized coil abscess as illustrated here seems unusual.

Recent comprehensive literature review of cerebral abscesses following intracranial aneurysmal coiling identified only six cases.¹ Four cases reported abscess formation adjacent to the coils but two had only distal downstream abscesses in the area of previous ischemic infarct.^{1,2} Symptomatic onset from weeks up to 3.5 years after coil placement is consistent with secondary coil seeding. Similarly, Rudell

and Chang³ reported a left external jugular coil (placed to treat a throat cancer) as the source of ipsilateral endophthalmitis and multiple ipsilateral hemispheric cerebral abscesses without localized infection five months after the initial coiling. Tooth extraction prior to onset of symptoms was thought to be causative in that case.

Initial coil placement in our case was complicated by subsequent bilateral ACA distribution stroke. In contrast to the two previously reported cases where the abscess formed at the site of the previous ischemic

stroke, the abscesses in our case spared the core of the ACA territory. This sparing of parasagittal region is consistent with the MCA as the conduit of embolism. Strategic coil placement likely resulted in cessation of distal antegrade flow in the ACA distribution. Stagnant flow proximal to the AComA coils combined with “vacuum” effect especially during diastole likely resulted in turbulent blood flow gaining access to distal MCAs. Previously, an elegant multi-directional velocity 3D MRI mapping has demonstrated how an atheromatous plaque from descending aorta can cause stroke by a retrograde mechanism during diastole.⁴

Embolization, whether from systemic – or as in this case, local – source tends to distribute to the distal cortical arterial watershed of the involved territory. Downstream septic embolic pattern in the distal cortical arterial watershed of the bilateral MCA, in a distribution that in outline is reminiscent of that of a ram’s horns, sparing medial frontal, parietal (supplied by ACA) and occipital regions (supplied by posterior cerebral artery), may be the result of an infected AComA coil.

DISCLOSURE

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