

Fig. 4. CT abdomen showing a contrast-enhancing focal thickening in the first part of the duodenum.

This emphasizes that radical tumour resection with a combination of radiotherapy, where indicated, leads to long-term disease progression-free survival.

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Successful coil embolization of a ruptured mycotic aneurysm that developed three days after septic embolic infarction: Case report and review of the literature



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ABSTRACT

Background: Infectious intracranial aneurysms (IIAs) are rare entities and are often associated with septic embolus from infectious endocarditis. They may develop rapidly and carry a higher risk of rupture and mortality compared to noninfectious aneurysms. However, the development and rupture of an IIA within 48 h in a patient with septic infarction patient is exceedingly rare.

Case description: In this report, we describe a 25-year-old male who presented with left-sided hemiparesis and dysarthria from septic embolus to the right middle cerebral artery. Thirty-nine hours after presentation, he became encephalopathic following a witnessed seizure. Angiography demonstrated a new, ruptured aneurysm, which was successfully treated with endovascular coil embolization. Our study documents the first report of coil embolization in a rapidly developed infectious aneurysm.

Conclusions: Importantly, this case demonstrates that septic infarction may precede and herald IIA development and rupture. If IIA is detected due to rupture, coil embolization can be a safe and effective therapy.

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Abbreviations: IIA, infectious intracranial aneurysm; SAH, subarachnoid hemorrhage; CT, computed tomography; MCA, middle cerebral artery; tPA, tissue plasminogen activator; HD, hospital day.

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1. Introduction

Infectious intracranial aneurysms (IIAs, also referred to as microbial or mycotic intracranial aneurysms) are rare entities, comprising 0.7–6.5% of all intracranial aneurysms [1]. These may develop rapidly and have a higher risk of rupture compared to

non-infectious aneurysms. Patients with IIA often present with symptoms associated with infection or aneurysmal subarachnoid hemorrhage (SAH), but rarely as significant neurologic deficits from cerebral thromboembolism. Here, we describe the first case of coil embolization in a patient presenting with ischemic stroke from septic embolism with subsequent development and rupture of IIA during the same hospitalization.

2. Case report

A 25-year old male presented to the emergency department with sudden onset of dysarthria and left hemiparesis. He had a prior history of deep venous thrombosis, dental caries, and multiple drug abuse with heroin and cocaine use earlier that day. His initial National Institutes of Health Stroke Scale was 3, and computed tomography (CT) images of the head showed a hyperdense right middle cerebral artery (MCA) sign. He was transferred to our comprehensive stroke center after receiving intravenous tissue plasminogen activator (tPA).

Upon arrival, a CT-angiogram showed partial occlusion of the right MCA proximal to its bifurcation (Fig. 1A and B). Hematology showed a white blood cell count of 12,800 cells/ μ L; C-reactive protein was 46.6 mg/L. He improved markedly overnight with repeat head CT on hospital day #2 (HD #2) demonstrating an acute infarct in the right basal ganglia and anterior insular cortex (Fig. 1C). On

auscultation, a new holosystolic murmur was identified. Blood cultures were positive for gram-positive streptococci and broad-spectrum antibiotics were started.

The patient experienced a generalized seizure overnight and received intravenous phenytoin and diazepam and was intubated (HD #3). Repeat head CT performed 39 h after initial presentation demonstrated diffuse subarachnoid hemorrhage with intraventricular extension and moderate acute hydrocephalus (Fisher grade 3, Hunt-Hess grade 4, Fig. 1D), for which an external ventricular drain was placed. Cerebral angiography performed on HD #4 demonstrated mild to moderate diffuse vasospasm and a 5 mm \times 3 mm aneurysm at the origin of one of the M2 branches (Fig. 2B), which was treated with coil embolization. Post-treatment angiography showed Raymond 3 residual filling in the aneurysm dome (Fig. 2C).

His course was complicated by cerebral vasospasm, which required daily administration of intra-arterial verapamil from the fifth day till eighth day. His blood cultures grew *Viridans Streptococci*, for which he received a course of intravenous penicillin. Transesophageal ultrasound demonstrated a flail anterior mitral valve leaflet with a small vegetation. He was discharged to a rehabilitation facility at 3 weeks.

Upon follow up at 5 weeks, he displayed remarkable recovery, with mild dysarthria, left facial droop, left upper extremity weakness, and a modified Rankin Score of 2. He is scheduled for a mitral valve replacement.

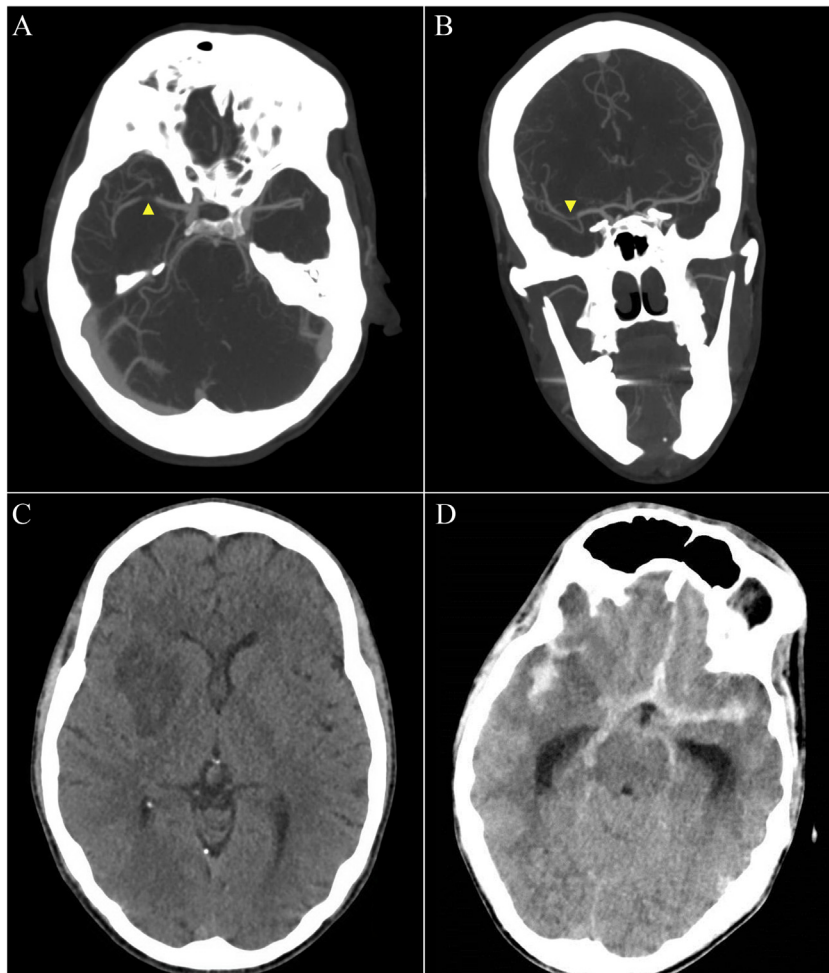


Fig. 1. Axial (A) and coronal (B) views of a computed tomography (CT) angiogram on hospital day #1 showing occlusion (arrowheads) in the M2 segment just distal to the middle cerebral artery (MCA) bifurcation. (C) Axial view of a CT of the head on hospital day #2 showing hypodensity within the right basal ganglia and insular cortex, representing an acute infarct. (D) Axial view of a CT of the head on hospital day #3 showing diffuse subarachnoid hemorrhage with intraventricular extension and resulting moderate acute hydrocephalus.

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