Isolated fever caused by an unruptured giant intracranial aneurysm

Stephanie Lenck

Pascal J. Mosimann, MD

Emmanuel Houdart, MD

Aside from rupture, giant intracranial aneurysms of the anterior communicating artery complex may be revealed by mass effect. Optic pathway compression will cause visual disturbances while compression of the brain or hydrocephalus may lead to cognitive impairment.¹ We report an isolated fever as a new clinical presentation of a giant aneurysm of the anterior communicating artery complex and discuss its pathophysiology.

Case report

A 50-year-old woman was admitted in 1994 for the workup of a 2-month-long isolated fever. Her medical history was marked by healed hepatitis B virus viral hepatitis 25 years ago, resection of a cold thyroid nodule, and tumorectomy of a benign mass in her left breast 12 years prior. There was no history of recent travel or contagious diseases in her close surroundings. Body temperature was mildly elevated, reaching 38.5°C. Clinical examination was unremarkable. White blood cell count, C-reactive protein, sedimentation rate, blood and urine cultures, chest x-ray, and cardiac workup were normal. A cerebral CT scan ruled out sinusitis but revealed a strongly enhancing basal frontal mass. Cerebral MRI identified a 27-mm partially thrombosed giant aneurysm located above the optic chiasm extending toward the floor of the third ventricle (figure). Digitally subtracted angiography (DSA) showed the aneurysm to be implanted on the origin of the A2 segment of the left anterior cerebral artery (ACA). Because of the high risk of subarachnoid hemorrhage, endovascular occlusion of the parent artery was decided. An occlusion test with a microballoon temporarily inflated in the left anterior cerebral artery was performed in order to assess the clinical tolerance and the presence of functional leptomeningeal anastomoses. The test occlusion was well tolerated and a definitive occlusion of the left ACA was achieved by balloon detachment. The patient was discharged home 4 days later with a normal neurologic examination, albeit persistent fever. At 4 weeks follow-up, body temperature still rose to 38.5°C. Aneurysm volume and mass effect were unchanged on MRI but complete thrombosis of the sac was now visible. At 3 months follow-up, complete shrinkage of the aneurysm had occurred and body temperature had normalized. The patient was subsequently followed clinically during 17 years and body temperature repeatedly remained within normal ranges. In 2011, a cerebral MRI was performed and confirmed disappearance of the previous mass effect on the midline structures and the absence of new aneurysms.

Department of Neuroradiology, Hôpital Lariboisière, Paris, France.

Correspondence to: emmanuel.houdart@lrb.aphp.fr

Figure Cerebral MRI shows the partially thrombosed giant aneurysm extending toward the floor of the third ventricle

DISCUSSION

We report an unruptured giant intracranial aneurysm revealed by an isolated fever. We suggest that the particular location of the aneurysm, displacing the hypothalamus, may explain this peculiar clinical presentation. Since the preoptic anterior hypothalamus (POAH), located on the floor of the third ventricle, mediates thermoregulation,² its dysfunction may be explained by 2 mechanisms: first by an inflammatory reaction caused by the partially thrombosed giant aneurysm, triggering a fever response of central origin,² and second by mass effect causing functional disruption of the POAH. Fever revealing tumors of the midline is exceptional but has previously been published. Pituitary lymphoma,³ glioblastoma of the pineal region,⁴ and recurrent Rathke cleft⁵ are the only reported cases of a midline mass presenting with fever without aseptic meningitis. In those cases, fever was associated with neurologic symptoms, visual disturbances, or neuroendocrine dysfunction. In our case, however, fever was strikingly isolated.

The correlation between fever disappearance and aneurysm shrinkage with 17 years of follow-up strongly supports our theory that the giant intracranial aneurysm was the cause of temperature dysregulation.

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DISCLOSURES

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