Commentary

The authors report the unusual story of a 28-year-old man with subarachnoid (SAH) and intracerebral hemorrhage from a right middle cerebral artery aneurysm.[1] He developed bilateral basal ganglia infarctions probably about 13 days after SAH, which left him permanently disabled. There was no symptomatic vasospasm, or as it is currently recommended to be called, delayed cerebral ischemia, in the middle cerebral artery territory.

Although the details are sparse, the patient seems to have been managed according to accepted standards with nimodipine and some attention to fluids and blood pressure. Current recommendations do not include "triple H" therapy, or prophylactic hemodynamic measures.[2] Unfortunately, vascular imaging was not done in a delayed fashion, which limits our ability to determine the etiology of the infarctions.

What can we learn from this case? Isolated perforator territory infarctions are not uncommon after SAH, but there is usually an obvious cause. Hoh and colleagues reviewed 619 patients who underwent neurosurgical clipping or endovascular coiling of ruptured aneurysms.[3] New infarctions were noted in 189 (30%) of patients. The etiology was perforator artery occlusion in 40 (8%). These were almost all a complication of neurosurgical clipping or occasionally, coiling. Naidech and colleagues reported single or multiple deep (presumably mostly

perforator territory) infarctions in 23 of 117 patients (20%) with SAH.[4] The etiology was not specifically determined. Rabinstein, *et al*, found 56 infarctions in 142 patients (39%) with aneurysmal SAH.[5] Isolated deep infarctions occurred in 10 (18%) cases. Thus, perforator-territory infarction occurs in up to 20% of patients with aneurysmal SAH. Perforator-territory infarctions also are well-described secondary to large artery (anterior or middle cerebral artery mainly) occlusion or severe angiographic vasospasm in patients with SAH, which by default involves perforating arteries.[3-5] This also is well described in early pathology studies[6] and clinical series.[3,4]

What is the cause of isolated perforator-territory infarctions? The unusual features of this case are the delayed appearance and remoteness from the site of the ruptured aneurysm. The causes of infarction after SAH are numerous, we classified them in recent clinical trials as complication of the aneurysm securing procedure, due to angiographic vasospasm, encephalomalacia from ventricular catheter or intracerebral hemorrhage, other identifiable cause (angiographic complication, increased intracranial pressure among others) and unknown.[7] The attribution of cause to the infarction is difficult, however, and not easily validated.[4,8] The most common cause of perforator infarction is surgical complication, which is not the case here. Crompton noted that in patients dying of SAH, perivascular blood was commonly seen tracking up the spaces around the penetrating arterioles but this tended to be where the aneurysm was, for example, along middle cerebral perforators with middle cerebral artery aneurysm rupture.[9] This was frequently associated with basal ganglia infarctions. The infarctions in this man, however, were remote from the aneurysm so this does not seem to be the cause here either. The authors suggest several possibilities, most prominently that the infarctions are due to vasospasm. Unfortunately, this is difficult to document. Others have suggested that isolated deep perforator territory infarctions can be due to vasospasm. Rabinstein and colleagues report 6 such cases, although the perforator territories in these cases were all next to the ruptured aneurysm.[5] They note these infarctions often did not correlate well with angiographic vasospasm, and suggest other mechanisms such as spasm of the perforating arteries, infarction secondary to autoregulatory disturbance and microthromboemboli.

In this patient, there was no documented hypotension so infarction from autoregulatory dysfunction seems unlikely. It also would be unusual to have isolated perforator artery spasm distal from the aneurysm. Microthromboemboli are postulated to contribute to delayed cerebral ischemia as well. Treatment of patients with SAH with antiplatelet agents, however, has so far not shown remarkable benefit and would be thus not recommended on a routine basis based on this case.[10] Did the patient have another underlying reason to develop infarction, such as pre-existing chronic hypertension (there was no evidence for this) or some hypercoagulable state? We do not know this either. Thus, this case is important for documenting an unusual complication after SAH. We were unable to find a similar case when searching the literature. Infarction after SAH remains a major contributor to poor outcome,[11] and hopefully, with understanding of cases such as this, we can learn more and begin to improve outcome in patients with SAH.

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References

1. Salunke P, Gupta SK. Symptomatic bilateral perforator vasospasm following aneurysmal subarachnoid haemorrhage. J Neurosci Rural Pract 2012;4:45-7.
2. Connolly ES Jr, Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, *et al*. Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke 2012;43:1711-37.
3. Hoh BL, Curry WT Jr, Carter BS, Ogilvy CS. Computed tomographic demonstrated infarcts after surgical and endovascular treatment of aneurysmal subarachnoid hemorrhage. Acta Neurochir (Wien) 2004;146:1177-83.
4. Naidech AM, Bendok BR, Bassin SL, Bernstein RA, Batjer HH, Bleck TP. Classification of cerebral infarction after subarachnoid hemorrhage impacts outcome. Neurosurgery 2009;64:1052-7.
5. Rabinstein AA, Weigand S, Atkinson JL, Wijdicks EF. Patterns of cerebral infarction in aneurysmal subarachnoid hemorrhage. Stroke 2005;36:992-7.
6. Birse SH, Tom MI. Incidence of cerebral infarction associated with ruptured intracranial aneurysms. A study of 8 unoperated cases of anterior cerebral aneurysm. Neurology 1960;10:101-6.
7. Macdonald RL, Higashida RT, Keller E, Mayer SA, Molyneux A, Raabe A, *et al*. Preventing vasospasm improves outcome after aneurysmal subarachnoid hemorrhage: Rationale and design of CONSCIOUS-2 and CONSCIOUS-3 trials. Neurocrit Care 2010;13:416-24.
8. Ibrahim GM, Weidauer S, Vatter H, Raabe A, Macdonald RL. Attributing

hypodensities on CT to angiographic vasospasm is not sensitive and unreliable. Stroke 2012;43:109-12.
9. Crompton MR. The pathogenesis of cerebral infarction following the rupture of cerebral berry aneurysms. Brain 1964;87:491-510.
10. Dorhout Mees SM, van den Bergh WM, Algra A, Rinkel GJ. Antiplatelet therapy for aneurysmal subarachnoid haemorrhage. Cochrane Database Syst Rev 2007;4:CD006184.
11. Fergusen S, Macdonald RL. Predictors of cerebral infarction in patients with aneurysmal subarachnoid hemorrhage. Neurosurgery 2007;60:658-67.

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