
Neuroimaging Highlight

Editors: Richard Farb, David Pelz

Delayed Neurological Deterioration in ICH Due to Cerebral Infarction

Submitted by: B.K. Menon, J. Singh, M.D. Hill

Can. J. Neurol. Sci. 2010; 37: 397-399

Cerebral infarction occurring shortly after hypertensive intracerebral haemorrhage (ICH) is uncommon and was not widely recognized until the recent publication of the Factor VIIa trials^{1,2}. The causes could be coincidental lacunar infarcts due to arteriolar disease (the same pathophysiology underlying the ICH), compression of surrounding arteries due to mass effect and iatrogenic hypoperfusion due to treatment of hypertension or rare other causes^{3,4}.

We describe a 85-year-old female with history of hypertension on treatment and no other modifiable risk factors, not on

any antiplatelets or anticoagulants, who presented to us with dysarthria, left hemiparesis and a National Institute of Health Stroke Scale score of 9 on admission. Blood pressure on admission was 185/110 mm hg. Computed tomogram (CT) brain showed a right external capsular haemorrhage measuring 17 ml and CT angiogram (CTA) showed a right M1-MCA near occlusion/tight stenosis. Our patient most likely had a hypertensive ICH due to small vessel disease. Her antihypertensive medication dosage was titrated for optimal control. She recovered slowly and was discharged to a



Figure 1: CT Brain on presentation showing a moderate size right external capsular haemorrhage.

From the Department of Clinical Neurosciences, University of Calgary, Health Sciences Centre, Calgary, Alberta, Canada.

RECEIVED JULY 9, 2009. FINAL REVISIONS SUBMITTED OCTOBER 6, 2009.

Correspondence to: Bijoy K Menon, Dept of Clinical Neurosciences, University of Calgary, Rm EG5, Foothills Hospital, 1403 - 29th Street NW, Calgary, Alberta, T2N 2T9, Canada.



Figure 2: CT Angiography Head on admission showing the right M1 MCA “near” occlusion.

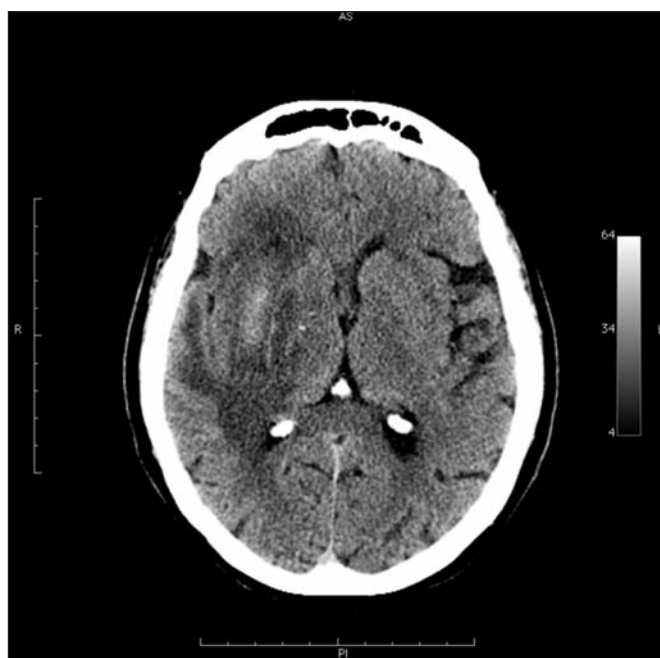


Figure 3: CT Brain two weeks later with clinical worsening showing resolving haemorrhage when compared to the previous CT and significant surrounding hypodensity.

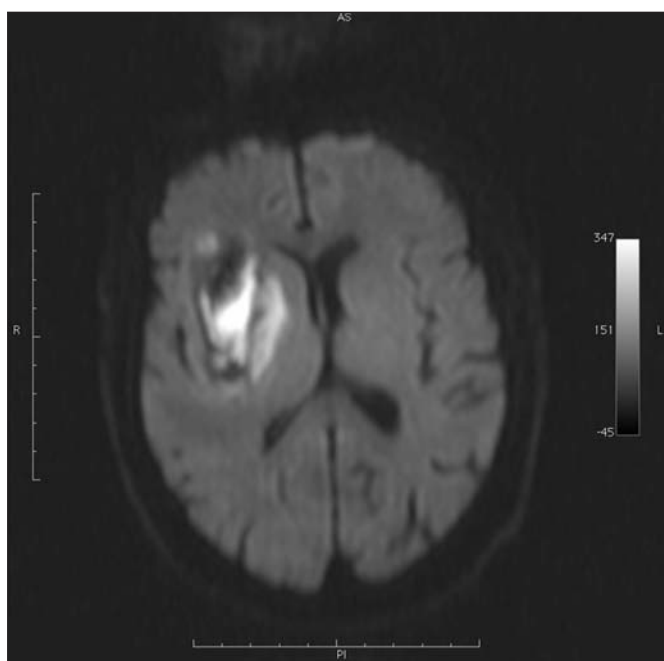


Figure 4: MRI Brain Diffusion weighted sequences (DWI) two weeks later with clinical worsening showing restricted diffusion surrounding the resolving haemorrhage.

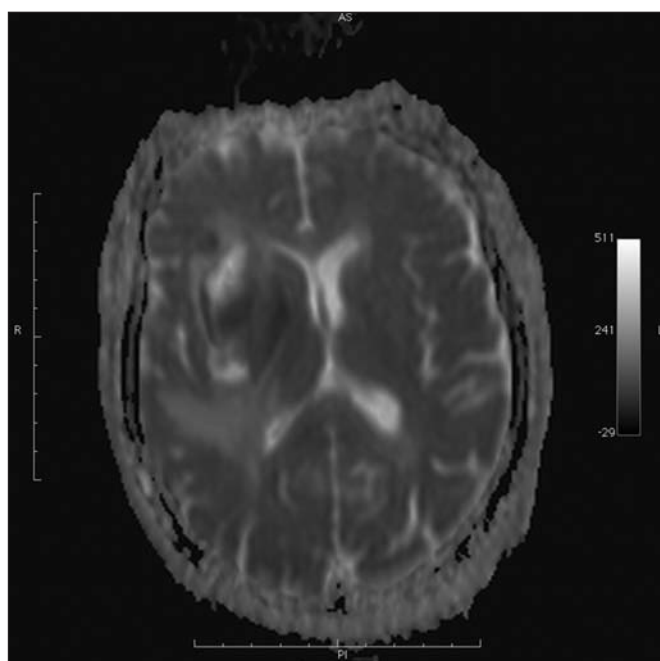


Figure 5: MRI Brain Apparent Diffusion Coefficient (ADC) map two weeks later showing decreased signal in the area of diffusion restriction suggesting an acute infarct.

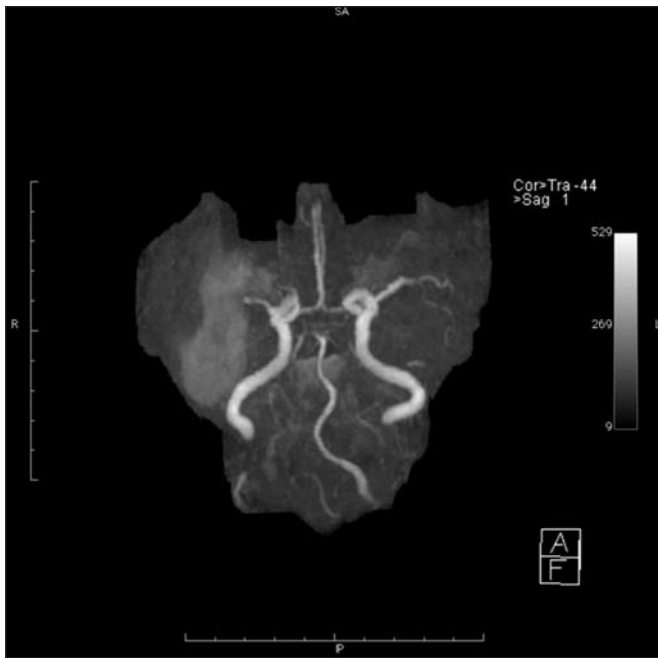


Figure 6: MRA Circle of Willis two weeks later showing right M1 MCA occlusion.

rehabilitation hospital for ongoing therapy. After two weeks, she re-presented with sudden worsening of her left hemiparesis. Blood pressure on admission was 140/88 mm Hg. Computed tomography showed an evolving haematoma but with significant surrounding hypoattenuation in the MCA arterial territory. Magnetic resonance imaging (MRI) showed diffusion restriction in the right frontal region, basal ganglia medial to the site of the ICH and corona radiata with occlusion of the right M1-MCA segment on magnetic resonance angiography (MRA).

Aetiology of peri haematoma lucency or edema is controversial and is postulated to be due to either interstitial edema (secondary to oncotic pressure exerted by serum proteins derived from the clot and occurring early) or a combination of cytotoxic and vasogenic edema (due to inflammatory response produced by serum proteases, blood degradation products, release of free radicals, apoptosis, etc and occurring later)⁵. Some studies suggest that late edema progression predominantly represents vasogenic rather than cytotoxic edema⁶.

Delayed neurological deterioration after spontaneous ICH is well known and is assumed to be due to increasing edema and the resultant mass effect surrounding the haematoma, unlike early neurological deterioration, which occurs due to haematoma expansion⁶. Progressive perihematoma edema without mass effect rarely causes neurological worsening⁷. Delayed neurological deterioration in our patient was associated with imaging evidence of perihematoma lucency on CT without significant mass effect and with evidence on MRI [Diffusion Weighted Imaging (DWI) and Apparent Diffusion Coefficient (ADC)] of cytotoxic edema/infarction involving a part of this

territory. The association of M1 MCA near occlusion/tight stenosis with the above imaging findings along with the absence of significant mass effect in the follow up scans leads us to speculate that delayed neurological worsening in our patient is likely due to hypoperfusion in the right M1-MCA territory distal to the stenosis and the resultant acute infarct seen on MRI (DWI and ADC). Perihematoma edema and mass effect due to the intracerebral haemorrhage along with iatrogenic lowering of blood pressure post ICH may have contributed to the resultant hypoperfusion. Our case highlights the need to look beyond perihematoma edema and associated mass effect in explaining causes of delayed neurological deterioration in spontaneous ICH.

REFERENCES

1. Mayer SA, Brun NC, Begtrup K, Broderick J, Davis S, Diringer MN, et al. Recombinant activated factor VII for acute intracerebral hemorrhage. *N Engl J Med*. 2005 Feb 24;352(8):777-85.
2. Mayer SA, Brun NC, Begtrup K, Broderick J, Davis S, Diringer MN, et al. Efficacy and safety of recombinant activated factor VII for acute intracerebral hemorrhage. *N Engl J Med*. 2008 May 15; 358(20):2127-37.
3. Kim CH, Kim JS. Development of cerebral infarction shortly after Intracerebral haemorrhage. *Eur Neurol*. 2007;57:145-9.
4. Sinar EJ, Mendelow AD, Graham DI, Teasdale GM. Experimental Intracerebral haemorrhage: effects of a temporary mass lesion. *J Neurosurg*. 1987;66:568-76.
5. Rincon F, Mayer SA. Novel therapies for intracerebral hemorrhage. *Curr Opin Crit Care*. 2004 Apr;10(2):94-100.
6. Zazulia AR, Diringer MN, Derdeyn CP, Powers WJ. Progression of mass effect after intracerebral hemorrhage. *Stroke*. 1999 Jun;30(6):1167-73.
7. Inaji M, Tomita H, Tone O, Tamaki M, Suzuki R, Ohno K. Chronological changes of perihematoma edema of human intracerebral hematoma. *Acta Neurochir Suppl*. 2003;86:445-8.