Re: the hyperdense middle cerebral artery sign in a polycythaemic child

The letter by Douis *et al.*[1] (p. 34) concerning the hyperdense middle cerebral artery (HMCA) sign brings to mind a case seen by myself several years ago. A 47-year-old man had developed an acute episode of jerking followed by paresis of his left arm and leg as well as facial numbness at approximately 14h00 one afternoon. After reaching the casualty department and following a neurological consultation he underwent an unenhanced CT scan of the brain at around 18h00. A mildly hyperdense appearance of the right internal carotid termination and adjacent proximal M1 segment of the right middle cerebral artery (MCA) was noted, more so than seen in other adjacent vessels, but not strikingly so (Fig. 1a). There was no evidence of parenchymal infarction or haemorrhage. In fact, his symptoms had largely resolved just prior to performing the CT scan. Given this CT appearance together with the short history and symptoms fitting a possible right-sided cerebral ischaemic episode, we took the patient directly to angiography within the same hour. This was done with a view to both confirmation of the presence of a clot and possible intra-arterial thrombolysis. The arteriogram showed no abnormalities of the right anterior cerebral vasculature including no proximal or distal intraluminal thrombus (Fig. 1b). One might have tended to ignore the initial CT picture except for the strongly correlative clinical signs. The fact that the symptoms had resolved in our patient could have meant collateral vessel recruitment or partial fragmentation of the clot with recanalisation, therefore warranting further investigation in our opinion. Our patient was not polycythaemic and the cause of this transient neurological episode was never established. Currently the initial investigation of choice for suspected acute intracranial arterial occlusion and/or infarction is MRI scanning. Many modern scanners allow rapid multisequence acquistions such as FLAIR, diffusion (and perfusion) and MR angiography sequences all within 10 - 15 minutes, permitting rapid confirmation of diagnosis and assessment of the degree of probable temporary versus permanent parenchymal damage particularly with reference to the potential therapeutic use of thrombolytic agents. However, as MRI facilities are not available at many centres throughout South Africa, reliance on CT scanning in this situation is still commonplace, primarily to distinguish between haemorrhagic and non-haemorrhagic stroke. We have noted the 'hyperdense' appearance of intracranial vessels to be more pronounced with some makes of CT scanner than with others, with changes in window settings also contributing to the overall visual impression of hyperdense vessels. A similar mildly hyperdense appearance is often seen in the major venous sinuses on CT scans of the brain, which we have also seen mistakenly diagnosed as acute sinus thrombosis. When an intraluminal thrombus is present then the vessel is usually somewhat more hyperdense than in the example shown in Fig. 1. We agree with Rauch *et al.*[2] that a hyperdense appearance to the MCA is in itself not a reliable indicator of vessel occlusion or impending infarction and that such findings should be correlated carefully with the clinical presentation before venturing on to more expensive and invasive investigations.

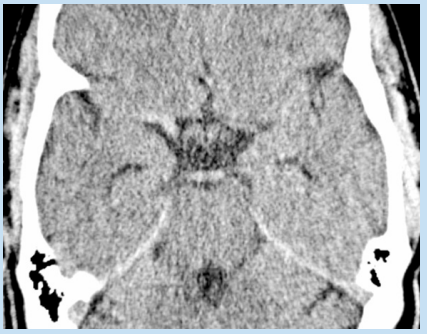

Fig .1a. Axial unenhanced CT scan showing the hyperdense appearance of the terminal right ICA and proximal MCA.

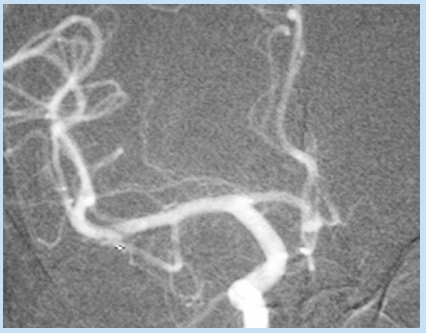

Fig .1b. Selective right internal carotid digital subtraction arteriogram shows normal patency of the distal right ICA and adjacent MCA.

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References

1. Douis H, Andronikou S, Jadwat S. The hyperdense middle cerebral artery sign in a polycythaemic child *South African Journal of Radiology* 2005; **9:** 34 (this issue).
2. Rauch RA, Bazon C 3rd, Larsson EM, Jinkins JR. Hyperdense middle cerebral arteries identified on CT as a false sign of vascular occlusion. *Am J Neuroradiol* 1993; **14:** 669-673.