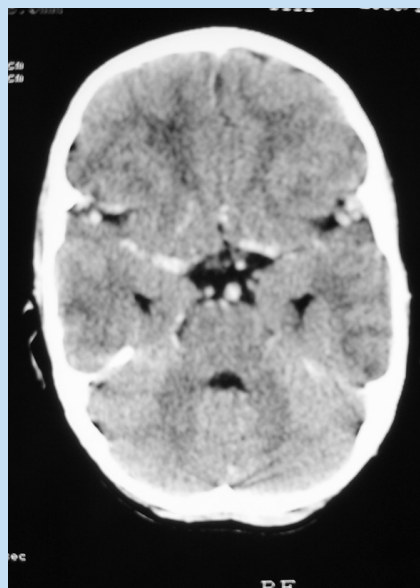


## The hyperdense middle cerebral artery sign in a polycythaemic child

**To the Editor:** The hyperdense middle cerebral artery (HMCA) sign is known to be an early sign of intracranial arterial occlusion or infarction during unenhanced CT imaging of the brain.<sup>1</sup> This sign has also been seen after treatment with bromide,<sup>2</sup> in cocaine abusers<sup>3</sup> and in adults with elevated haematocrit,<sup>4</sup> but has not previously been reported to be a result of polycythaemia in children. We recently encountered this sign as an incidental finding throughout the vessels of the circle of Willis in a child with a cyanotic heart condition and speculate that the dense appearance of these intracranial vessels was the result of polycythaemia. A 4-year-old girl was admitted with headaches, vomiting and fever. The child had an atrial septal defect, ventricular septal defect, pulmonary atresia and multiple aorto-pulmonary collateral arteries. Clinically, she was cyanotic and clubbed. The neurological examination was normal. Laboratory tests showed a haematocrit of 67.6% and haemoglobin level of 21.9 g/dl in keeping with polycythaemia. A CT scan of the head was performed in



Figs 1 a and b. Sequential non-contrast CT slices in a 4-year-old girl show increased density of the arteries of the circle of Willis due to polycythaemia.

order to exclude a brain abscess or meningitis. The uncontrasted CT scan did not show any findings of a brain abscess but the HMCA sign was seen involving both middle cerebral arteries as well as the basilar artery (Figs 1 a and b). There was no clinical or radiological evidence of cerebral infarction or vessel occlusion. The high haematocrit may therefore have increased the density of the blood leading to an HMCA sign. Although the HMCA sign has been shown to be a false indicator of vascular occlusion in adults,<sup>4</sup> an association between an elevated haematocrit and a HMCA in children has not yet been reported.

**H Douis  
S Andronikou  
S Jadwat**

*Department of Paediatric Radiology  
Red Cross Children's Hospital  
University of Cape Town*

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

The letter by Douis *et al.*<sup>1</sup> (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 clin-

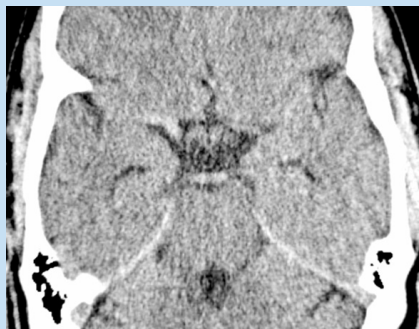


Fig. 1a. Axial unenhanced CT scan showing the hyperdense appearance of the terminal right ICA and proximal MCA.



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

ical 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 acquisitions 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 par-

ticularly 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.*<sup>2</sup> 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.

### I C Duncan

Unitas Interventional Unit  
Centurion

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