Arteriovenous shunting and early cortical venous filling in subacute cerebral infarction — an old angiographic finding revisited

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Abstract

Demonstrated in this report is an example of arteriovenous shunting and early venous filling in an area of cerebral infarction recorded on digital subtraction angiography. This angiographic appearance is largely of historical interest given the current use of sectional imaging (CT and MR) and altered role of angiography in the imaging of stroke, but should nevertheless still be considered amongst the differential causes of cerebral arteriovenous shunting.

Introduction

Since the 1970s sectional imaging with computed tomography (CT) and later magnetic resonance imaging (MRI) have replaced cerebral angiography as the methods of choice in the acute and follow-up imaging of cerebral infarction. Angiography tends to be performed early either to identify any treatable underlying pathology or for direct transarterial treatment by thrombolysis. As a result few cerebral angiograms are done nowadays in the subacute and chronic stages of an infarction and so many of the related angiographic changes during these stages are no longer seen. The case reported here is shown primarily to demonstrate the changes of luxury perfusion and arteriovenous shunting and early cerebral cortical venous filling as captured on digital subtraction angiography.

Case report

A 39-year-old male patient presented to a local academic hospital with clinical evidence of an acute subarachnoid haemorrhage. He was stuporose on admission. An unenhanced CT scan done on the day of admission showed a small clot in the basal subarachnoid cisterns and an area of non-haemorrhagic infarction in the right parietal lobe and posterosuperior aspect of the right temporal lobe.

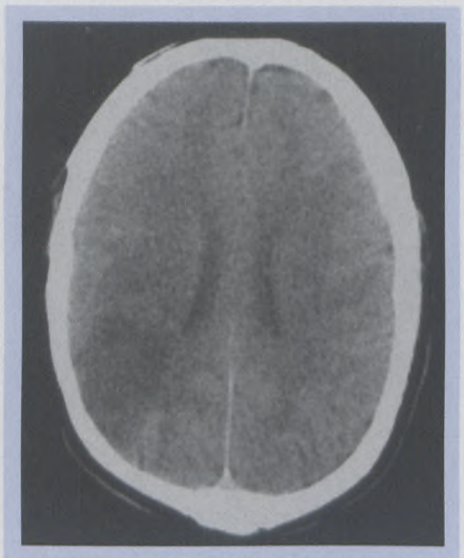

Fig. 1. Unenhanced axial CT scan showing the area of infarction in the right parietal lobe.

Cerebral angiography was performed 6 days after this admission. Selective right internal carotid angiography showed the presence of an elongated saccular aneurysm arising from the communicating segment of the right internal carotid artery (ICA). Vasospasm was seen in the region of the right middle cerebral bifurcation and in the right posterior communicating artery. A vascular blush was apparent in the region of the cerebral infarction during the late arterial and capillary phases of the selective right ICA run. Early filling of cortical veins in this region was noted during the late capillary phase prior to the normal venous return phase from the rest of the right cerebral hemisphere. This appearance is in keeping with luxury perfusion and rapid arteriovenous shunting through the infarcted area.

Discussion

The angiographic pattern of arteriovenous shunting with early filling of cortical veins related to an area of cerebral infarction will be well known to radiologists who performed cerebral angiography in the era prior to cross-

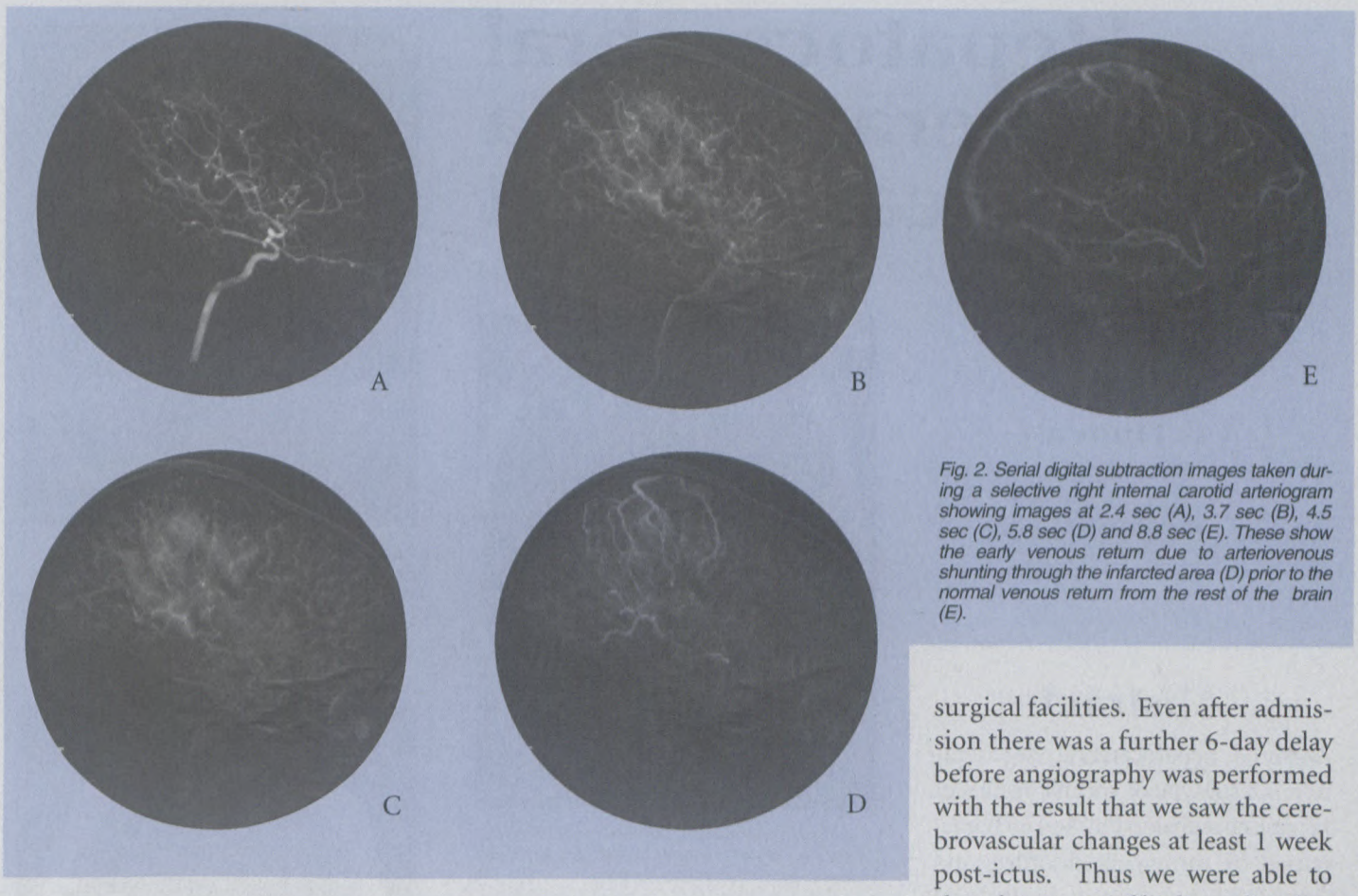

Fig. 2. Serial digital subtraction images taken during a selective right internal carotid arteriogram showing images at 2.4 sec (A), 3.7 sec (B), 4.5 sec (C), 5.8 sec (D) and 8.8 sec (E). These show the early venous return due to arteriovenous shunting through the infarcted area (D) prior to the normal venous return from the rest of the brain (E).

sectional imaging. In 1966, Lassen[1] introduced the term 'luxury perfusion' to describe hyperaemia within damaged brain tissue. Regional loss of autoregulation was thought to be due to lactic acid and carbon dioxide accumulation. This reaction could occur within or around an infarcted area. These changes resulted in the shunting of blood, either through dilated capillaries or arteriolo-venular shunts. Angiographically this was visible as an enhanced early capillary blush and early venous filling. These angiographic changes are particularly evident in the second to third weeks post-ictus, but early venous filling can occasionally be seen very soon after an infarct.[2,3] In 1973, Leeds and Goldberg[4] identified a host of pathologies that could exhibit 'luxury perfusion' including cerebral infarction, cerebral trauma, inflammatory diseases (including encephalitis, meningitis and abscess), vasculitis, parenchymal degenerative disease, encephalopathy, compression of brain around a space-occupying lesion, vascular spasm (secondary to subarachnoid haemorrhage), and repeated seizures or status epilepticus.

Today brain infarcts are diagnosed and followed up either with CT or MRI. Angiography is usually done early either to exclude an underlying arterial pathology in the neck or brain, or it is done for endovascular treatment with thrombolytic agents. As a result, angiography is not routinely done within days to weeks post-ictus. In our case there was an unknown period of delay before reaching a hospital with scanning and neurosurgical facilities. Even after admission there was a further 6-day delay before angiography was performed with the result that we saw the cerebrovascular changes at least 1 week post-ictus. Thus we were able to show by means of high-quality digital subtraction angiography this excellent example of luxury perfusion with arteriovenous shunting and early venous filling related to cerebral infarction, an angiographic appearance seldom seen nowadays.

References

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