

## LESSON OF THE MONTH

## Simultaneous onset of haemorrhagic and ischaemic strokes in a haemodialysis patient

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Primary brain haemorrhage and infarction only very rarely occur simultaneously. A patient with end stage renal disease from diabetic nephropathy suddenly had motor aphasia and horizontal nystagmus soon after finishing haemodialysis. Neuroradiological studies showed a haematoma on the right side of the pons and an infarct in the left frontal lobe with occlusion of the left internal carotid artery. Specific conditions of the haemodialysis—including anticoagulant use, relative hypovolaemia and hypertension just before haemodialysis, and an abrupt decrease in blood pressure during haemodialysis—seemed to be the major reason for the simultaneous onset of dual strokes.

Primary brain haemorrhage and infarction, especially lacunar infarction, develop on the basis of the same vascular pathology, that is, arteriosclerotic changes in the small perforating arteries. The two types of stroke, however, hardly ever occur simultaneously. We describe a patient with end stage renal disease who did develop primary haemorrhage and infarction in different regions of the brain simultaneously. Specific conditions of haemodialysis seem to be a major reason for the dual strokes in this patient.

## CASE REPORT

A 74 year old woman with hypertension and a 15 year history of maintenance haemodialysis for diabetic nephropathy suddenly became unable to speak soon after the end of her routine haemodialysis, for which heparin was used as an anticoagulant. In addition, she was barely able to look to the right. During the second half of the four hour haemodialysis period, involving the removal of 1700 ml of fluid on that day, her blood pressure had decreased from 190/90 to 125/65 mm Hg.

On arrival at our hospital 30 minutes after the onset of symptoms, her blood pressure was 191/83 mm Hg and her pulse rate was 81 beats/min and regular. She could only say her first name. She responded appropriately to verbal and written orders. She did not look to the right voluntarily or follow a target in that direction. She had low frequency nystagmus to the right and high frequency to the left. There was no paresis of her face, tongue, or limbs.

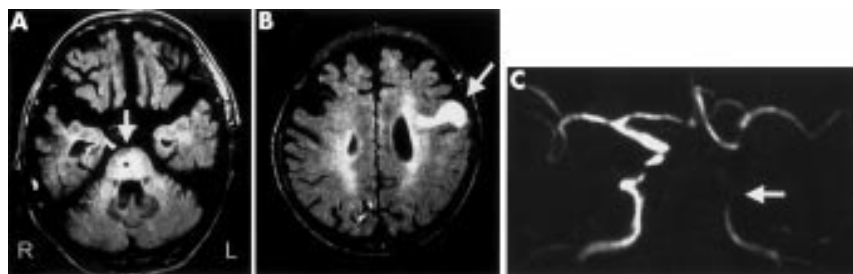
Cranial computed tomography immediately after her arrival at hospital showed a small haematoma 3 mm in diameter in the right pontine tegmentum, without fresh lesions in the supratentorium. On a carotid echogram, diastolic flow was not documented in the left common and internal carotid arteries, indicating occlusion of the left internal carotid artery. Thus we diagnosed a pontine haemorrhage with oculomotor disorder and left cerebral ischaemia with motor aphasia. Magnetic resonance imaging on day 3 showed a fresh infarct in the cortex and subcortex of the left inferior frontal gyrus (fig 1, A and B). Magnetic resonance angiography showed severe stenosis or occlusion of the left intracranial internal carotid artery with normal flow in the left middle cerebral artery, presumably derived from the anterior communicating artery (fig 1C). Blood tests showed mild anaemia and raised levels of thrombin-antithrombin III complex and D dimer, indicating a hypercoagulable state. Neither echocardiography nor prolonged electrocardiography revealed any cardiac source of emboli.

During first two days, we lowered her blood pressure to around 170/90 mm Hg and avoided the use of antithrombotic drugs in order to prevent enlargement of the haematoma. On day 3, because the pontine haematoma did not expand on MRI, we stopped antihypertensive treatment and started a continuous intravenous infusion of argatroban, an anti-thrombin anticoagulant. One month later, the haematoma in the pons had disappeared, and her oculomotor disorder had resolved. Her speech had also almost returned to normal.

## DISCUSSION

This is the first report of the simultaneous onset of haemorrhagic and ischaemic strokes immediately after haemodialysis.

The pontine haemorrhage in this patient seemed to follow rupture of the intrapontine perforating artery secondary to hypertension—the most common underlying pathology of brain haemorrhage.<sup>1</sup> Heparin use during haemodialysis might enhance bleeding. Other causes, including vascular malformations, tumours, amyloid angiopathy, and vasculitis, were not detected in this patient. The mechanism of the cerebral infarction in our patient seemed to be haemodynamic—that is, a critical decrease in distal cerebral perfusion caused by severe stenosis or occlusion of the proximal arterial supply.<sup>2</sup>



**Figure 1** Cranial magnetic resonance imaging (MRI; FLAIR imaging) on day 3, showing a hypointense signal in the right pontine tegmentum indicating a haematoma (panel A, arrow) and a hyperintense signal indicating infarction in the left inferior frontal cortex and subcortex (panel B, arrow). (C) Magnetic resonance angiography on day 3, showing severe stenosis or occlusion of the left internal carotid artery at the intracavernous segment (arrow).

She had occlusion of the left internal carotid artery and her systolic blood pressure fell by 65 mm Hg during haemodialysis on the day of stroke onset. Diminished vascular responses caused by diabetic autonomic neuropathy and advanced arteriosclerosis may explain an abrupt decrease in blood pressure during haemodialysis. We normally interrupt aggressive anti-hypertensive treatment in such patients and keep their blood pressure relatively high on the morning of haemodialysis to prevent a hypotensive accident. This could have the effect of increasing the risk of a haemorrhagic event during haemodialysis. Thus haemodialysis patients appear to be more susceptible to both haemorrhagic and ischaemic strokes than the general population.

Stroke is a common cause of death in patients on chronic haemodialysis. Recent studies in haemodialysis patients have shown a high annual incidence of stroke (12 to 18 per 1000 person-years) compared with the general population, and the incidence of brain haemorrhage is two to three times higher than the incidence of infarction.<sup>3,4</sup> Among haemodialysis patients in our institute and in our satellite clinics, approximately one fifth of cases of brain haemorrhage and one third of cases of brain infarction occur during or immediately after haemodialysis (Toyoda K, *et al*, personal communication). This suggests that haemodialysis treatment on its own is one of the major triggers of both brain haemorrhage and infarction in patients on haemodialysis for end stage renal disease.

For patients with stroke or a high risk of stroke, various technical changes to the haemodialysis regimen may be needed, including the use of low molecular weight heparin

instead of standard unfractionated heparin to decrease bleeding accidents,<sup>5</sup> and allowing a longer period for fluid removal to prevent an abrupt decrease in blood pressure.

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