sogenic edema in patients with conditions including hypertensive encephalopathy, preeclampsia-eclampsia, neurotoxicity of immunosuppressive drugs, and uremic encephalopathy [1]. The pathophysiology of PRES is, however, unclear. Endothelial dysfunction and failure of cerebral autoregulation in distal arterioles and capillaries are proposed mechanisms [2]. Although most patients with PRES have normal cerebral angiograms, conventional angiograms or MRA may demonstrate cerebral vasospasm in patients with hypertensive encephalopathy associated with pregnancy or intrathecal chemotherapy [8, 9]. Transcranial Doppler sonography has demonstrated that after the disappearance of signs and symptoms in a case of eclampsia, the flow velocity began to increase, likely due to delayed vasospasm [10]. Singhal [3] reported 4 postpartum patients with reversible cerebral vasospasm who did not meet the criteria for preeclampsia-eclampsia, and were diagnosed as having postpartum angiopathy (PPA). It is well known that cerebral vasospasm has an important role in ischemic injury, and PPA is frequently complicated by ischemic stroke. It should be noted, however, that the PPA patients reported by Singhal showed transient nonischemic brain lesions consistent with PRES. Taken together, cerebral vasospasm may occur in patients with PRES, and there is a possible interrelationship between PRES and vasospasm.

An excessive maternal systemic inflammatory response involving leukocyte activation and cytokine production has been implicated in the pathogenesis of preeclampsia-eclampsia [11]. Vasogenic edema, if significant, causes hypoxia and ischemic injury by elevating tissue pressure and impairing microcirculation [4]. Both ischemia and hypoxia also induce an inflammatory response. This response activates both leukocytes and platelets, with a subsequent release of inflammatory cytokines such as interleukin (IL)-1 β , IL-6 and tumor necrosis factor- α . All these cause cerebral vasospasm [12]. Thus, the hyperinflammatory state in preeclampsia-eclampsia may cause cerebral vasospasm that is followed by vasogenic edema in preeclampsia-eclampsia. Alternatively, the potent vasoconstrictors released by endothelial cell damage could mediate cerebral vasospasm [7]. Further studies are necessary to fully understand the pathophysiological features, causes, and interrelationships of cerebral vasospasm and PRES in preeclampsia-eclampsia.

Acknowledgments

This study was supported in part by a Grant-in-Aid for Scientific Research from the Japanese Society for the Promotion of Science (18591804).

References

- Hinchey J, Chaves C, Appignani B, et al: A reversible posterior leukoencephalopathy syndrome. *N Engl J Med* 1996;334:494–500.
- Mukherjee P, McKinstry RC: Reversible posterior leukoencephalopathy syndrome: evaluation with diffusion-tensor MR imaging. *Radiology* 2001;219:756–765.
- Singhal AB: Postpartum angiopathy with reversible posterior leukoencephalopathy. *Arch Neurol* 2004;61:411–416.
- Koch S, Rabinstein A, Falcone S, et al: Diffusion-weighted imaging shows cytotoxic and vasogenic edema in eclampsia. *AJNR Am J Neuroradiol* 2001;22:1068–1070.
- Dodick DW, Eross EJ, Drazkowski JF, et al: Thunderclap headache associated with reversible vasospasm and posterior leukoencephalopathy syndrome. *Cephalalgia* 2003;23:994–997.
- Oehm E, Hetzel A, Els T, et al: Cerebral hemodynamics and autoregulation in reversible posterior leukoencephalopathy syndrome caused by pre-/eclampsia. *Cerebrovasc Dis* 2006;22:204–208.
- Kinoshita T, Moritani T, Shrier DA, et al: Diffusion-weighted MR imaging of posterior reversible leukoencephalopathy syndrome: a pictorial essay. *Clin Imaging* 2003;27:307–315.
- Ito T, Sakai T, Inagawa S, et al: MR angiography of cerebral vasospasm in preeclampsia. *AJNR Am J Neuroradiol* 1995;16:1344–1346.
- Henderson RD, Rajah T, Nicol AJ, et al: Posterior leukoencephalopathy following intrathecal chemotherapy with MRA-documented vasospasm. *Neurology* 2003;60:326–328.
- Hashimoto H, Kuriyama Y, Naritomi H, et al: Serial assessments of middle cerebral artery flow velocity with transcranial Doppler sonography in the recovery stage of eclampsia. A case report. *Angiology* 1997;48:355–358.
- Redman CWG, Sargent IL: Pre-eclampsia, the placenta and the maternal systemic inflammatory responses – a review. *Placenta* 2003;24:S21–S27.
- Fassbender K, Hodapp B, Rossol S, et al: Inflammatory cytokines in subarachnoid haemorrhage: association with abnormal blood flow velocities in basal cerebral arteries. *J Neurol Neurosurg Psychiatry* 2001;70:534–537.

Kiyomi Tsukimori, MD, PhD
Department of Obstetrics and Gynecology
Graduate School of Medical Sciences, Kyushu University
Maidashi 3-1-1, Higashi-ku
Fukuoka 812-8582 (Japan)
Tel. +81 92 642 5394, Fax +81 92 642 5414
E-Mail tsuki@med.kyushu-u.ac.jp

Cerebrovasc Dis 2008;25:380–381
DOI: [10.1159/000120690](https://doi.org/10.1159/000120690)

A Case of Presumed Granulomatous Carotid Stenosis

P. Dassan^a, M.M. Brown^a, C. Bishop^b, I. Hopkins^c, D.J. Werring^a

^aStroke Research Group, UCL Institute of Neurology, National Hospital of Neurology and Neurosurgery, and Departments of ^bVascular Surgery and ^cHistopathology, University College Hospital, London, UK

A 75-year-old right-handed Caucasian gentleman presented with sudden left arm weakness and dysarthria on rising from bed in the middle of the night. These symptoms resolved over 1 month. Twelve months later he was admitted to hospital for an open cholecystectomy. Two days post-operatively, he had a further attack of sudden left arm weakness lasting 1 month. Thereafter his family reported that he had memory problems and difficulty dressing himself, which subsequently improved. When seen 11 months later on our unit, neurological examination was normal. Vascular examination revealed a right carotid bruit. Fasting glucose, cholesterol, ESR, autoimmune screen, treponemal serology and chest X-ray were normal or negative. CT of the brain showed an area of

Fig. 1. a Magnetic resonance angiogram showing right common carotid stenosis (arrow). **b** Histology of lymph node biopsy (stained with haematoxylin and eosin) showing epithelioid granuloma (arrow).

low attenuation in the right parietal region consistent with mature infarction. Carotid Doppler ultrasound revealed >70% stenosis of the right common carotid artery. Magnetic resonance angiography with contrast confirmed focal stenosis of the right common carotid artery (fig. 1a). In view of the unusual location and morphology of the stenosis, diagnostic digital subtraction angiography was performed, which confirmed these aspects. The patient was taken to theatre with the plan to perform a right carotid endarterectomy.

At surgery a very inflamed right carotid artery was noted, as evidenced by punctuate haemorrhages in the adventitia and a thickened carotid artery which appeared to be stuck to the surrounding tissues. These changes were especially prominent at the bifurcation. A large number of lymph nodes were also seen in the carotid triangle, and the planned endarterectomy was abandoned. Post-operatively, a whole-body PET scan showed no evidence of a large-vessel vasculitis. Biopsy of one of the cervical lymph nodes revealed epithelioid granuloma with caseation (fig. 1b) typical of tuberculosis (TB). The patient was immediately started on empirical treatment with anti-TB medications and the carotid stenosis was treated by stenting without complication. Subsequently a polymerase chain reaction for TB has been performed and is negative. At his last follow-up appointment, 2 years later, the patient remains well and has had no further episodes.

Discussion

Atherosclerosis is the commonest cause of carotid stenosis, most frequently affecting the bifurcation of the artery. Atherosclerosis of the common carotid artery below the bifurcation only rarely causes symptomatic stenosis. This case emphasises that if symptomatic stenosis is found in the common carotid artery, then other causes of arterial disease must be considered. These include post-radiation stenosis (of which there was no history in this case) and large-vessel vasculitides including Takayasu arteritis or giant cell arteritis. Granulomatous disease is a very rare cause of vascular disease but an important differential diagnosis to consider in

a patient with atypical carotid stenosis as the management is different [1–3]. To the best of our knowledge this is the first reported case of presumed granulomatous symptomatic common carotid artery stenosis. The presence of caseating granulomata is highly suggestive of TB, and even though the polymerase chain reaction for TB was negative, this does not exclude the diagnosis as the sensitivity of the test is variable and may be lower in extrapulmonary TB. The good clinical recovery with empirical anti-TB treatment provides further support for the diagnosis.

References

- 1 Kolawole TM, Onadeko BO: Vascular lesions in tuberculosis. *Diagn Imaging* 1980;49:303–310.
- 2 Luswazi DC, Robbs JV: False aneurysm of the common carotid due to tuberculosis: a case report. *S Afr Med J* 1976;50:2076–2077.
- 3 Neugebauer MK, Hoyt TW: Carotid artery aneurysm of granulomatous origin. *Am J Surg* 1975;130:362–365.

Dr. Pooja Dassan
Clinical Research Fellow, UCL Institute of Neurology
Box 6, National Hospital of Neurology and Neurosurgery
Queen Square
London WC1N 3BG (UK)
Tel. +44 207 837 3611, ext. 4113, Fax +44 207 837 9632
E-Mail p.dassan@ion.ucl.ac.uk