bruits. A soft ejection systolic murmur was present over the left sternal border. Routine blood tests were normal. A fasting cholesterol was slightly raised at 8.6 mm/l. ECG and echocardiogram were normal. CT scan showed small low density lesions in the right frontal and parietal lobes consistent with small infarcts. Digital subtraction angiography was technically unsuccessful and four vessel angiography was done under general anaesthetic which showed severe irregular narrowing of the right internal carotid artery with a minimum diameter of less than 1 mm.

There was severe hypoplasia of the proximal segment of the left anterior cerebral artery, the distal parts of both anterior cerebral arteries being supplied from the right carotid. There was atheroma without stenosis of the proximal left internal carotid. The vertebral arteries were normal.

Shortly following admission the patient complained of sudden weakness in the left hand and re-examination at that time showed a global paralysis of the left hand including wrist flexion and extension; strength gradually returned to its previous level after a few hours. He also had an episode of weakness and numbness in the leg which returned to normal after twenty-four hours. Carotid endarterectomy was performed. He made a good recovery and has had no further episodes of transient neurological deficit since the time of operation ten months ago even though he continued to have a mild sensory deficit in the left hand.

The pattern of our patient's neurological deficit was initially interpreted as being due to ischaemia in the territory of the vertebrobasilar system though the absence of other features of brainstem disturbance was unusual. There was no evidence on clinical examination or on an echodoppler scan of a cardiac source of emboli. It seemed probable from the angiographic findings that the severe stenotic lesion of the right internal carotid artery was responsible for his symptoms. Since the severe hypoplasia of the proximal segment of the left anterior cerebral artery left both anterior cerebral arteries dependent on the right internal carotid artery, emboli from the latter could clearly cause ischaemia in the territory of the midle cerebral artery on the right and of both anterior cerebral arteries and would explain the episodes of transient weakness affecting both lower limbs. The critical nature of the stenosis also raised the possibility of the attacks being "haemodynamic" in origin though symptoms suggestive of generalised hypoperfusion were only recorded in one of the attacks.

Severe hypoplasia of the proximal segment of the anterior cerebral artery (A1 segment) has an incidence of 4%. Embolisation from the internal carotid artery into the anterior cerebral artery is also rare with an incidence of about 7%; the remaining 93% occurring in the middle cerebral artery and its branches. However, in situations when multiple emboli have been thrown off, the anterior cerebral artery is affected in about 50% even though experiments using spherical steel emboli showed that the first embolus was never lodged in the anterior cerebral artery and it was concluded that only after occlusion of the middle cerebral artery or its branches is flow redirected towards the anterior cerebral artery.

The latter reasoning can partly explain the disproportionately high incidence of transient ischaemic attacks affecting the anterior cerebral circulation (approximately 40%) in our patient since there was both clinical and radiological evidence of infarction in the right middle cerebral artery territory. One can also postulate that the common origin of both anterior cerebral arteries had in some way biased flow towards that circulation possibly due to the anatomical variation but also possibly related to an increase in blood flow secondary to both fontal lobes being supplied from the same side.

Absence of immunoreactive interferon-α in CSF from patients with multiple sclerosis

Sir; It has been suggested that multiple sclerosis may be due to an infectious agent (a virus) with occurrence only in people immunogenetically at risk. The interferons are antiviral proteins which also modulate immune systems and cell growth. Interest has arisen in the possible therapeutic role of interferons in multiple sclerosis, but a number of studies of interferons in multiple sclerosis by biological assay have produced conflicting results. Thus some laboratories reported reduced capability of lymphocytes from multiple sclerosis patients to produce interferons in vitro, whereas others failed to confirm these findings. Early reports also suggested that measurable quantities of interferons were present in serum and CSF of some multiple sclerosis patients, but a more recent study failed to detect them in 31 patients with multiple sclerosis.

We have used a recently developed highly sensitive immunoaassay for immunoreactive interferon-α to study patients with multiple sclerosis. This is a two-site immunoradiometric assay (IRMA) ('Sucrosep', Boots Celltech Diagnostics Ltd., Slough) based on a monoclonal antibody (Yok's19) to interferon-α. The assay can detect as little as 0.3 Hu interferon-α/ml serum/CSF and shows no cross reactivity to human interferons β and γ. We measured interferon-α in CSF from 15 patients with multiple sclerosis, 10 female, 5 male, mean age 42.3 years. All had definite multiple sclerosis according to recent diagnostic criteria. Twelve had relapsing and remitting and three chronic progressive disease. In the former group lumbar puncture was performed in the early stages of an acute relapse, and prior to the commencement of ACTH treatment. In 11 patients serum was taken at this time of lumbar puncture and assayed for interferon-α. All specimens were stored at −20°C prior to assay, conditions under which interferon-α is stable. We detected no interferon-α by IRMA in any of the specimens of CSF or serum.

Previous discrepancies in studies of interferons in multiple sclerosis probably relate to technical differences in the bioassays used. These assays depend on cell culture and virus challenge and are thus very prone to biological variability. Our results, using a highly specific, sensitive and reproducible immunoaassay confirm the findings by bioassay of Salonen that interferon-α is not detectable in the serum or CSF of multiple sclerosis patients.

References


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recently been detected in the serum of patients with acquired immuno-deficiency syndrome,\textsuperscript{11} systemic lupus erythematosus,\textsuperscript{12} rheumatoid arthritis\textsuperscript{13} and asbestosis\textsuperscript{14} and it has been suggested that persistent lymphocyte stimulation due to an immune disorder may lead to high levels of interferon in the serum. Our study, using a reliable immunological assay, suggests that elevated CSF interferon-\(\alpha\) concentrations are not present in multiple sclerosis. However, further studies using IRMAs for interferon-\(\gamma\) will allow further insight into the possible role of interferons in the aetiology of multiple sclerosis.\textsuperscript{15}

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