
The following is in response:

To the Editor:

We thank Doctors Watson and Dietrich for their letter. They have made several useful points, which may further assist in understanding the disparity between animal stroke models and the human situation.

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Gonadotrophins, Livedo Reticularis, and Strokes

To the Editor:

Sneddon's syndrome is an entity characterized by idiopathic livedo reticularis and recurrent strokes. Its pathogenesis is unknown, and the part played by antiphospholipid antibodies remains controversial.1 Recently Rautenberg and coworkers2 reported 16 patients (15 female) with Sneddon's syndrome, all of them with negative tests for anticardiolipin antibodies. Bruyn et al., reviewing the literature, found that many patients were cigarette smokers, oral contraceptive users, or both. Thus, it is likely that multiple factors interact, at least in some cases, to produce this peculiar dermal and cerebral vasculopathy. We wish to report the case of a woman with Sneddon's syndrome whose first stroke was probably triggered by gonadotrophin therapy.

This 27-year-old patient had a past history of migraine and heavy tobacco use. Livedo reticularis over all four limbs and Raynaud's phenomenon were present since puberty. Anovulation was the cause of infertility and a first course of gonadotrophins was administered in November 1980. The patient received 21 intramuscular injections of human menopausal gonadotrophins and then 5,000 IU human chorionic gonadotropin (HCG). The day following the last injection she complained of pain in the right foot, which was cold and livid. The symptoms resolved spontaneously in 2 hours. Three days later, she developed the sudden onset of expressive aphasia and right hemiparesis. Brain computed tomography was consistent with left middle cerebral artery territory infarction. Cardiovascular examination and routine laboratory tests were unremarkable. Chest X-ray, echocardiography, electrocardiographic monitoring, right cardiac angiography, and bilateral carotid Doppler examination were normal. Platelet hyperaggregability to ADP was noted. Neurologic symptoms resolved within the following 6 months.

We maintained the patient on anticoagulant therapy until June 1989, and then changed to aspirin (250 mg daily) and diprydamole. In November 1989, she presented with left facial and arm weakness that resolved over 48 hours. We also noted a diffuse pyramidal syndrome and a slight slowing of mentation, but without cognitive impairment. Normal or negative values were obtained for ESR, full blood counts, serum glucose, renal and liver function tests, rheumatoid factor, cryoglobulins, circulating immune complexes, antinuclear antibody (ANA), IFA anti-DNA, C3, C4, CH50, VDRL, TPHA, protein C and S, antithrombin III, and prothrombin time. On repeated examinations using different reagents, partial thromboplastin time was normal, and the search for anticardiolipin antibodies by ELISA was twice negative. Biopsy of the livedo reticularis revealed a small inflammatory infiltrate in the dermis, but no vasculitis, and immune-fluorescence studies were negative. The patient refused permission for cerebral angiography. Magnetic resonance imaging of the brain using T2-weighted sequences showed multiple white matter hyperintensities in both centrum semi-ovale.

Although our patient had other vascular risk factors (i.e., migraine and tobacco use), we believe that because of the close temporal relationship, gonadotrophin administration triggered the first stroke. Strokes related to gonadotrophin therapy are extremely rare although its use is widespread. Previously reported cases have been linked with the ovarian oversimulation syndrome, which causes a rapid shift in body fluid balance, abdominal pain, ascites, pleural effusions, hemocoagulation, and blood hypercoagulability.4,5 In this case, although clinical features of ovarian oversimulation were absent, it is possible that gonadotrophin-induced hyperestrogenism favored thrombosis via a transient increase in clotting factors (VII, IX, XI) or in platelet aggregability.6 This observation also suggests that women with livedo reticularis could be at risk for stroke when treated with gonadotrophins and should probably have a careful dermatological examination before their prescription.

Sneddon's syndrome is known to show a marked female preponderance (sex ratio 3:1).2 Moreover, long-lasting periods of hormonal changes, such as pregnancy and oral contraceptive use, have both been linked to a possible increased risk of stroke in women with Sneddon's syndrome.7 Thus, we should pay attention to the putative involvement of hormonal factors in the pathogenesis of this cerebral–dermal vasculopathy, particularly when the presence of anticardiolipin antibodies cannot be demonstrated.

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References
Imaging Techniques in Suspected Internal Carotid Artery Dissection

To the Editor:

The case report by Panisset and Eidelman brought my attention to a consequence of internal carotid artery dissection that I have not previously encountered in my practice, namely, multiple lower cranial nerve pareses. While I appreciate their enlightening me on that process, I must object to the interpretations of normal computed tomography (CT) scans in two of the patients.

Figure 5 demonstrates the bull’s-eye sign of the narrowed, enhancing true lumen surrounded by hypodense intramural hematoma. It stands out as a virtual negative in contrast to the magnetic resonance image shown on the opposite page. Figure 1 demonstrates a round, probably vascular structure just medial to the right styloid process, which is in the expected location of the right internal carotid artery, but larger than would be expected. The left internal carotid artery is visible on the opposite side and normal in size. Contrast-enhancement effect seems somewhat suboptimal and certainly insufficient to distinguish enhancing true lumen from intramural hematoma. Nevertheless, the dilated structure in the expected position of the internal carotid artery is highly suspicious for carotid dissection.

A diagnosis of internal carotid artery dissection can be made on CT, and we have done so multiple times in the past. We are constantly vigilant to examine the cervical internal carotid artery in

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