LETTER TO THE EDITOR

DEAR SIR:

CEREBRAL THROMBOPHLEBITIS IN PUERPERIUM

In reference to Dr. Famewo's article "Headache Following Epidural Anaesthesia for Obstetrical Delivery – A Case Report" (Can. Anaesth. Soc. J. 22: 370 (1975)), I would like to supplement it by placing more emphasis on the patient's underlying pathology which was responsible for her initial complaints and eventually led to her demise, namely, cerebral thrombosis.

We are told that her pregnancy and delivery were uneventful. She had no evidence of pre-eclampsia and sickle cell and VDRL tests were negative. Cerebral angiography prior to death revealed marked narrowing of the supraclenoid portion of both internal carotids and their anterior and middle cerebral branches. This was interpreted as spasm. There was moderate hydrocephalus but no shift or aneurysms. Post-mortem examination disclosed positive CNS findings. The brain was grossly swollen with intracerebral haemorrhage ruptured into the ventricular system.

Microscopy revealed thrombosis of a large right cerebral vein (Figure 1). The venous wall was focally infiltrated with polymorphonuclear leukocytes. Inflammatory reaction, although not so intense, was also present in a patchy fashion in the adjacent subarachnoid space. In the paraventricular area inflammatory reaction was present even in the wall of veins in which there was no thrombus (Figure 2). At the site of lenticular nuclear haemorrhage, there was a slight leukocytic infiltration and proliferation of macrophages. There were no affected vessels in the immediate vicinity of the lesion. Special stains for bacteria, fungi, myobacteria and treponema failed to demonstrate any organisms in the inflammatory infiltrate. Acute neuronal ischaemic changes were widespread in frontal and temporal lobes.

It is very interesting that the degree of phlebitis seemed to be out of proportion to thrombosis. There was no evidence of thrombophlebitis in the spinal cord or elsewhere.

This patient's presentation was by no means classical. Kendell¹ reported a series of 11 patients all of whom were treated upon making the diagnosis. Although in none of his series was the condition fatal, in all there seemed little doubt of the diagnosis on clinical grounds alone. Carroll² on the other hand, presents a more realistic picture. He reports the incidence of cerebral thrombophlebitis in pregnancy and puerperium as one in 1,000 to 3,000 pregnancies, where the majority of cases presented with paresis, headache, or convulsions and with a mortality rate of 33 per cent.

A recent paper by Dindar, *et al.*³ draws attention to the fact that fatal intracranial venous thrombosis was related to a short course of oral contraceptive agent.

Mullock and Scolnick⁴ presented three cases of post partum cerebral thrombophlebitis, with no fatality but significant morbidity. A recent report from India⁵ indicates that this condition is somewhat more frequent in that part of the world. Although a majority of their patients deliver at home, the mortality of this condition is the same as in Western countries. In their review of eight cases they found superficial longitudinal sinus (S.L.S.) with one or two transverse sinuses to be

FIGURE 1. Cortical vein with large thrombus, underlying cerebral infarct ($\times 100$, H.P.S. stain).

involved in six, and the vein of Galen was thrombosed with the straight sinus and one or both transverse sinuses in two cases. The oldest thrombi were found in S.L.S. and the most recent ones were in the superficial cerebral veins. In the case presented S.L.S., transverse sinuses and vein of Galen were uninvolved. As in the

FIGURE 2. Vein wall with inflammatory infiltrate ($\times 400$, H.P.S. stain).

present case, there was no infection to be implicated as an aetiological agent in their study.

Although there are a few theories on aetiology of this phenomenon in pregnancy and the puerperium, the precise mechanism of its generation is not settled.

It is well recognized that there is a hypercoagulable state in pregnancy and the puerperium. However, if the hypercoagulable state of blood alone were responsible, cases should be more frequent and uniform throughout the world.

Infection cannot be implicated in this case although it is recognized as a causative factor in cerebral thrombophlebitis. Dehydration is one of the factors that may play a role.⁵

At present supportive measures such as prevention of convulsions are recommended but use of anticoagulants is perhaps too hazardous and not recommended.²

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