stay in hospital after the results are back from the laboratory, to re-establish some meaning in her life. I think that I can help her to do this by seeing her regularly and establishing a friendly supportive relationship with her. Perhaps we can go out and engage in some normal activities together while she is in the ward, and so diminish the stress in her life. I need to help Jane to feel worth while and find some mechanisms she can use to cope with the unhappy situation at home. My brief meeting with her gives me hope that we can achieve this. She has insight, and some inner strength to work on. I can help her to feel that she can make changes, and this support may continue after her discharge from hospital for as long as she needs.

Mr Clegg has been in and out of hospital several times in the past few months. He had not been able to cope with the change in his life after an industrial accident. He will never go back to being a motor mechanic with a damaged arm and wrist. My colleague has spent time working with him and his family, and he is just beginning to adjust. Other family members have had a lot of problems, but progress is being made. Suddenly, the colleague is told that Mr Clegg has been referred to a psychiatrist. Naturally, she wonders how

the doctor could have taken this step without consulting her. Does he think she is not skilled in working with families? What about team work?

Has this happened because the consultant believes in the stereotype of the social worker as someone who is concerned only with solving practical problems and applying for sickness benefit? If so, we should try and change his image of the social worker and point out that, in this case, a trusting relationship had been established with Mr Clegg and his family. Mr Clegg was bewildered, and felt abandoned by the social worker when he was referred elsewhere.

Differences of opinion are bound to arise from time to time. On the whole, I think that we work well with doctors. So much of our work is similar. We both have problems of priorities. We both accept responsibilities for other people's decisions. We both hold the same value about treating the whole patient. There are mutual advantages in this partnership, which in some cases have not yet been fully accepted. Perhaps the social work profession made a mistake in taking the medical profession as its model. Part of the misapprehension and confusion about the role and function of the social worker is

due to the paucity of input to the medical curriculum.

Social workers undergo a four-year degree training which includes interpersonal counselling skills, group dynamics, and community organisation.

With no visible tools of the trade—no stethoscope, plaster room or diet sheetthe social worker may seem to lack expertise. The basic resource is the interview, the heart of the social work process, and the establishment of a significant relationship with the patient. Talking and counselling are not nearly so central to other professional-client encounters as they are to social work. We usually expect a doctor to examine and prescribe, a teacher to instruct, a lawyer to read documents and advise. The goal of the interview is to define the problem, and determine the available alternatives for the solution. These may include the need for further talk on the emotional aspects in future interviews, or the choice and mobilisation of practical resources. The interview and the social worker's knowledge of the community will frequently help the patient to mobilise the resources within himself to cope with life. Thus, doctors and social workers together bring their different skills to cure and care.

# Case Reports

# Puerperal superior sagittal sinus thrombosis

Victor J. Ojeda, John M. N. Hilton and Edward G. Stewart-Wynne

ABSTRACT: A 31-year-old woman became unconscious shortly after delivery of a stillborn fetus in the thirty-sixth week of gestation. Tonic-clonic seizures and deepening coma occurred soon after delivery. Despite intensive supportive therapy, the patient died three days after delivery. A superior sagittal sinus thrombosis and extensive bilateral cerebral infarction were found on necropsy.

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NON-INFECTIVE cerebral sinus thrombosis is a rare complication of pregnancy or the puerperium.<sup>1-5</sup> The clinical signs include paresis (45%), headaches (34%),

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convulsions (30%), speech disturbances (24%), and confusion/drowsiness (17%). The mortality rate is about 30%. 2.6 In a large review, toxaemia of pregnancy was present in up to 10% of the cases, 2 but other authors exclude toxaemic patients from their series.6

The incidence of this disease varies in different countries; Canadian workers found three post-partum cases of cerebral sinus thrombosis in 20 years, while in Mexico, 13 cases occurred in two years,

and from India, a report cites 138 cases.<sup>6</sup> Two maternal mortality studies from Australia contained details of 72 maternal deaths, none of which was related to sinus thrombosis;<sup>8.9</sup> therefore, it seemed pertinent to us to report this case.

### Clinical record

A 31-year-old woman was admitted to a regional hospital during the thirty-sixth week of her fourth pregnancy to control persistent nausea and vomiting; this settled rapidly after mild sedation. Her pregnancy was supervised from the tenth week of gestation, and no evidence of toxaemia was detected. Two days after admission to hospital, she spontaneously delivered a stillborn male fetus. A few hours after delivery, the patient became hypotensive. A rapid infusion of saline led to prompt recovery but she was found unconscious shortly thereafter. Three tonic-clonic seizures occured, and the patient was transferred to the Royal Perth Hospital.

On arrival, she was comatose, and showed no responses to painful stimuli. The optic discs were normal. Her pupils each measured 3 mm in diameter and reacted sluggishly to light; the eyes were slightly convergent. Oculocephalic reflexes were absent while oculovestibular reflexes were initially present. Her limbs were flaccid, the tendon reflexes were brisk, but the plantar responses were absent. Focal seizures of the left hand and the left side of the face occurred. The patient's condition rapidly deteriorated; renal failure and deepening coma developed.

Computerised tomography of the head, without intravenous administration of contrast medium, showed small lateral ventricles, a central third ventricle, and patchy areas of high and low attenuation which suggested haemorrhagic infarction. The cerebrospinal-fluid (CSF) pressure was 1.47 kPa (15 cm  $H_2O$ ); the CSF protein level was elevated at 3.0 g/L (normal 0.15 g/L to 0.45 g/L); and microscopy showed  $8.0 \times 10^9$ /L red blood cells and  $2 \times 10^9$ /L lymphocytes. Cultures grew no organisms.

The patient died on the third day after delivery.

# **Necropsy findings**

A complete necropsy was performed. No toxaemic lesions were found either macroscopically or microscopically. The superior sagittal sinus was completely



FIGURE 1: Coronal section of the brain at the level of the mamillary bodies. Extensive haemorrhagic necrosis is present in the parietal lobes. (The cleft is artefactual.)

occluded by fresh thrombus. The brain weighed 1405 g, and was examined after fixation in formalin. The cerebral hemispheres were symmetrical. Recent subarachnoid haemorrhage over both frontal and parietal lobes was present. No superficial cortical vein thrombosis was noted. The arteries of the circle of Willis were normal. Coronal sections of the cerebral hemispheres at 1-cm intervals demonstrated extensive, almost symmetrical, haemorrhagic necrosis affecting the cortex and subjacent white matter of the posterior part of the frontal lobes, the parietal lobes, and the anterior and superior portion of the right occipital lobe (Figure 1).

The ventricular system was collapsed. The brain stem was normal. Grooving and necrosis of the tonsils were noted in the cerebellum.

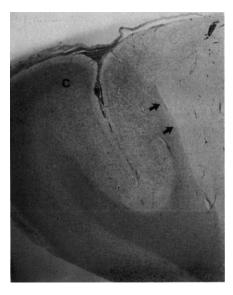


FIGURE 2: Section of the parietal lobe demonstrating the edge of the infarct (arrows). (Weil's stain for myelin; magnification  $\times$  14.5.) C = cortex.

Sections of the parietal cortex (Figure 2) confirmed extensive and recent necrosis. The meningeal veins (Figure 3) contained fresh thrombi. The arteries were normal. All the other organs were histologically normal, although most renal glomeruli showed mesangial thickening.



FIGURE 3: Meningeal vessels. The arteries (a) are patent while the veins (arrows) are filled with fresh thrombi. (Haematoxylin and eosin stain; magnification × 102.)

The diagnoses listed in the necropsy report were "multiple haemorrhagic cerebral infarcts secondary to superior sagittal sinus thrombosis".

# Discussion

The clinical and pathological features described are classic features of the effect of superior sagittal sinus thrombosis, the extent of the cerebral damage being incompatible with life. Carroll *et al.*, analysing 181 cases (177 from the literature and four new cases), disclosed an over-all mortality of 32% and permanent neurological sequelae in 19% of the survivors.<sup>2</sup>

Theories to explain the pathogenesis of this disease include retrograde venous embolisation, hypercoagulation, and endothelial damage or proliferation. None of these theories has been confirmed, and the pathogenesis of non-infective sinus thrombosis remains unknown.<sup>7,10</sup>

The geographical variation in incidence is striking; numerous cases have been reported from developing countries such as India, and Mexico, while cerebral sinus thrombosis is not even mentioned in maternal mortality reports from two Australian States. 9.9

Intracranial venous or sinus thrombosis occurring in young women taking oral contraceptives has been reported;<sup>7,11,12</sup> so far, the relationship is not statistically significant.

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