Brain tumour and pregnancy

JOST J. MICHELSSEN AND PAUL F. J. NEW

From the Neurosurgical Service and Department of Radiology of the Massachusetts General Hospital and Harvard Medical School, Harvard, U.S.A.

Some 35 years ago, in Brouwer’s clinic, Oljenick, deVet, and one of the authors (J.J.M.) observed a female patient with intermittent disturbances of the visual field due to compression of the optic chiasm by a meningioma. Her history indicated that the visual difficulties occurred toward the end of each of her frequent pregnancies—she had seven—and disappeared again after each delivery. They became permanent shortly before her hospitalization 10 years after their onset.

Similar clinical observations have been reported since that time (Hagedoorn, 1937; Rand and Audler, 1950; Weyand, MacCarty, and Wilson, 1951; Bickerstaff, Small, and Guest, 1958; Tarnow, 1960), dealing with the problem of cerebral tumours, especially meningiomas, in pregnancy. There is agreement that meningiomas and neurinomas enlarge during pregnancy. Water retention in the body and increased fluid content in the tumour are thought to be the cause. Other observers believe that there is acceleration of growth, presumably as a result of hormonal changes.

CASE REPORT

The patient (R.K.), whose case is presented in this report, noted the onset of generalized epileptic seizures at the age of 26, during the seventh month of her first (and only other) pregnancy. Phenytoin (Dilantin) controlled these seizures for years, but had lost its effect when she was admitted to the Massachusetts General Hospital at the age of 34, in the sixth month of her second pregnancy. These seizures were frequent and focal in character, starting with motions of the fourth and fifth fingers of the right hand, spreading to the index finger, elbow, shoulder, and face. Neurological findings were minimal. An electroencephalogram disclosed poorly organized alpha waves and slowing over the left cerebral hemisphere. A meningioma in the superior frontal areas was suspected. The drugs regimen was changed, but the seizures remained uncontrolled. A mild right-sided motor deficit and dysphasia developed as post-ictal phenomena.

Radiography of the skull demonstrated erosion and decreased density of the cortex of the floor and dorsum of the sella turcica, and slight expansion of the pituitary fossa was present, indicating chronic increase in intracranial pressure (Fig. 1), although spinal fluid pressure at the time of lumbar puncture was 172 mm of cerebrospinal fluid. Left common carotid angiography revealed a medial superior frontal mass. No tumour vessels were identified, but a lateral serial study was inadequate, due to technical problems. The pericallosal artery was displaced 12 mm to the right of midline and the internal cerebral vein showed no definite lateral displacement (Fig. 2a, b).

Both the patient and her husband were anxious to have another child. It was therefore decided to defer further diagnostic procedures and operation until after delivery, at the earliest possible time, by Caesarean section, provided that there was no further deterioration in her neurological status. On readmission for surgery seven weeks after delivery of a healthy baby, the patient reported that her seizures had been under control (with diphenylhydantoin (Dilantin), primidone (Mysoline), and mephobarbital (Mebaral)). She complained of bifrontal headaches relieved by Bufferin, an aspirin compound.

FIG. 1. Lateral view of the sella, showing considerable decrease in density of cortex of the pituitary fossa and dorsum sellae, with some expansion of the pituitary fossa.

1Read in part before the annual meeting of the Scandinavian Neurosurgical Society, Århus, Denmark, 30 August 1968.
superior frontal region. The mass was clearly smaller than at the time of the previous angiogram. There was evidence of a few irregular pathological vessels in the medial superior frontal area and indication of a very faint capillary staining in the mass. This staining did not persist into the venous phase and the intrinsic tumour

Neurological examination was grossly normal except for some uncertainty of gait and slowness in response, both compatible with drug toxicity.

Cerebral isotope scans were not helpful. Bilateral carotid angiography was undertaken. At this time, the pericallosal artery was displaced to the right of the midline for a distance of 9 to 10 mm and the displacement was over a shorter distance than previously (Fig. 3). There was also less stretching of the regional arterial branches in the

FIG. 2a. Left common carotid angiogram, in frontal projection. Initial arterial phase. The bowing and displacement of the pericallosal artery extends 12 mm from the midline (marked by a vertical line). Stretching of branches of the cingulate artery in the superior frontal area is demonstrated.

FIG. 2b. Frontal projection of the early venous phase of the angiogram shown in 2a. No definite lateral displacement of the internal cerebral vein is identified.

FIG. 3. Left common carotid angiogram obtained after delivery. The displacement of the pericallosal artery is now less than before (9 to 10 mm), and the stretching and displacement involves a shorter segment of the artery. There is also evidence of less marked stretching of the frontal branches of the cingulate artery.

FIG. 4. Lateral arterial phase of the second angiogram. Vascular dislocation and stretching secondary to a superior frontal mass is shown. Only a few tumour vessels can be seen. There is very slight diffuse capillary staining of the mass, which appears to measure some 4 cm in maximum diameter.
vascularity was thought to be insufficient for satisfactory evaluation of the histology of the mass (Fig. 4).

In spite of the lack of persistence of the capillary staining and of external carotid supply to the tumour, the obvious decrease in size of the mass since pregnancy suggested meningioma.

A left frontal craniectomy was performed, with partial frontal lobectomy, for removal of an astrocytoma, Grade I.

Follow-up studies on this patient revealed that considerable repair of the sella erosion and expansion occurred after surgery, and angiography five months later revealed no evidence of recurrence of the neoplasm. Management of her focal seizures remained a problem.

DISCUSSION

This seems to be the first instance in which both the clinical course and angiographic abnormalities of an intrinsic brain tumour have been recorded during and after gestation. The development of symptoms—in this case seizures—towards the later stages of pregnancy is in keeping with clinical observations in slowly growing extrinsic brain tumours, such as meningiomas or neurinomas, in the vicinity of cranial nerves. Reduction of fluid content within the tumour or of oedema in adjacent brain, after termination of the pregnancy, appears to be a plausible explanation for the appreciable reduction in total volume of the frontal lobe mass demonstrated by angiography at an interval of almost four and a half months.

REFERENCES


Brain tumour and pregnancy.

J J Michelsen and P F New

*J Neurol Neurosurg Psychiatry* 1969 32: 305-307
doi: 10.1136/jnnp.32.4.305

Updated information and services can be found at:
http://jnnp.bmj.com/content/32/4/305.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/