Brief Reports

Recurrent bilateral reversible migrainous hemiparesis during pregnancy

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Migraine is a common disorder, occurring in an estimated 5% of the general population. There is often a strong family history among first-degree relatives. Females are slightly more susceptible than males, and there is a tendency for the attacks to occur during the period of so-called premenstrual tension associated with fluid retention; similarly, attacks may be initiated or exacerbated by oral contraceptive therapy. Often, however, the attacks cease during pregnancy itself.[1-4]

The pathogenesis of migraine has traditionally been explained by the occurrence of cranial artery constriction followed by vasodilation.[5] Classic migraine is often ushered in by disturbances of neurologic function, such as hemianopia and teichopsia, central blindness, hemiparesthetic disturbances and slight speech abnormality, that may proceed to transient hemiparesis. Indeed, if the arterial spasm is profound enough it may lead to cerebral infarction, with irreversible results.[6] The neurologic disturbance is usually (but not invariably) followed by hemicranial headache, nausea and vomiting.

I report a case in which a young woman presented unusually with migraine for the first time during her fourth pregnancy, exhibiting recurrent bilateral reversible hemiparesis and Parinaud's syndrome.

Case report

A 25-year-old right-handed white woman woke one morning with left facial weakness and numbness. Her speech was slurred. During the next 20 minutes a number of things happened. She went completely blind for about 1 minute, and when her sight returned she noticed double vision, with blurring of the images; this was followed by the gradual onset of a severe persistent right fronto-occipital headache, then resolution of the left facial weakness, and finally tingling and weakness of the left hand. She had mild photophobia and was nauseated. She remembered having wakened a number of times during the night with throbbing fronto-occipital headaches.

She had previously been well and had no history of headaches, visual disturbances, motor or sensory problems, asthma, eczema, hay fever or food allergy. Her mother had a history of migraine. The patient was in the 26th week of her fourth pregnancy, which had been completely normal. One of the previous pregnancies had ended in spontaneous abortion.

When admitted to hospital she was alert but in moderate distress. Her blood pressure was normal, and there were no apparent cardiovascular abnormalities; there were no carotid or cranial bruits. The height of the uterine fundus was commensurate with a 26-week pregnancy, and the fetal heart rate was normal. She was fully oriented, showed no signs of meningeal irritation and had normal cranial nerve function except for mild restriction of upward gaze, which was painful. Pupillary light reflexes were absent. There was possibly a reduction of the left temporal field to confrontation, and the grasping power of the left hand was slightly reduced. The tendon reflexes were brisk but equal, and both plantar responses were flexor.

Skull roentgenograms and an electroencephalogram were normal. The cerebrospinal fluid was clear, under normal pressure, free of cells and sterile; the protein concentration was normal. It was felt that she was having an attack of migrainous hemiparesis.

After 2 days of nausea, repeated profuse vomiting and severe intractable headaches she was transferred to the regional neurologic unit. Upward gaze was definitely restricted and associated with pain and dizziness, but there were no other focal neurologic abnormalities. Computer-assisted tomography with and without enhancement yielded normal scans.

It was agreed that she was probably suffering from a severe migraine attack with persistent Parinaud's syndrome (painful limitation of upward gaze with loss of pupillary light reflexes), and symptomatic management was continued. Over the next 2 weeks the restriction of upward gaze gradually lessened. Cerebral angiography was deferred in view of her pregnancy. Following discharge she remained well, and her pregnancy continued to progress normally.

One month after discharge she was readmitted because of the sudden onset of severe bilateral frontal headache associated with blurring of vision, leading to transient amaurosis and numbness and weakness of the right side of the face and right arm and leg. She denied teichopsia. She again had nausea and vomiting. She had obvious right upper motor neuron facial weakness, with markedly reduced power in the right arm and leg. The visual fields and fundi were normal, but again upward gaze was mildly limited and uncomfortable. The tendon reflexes were equal and the plantar responses flexor.

It was clear that she had suffered a second attack of migraine, with the return of Parinaud's syndrome and migrainous hemiparesis on the side opposite to that affected 6 weeks earlier. The neurologic abnormalities disappeared in about 48 hours, but the severe headache, nausea and vomiting were again persistent, though once they had resolved she remained well. A normal girl was born at term by uncomplicated spontaneous vaginal delivery. The mother remained well.

Discussion

The association of migraine with transient neurologic abnormalities (including hemiparesis) that are usually reversible is not uncommon. An association with Parinaud's syndrome is rare but has been reported.[7] The limitation of conju-

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gate upward gaze results from a disturbance in the superior colliculi and tegmentum of the upper midbrain near the posterior commissure, and often movements of convergence as well as the pupillary light reflexes are abolished. The migrainous vasospasm in this patient must, therefore, have involved the thalamoperforate branches of the posterior cerebral artery, which supply this area. In addition, spasm of the stem of the posterior cerebral artery may lead to crossed hemiplegia by occluding the peduncular arteries. On the second occasion, since the transient hemiparesis recurred on the opposite side, the vasospasm likely occurred in the territory of the opposite posterior cerebral artery. Both episodes may be considered variants of basilar artery migraine, which would also explain the transient amaurosis.

The cause of migraine may be multifactorial, and the role of allergic, pharmacologic, neurologic, vascular and humoral factors (such as kinins, histamine and 5-hydroxytryptamine) has been much discussed. Migraine may, indeed, coexist with other conditions (such as angina and Raynaud's disease), with a common humoral factor affecting more than one vascular bed.[8] The frequent cessation of migraine after the first trimester of pregnancy may be related to the influence of the high circulating level of endogenous estrogens on the threshold of contractility of vascular smooth muscle. It cannot be correlated with the sex of the fetus or the plasma progesterone level.[3] Among other things, in pregnancy the sensitivity of intracranial and extracranial vessels to α- and β-adrenergic agonists is reduced.[9] When the circulating estrogen level falls after delivery, attacks may be precipitated in those with a history of migraine, and migrainous hemiparesis during breast-feeding has been described.[10] It is uncommon for migraine to present for the first time during the third trimester, and I can find no recorded cases of migrainous hemiparesis during pregnancy.

References

1. EPSTEIN MT, HOCKADAY JM, HOCKADAY TDR: Migraine and reproductive hormones throughout the menstrual cycle. *Lancet* 1975; 1: 543–548
2. AMINOFF MJ: Neurological disorders and pregnancy. *Am J Obstet Gynecol* 1978; 132: 325–335
3. MASSEY EW: Migraine during pregnancy. *Obstet Gynecol Surv* 1977; 32: 693–696
4. SOMERVILLE BW: A study of migraine in pregnancy. *Neurology (Minneap)* 1972; 22: 824–828
5. WOLFF HG: *Headache and Other Pain*, 2nd ed, Oxford, New York, 1963: 269
6. Migrainous cerebral infarction (E). *Br Med J* 1977; 1: 532–533
7. CHAY OM, TONG HI, WONG PK: Parinaud's syndrome complicating migraine — a case report. *Singapore Med J* 1977; 18: 151–152
8. COFFMAN JD, COHEN RA: Vasospasm — ubiquitous? *N Engl J Med* 1981; 304: 780–782
9. HARDEBO JE, EDVINSSON L: Reduced sensitivity to alpha- and beta-adrenergic receptor agonists and intra- and extracranial vessels during pregnancy. Relevance to migraine. *Acta Neurol Scand [Suppl]* 1977; 56: 204–205
10. DOOLING EC, SWEENEY VP: Migrainous hemiplegia during breast-feeding. *Am J Obstet Gynecol* 1974; 118: 568–570

Spurious detection of a high serum imipramine level due to coating of Vacutainer stopper

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We report a case of atypical toxic psychosis due to lithium intake complicated by an erroneous laboratory report of extremely high serum imipramine concentrations.

Case report

Clinical observations

A 51-year-old man presented with a bipolar affective disorder and a history of suicide attempts. Because he had responded poorly to lithium, he was receiving carbamazepine (Tegretol), 200 mg once a day, and methotrimeprazine (Nozinan), 25 to 50 mg every 4 hours, by mouth. He had allegedly also received imipramine within the previous two months.

Two days before admission to hospital he had become increasingly confused and somnolent, and was having episodes of vomiting and diarrhea. At the time of admission he was drowsy and disoriented but was easily roused. There was no tremor or hyper-reflexia, his pupils were normal, and there were no focal neurologic abnormalities. His pulse rate was initially 110 beats/min but it declined to 90 beats/min within hours. His blood pressure was 110/80 mm Hg. He was incontinent of urine; the remainder of the physical examination was not contributory.

The serum lithium level at the time of admission was 4.9 mmol/l. Qualitative thin-layer chromatography showed methotrimeprazine but no imipramine in the urine. The serum sodium level was 137 mmol/l and the serum creatinine level 1.2 mg/dl (106 μmol/l). An electrocardiogram showed sinus tachycardia, the heart rate being 90 beats/min.

Treatment

The patient was treated with intravenously administered normal saline, 2 litres over 10 hours, and an oral sodium chloride supplement, 12.5 mmol/d. However, he showed only slight improvement, and by 24 to 48 hours after admission he was increasingly agitated and difficult to restrain, although tremor, hyper-reflexia, increased muscle tone, hypotension, vomiting, diarrhea and dysrhythmia were not present. Within 72 hours after admission the serum lithium level had decreased to 2.7 mmol/l. At this time the serum imipramine level (measured by gas chromatography with an external standard), from a sample drawn at the time of admission and sent to another laboratory, was reported as 12.9 (therapeutic level 0.15 to 0.25) mg/l, and the serum carbamazepine level measured from a sample drawn 2 days after admission was less than 1 (therapeutic level 6 to 8) mg/ml. Because the patient's agitation could not be controlled he was treated with divided doses of 17.5 mg of haloperidol intramuscularly over 10 hours, but with no effect. The patient then suffered a focal seizure of short duration. Because the seizure may have been caused

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