papaverine resulted in a fully rigid erection lasting almost 90 minutes. Computerised analyses of both dorsal and cavernosal arterial Doppler waveforms,[1] recorded during the tumescent phase, also proved normal. Thus there was no evidence of penile haemodynamic disturbance. Electrophysiological elicitation of the bulbocavernosus reflex, whose afferent and efferent limbs are composed of somatic pudendal nerves relayed through sacral segments 2, 3, and 4, showed a normal latency of 26 ms to the onset of response (mean (2 SD) value for our laboratory 28·1 (5·7) ms).[2] The mean (of three measurements) sensory perception threshold of the dorsal nerve of the penis was raised at 110 volts (mean (2 SD) value for our laboratory 64·2 (23·2) volts).[2]

Because a spontaneous improvement was already occurring he was treated conservatively. Three months later he reported complete resolution of his symptoms and a return to full potency.

Comment

We have found only one other report of temporary erectile impotence after prolonged cycling.[3] The likeliest cause of this complication in our patient seems to be an ischaemic neuropathy of the dorsal (sensory) and cavernous (vasomotor) nerves of the penis induced by compression of the penile crura, to which they are anatomically related, against the pubic bone by the hard narrow saddle. The raised sensory threshold of the dorsal nerve, even five months after the injury, supports this hypothesis. Penile and scrotal hypoaesthesia has been shown in men who have ridden on such saddles, ostensibly as a result of pudendal neuritis.[4] A further factor in our patient may have been a transient compromise in penile arterial inflow. This is suggested by one study which showed a significant fall in the penile blood pressure of 20 healthy men who sat on an unpadded cycle seat for five minutes.[5] The vulnerability of the penile neurovascular supply to compression leads us to believe that short term erectile impotence may be much more common in long distance cyclists than is recognised.

We thank Mr W B Peeling, consultant urologist at St Woolos Hospital, Newport, Gwent, for referring this case.

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Social adversity and perinatal complications: their relation to postnatal depression

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Postnatal depression, long acknowledged as an important mental health problem for mothers, may also have serious consequences for the child.[1] We looked at two sets of variables of possible aetiological importance to postnatal depression that have received surprisingly little attention—namely, the role of perinatal complications and factors reflecting socioeconomic disadvantage.

Patients, methods, and results

We recruited 483 women over nine months in 1982 from the antenatal clinic and booking diary of the John Radcliffe Hospital, Oxford, by approaching every second expectant mother in the last trimester of her pregnancy.[2] The women were interviewed at home six to eight weeks before their expected date of delivery. Social and socioeconomic data were obtained from a semistructured interview. Perinatal data were obtained from the Oxford obstetric data system and included information on length of each stage of labour, antepartum haemorrhage, abnormal presentation, obstetric intervention, perineal and other tears, postpartum haemorrhage, and any other complications at any stage of labour. Data on the infant included information on gestational age, birth weight, neonatal asphyxia, congenital abnormalities, other complications, and admission to the special care baby unit.

Of the 483 women seen antenatally, 460 were available for follow up three months postnatally. Twenty nine women were psychiatrically disturbed at the antenatal assessment, of whom two were unavailable for follow up. Using the general health questionnaire[3] three months postnatally to screen for probable psychiatric disorder and the Montgomery Asberg depression scale and the present state examination[4] interview to examine mental state we identified 31 women as having non-psychotic psychiatric disturbance (present state examination index of definition ≥5), of whom seven had been disturbed antenatally.[2] The 24 women with disorders of postnatal onset were all suffering from depression of at least moderate severity as assessed by the Montgomery Asberg depression scale. None were sufficiently disturbed to require admission.

None of the perinatal factors investigated were related to postnatal depression. Because this surprised us we carried out further analyses to determine whether perinatal factors were related to less severe psychological symptoms with a low threshold for disturbance (general health questionnaire score ≥12). No relation was found.

In contrast, significant relations were found between depression of postnatal onset and certain social factors (table)—namely, having a low family income, neither partner being employed, and not having a confidant. Logistic regression analysis[5] showed that only two

*Relation between social factors and postnatal depression**

Social factor	No (%) with postnatal depression (n=24)	No (%) without postnatal depression (n=410)	p Value
Working class	12 (50)	140 (35)†	NS
Low income‡	10 (42)	67 (16)§	<0·01
Neither partner employed‖	7 (29)	46 (11)	<0·05
No partner	4 (17)	18 (4)	NS
No confidant	5 (21)	14 (3)	<0·01
Primiparous	8 (33)	182 (44)	NS
≥3 Children		22 (5)	NS

χ^2 Or Fisher's exact probability test was used as appropriate.
*All cases of antenatal psychiatric disturbance were excluded from the analysis.
†n=402.
‡Total yearly family income <£4000 (1982 figures).
§n=408.
‖Includes unemployed single mothers.

factors significantly predicted postnatal depression—namely, a low family income and lack of a confidant. There was no interaction between these two factors in their effect. The logistic regression of probability (case)/probability (non-case) on income and the presence of a confidant showed a good fit with an additive model, the mean (SE) estimates for their effects being 1·16 (0·45) and 1·80 (0·59) respectively. This means that, compared with a higher income, a low income increased the odds of developing depression after childbirth by a factor of about 3·2 (95% confidence interval 1·3 to 7·6); and, independently, compared with having a confidant, not having one increased the risk of developing depression by a factor of about 6·1 (95% confidence interval 1·9 to 19·3). Thus having a low income and no confidant increased the odds of postnatal depression by a factor of more than 19.

Comment

Our results suggest that poverty and lack of a confiding relationship are important independent risk factors for the development of postnatal depression and that perinatal complications are not. Health care professionals, particularly general practitioners, health visitors, and midwives, need to be aware of the risk of postnatal depression among women in such socially adverse circumstances.

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Effect of zidovudine on platelet count

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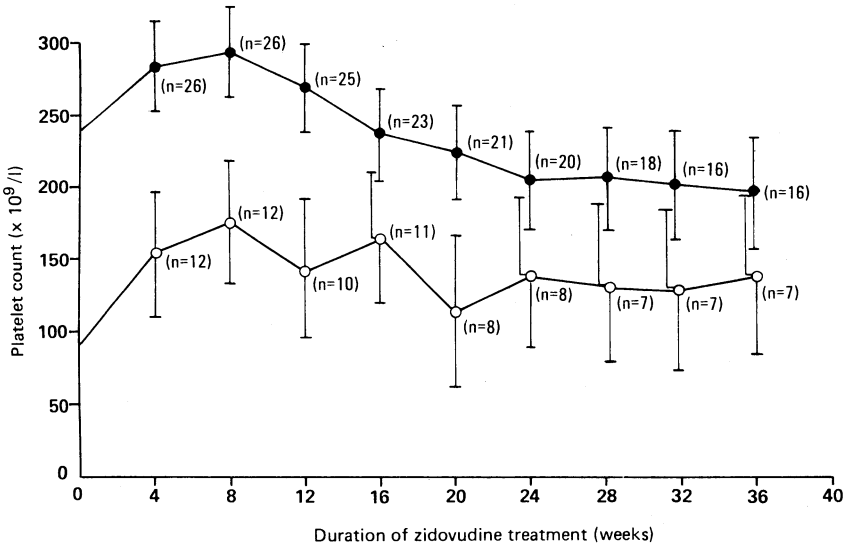
One of the short term effects of zidovudine (3'-azido-3'-deoxythymidine, AZT) is a rise in the platelet count in thrombocytopenia related to HIV infection.[1,2] The long term effects on platelets have not been reported, and doctors have been reluctant to use zidovudine in the presence of severe thrombocytopenia because of its association with myelosuppression. We report our experience of up to 36 weeks of treatment with zidovudine in both patients with thrombocytopenia and those with normal platelet counts.

Patients, methods, and results

Thirty eight patients, 14 with AIDS and 24 with symptomatic HIV infection, were treated with zidovudine for over 12 weeks. Platelet counts were estimated with a Coulter counter (S PLUS IV), the normal reference range being 150-450×10⁹/l, and results were available up to 36 weeks for 23 patients. Before treatment 12 patients (10 intravenous drug misusers, one homosexual, and one recipient of blood products) had platelet counts that were persistently below normal (mean count 91·5×10⁹/l, 95% confidence interval 49 to 134×10⁹/l). In two patients with symptoms of spontaneous bruising the initial counts were less than 15×10⁹/l. Twenty six patients had normal counts before treatment (mean 241×10⁹/l, 211 to 271×10⁹/l).

Platelet counts rose in both groups over the first eight weeks (mean rise from value before treatment 64×10⁹/l). Among the group with a normal platelet count before treatment, however, the count fell gradually after eight weeks to a value below that recorded initially. Counts were better maintained among the patients with thrombocytopenia (figure), and there was a significant association between an eventual rise in platelet count and an initial count below normal (nine out of 12 patients, $p<0·05$, χ^2 test with Yates's correction). The two patients with severe thrombocytopenia had platelet counts of 119×10⁹/l and 102×10⁹/l after 32 and 16 weeks' treatment respectively.

To assess which factors determined whether the platelet count fell eventually variables were studied before and during treatment. An eventual fall in platelet count was associated with the development of severe anaemia (haemoglobin concentration <80 g/l) that necessitated blood transfusion (five patients, $p<0·05$) but not with other markers of possible myelosuppression such as neutropenia and lymphopenia, or with risk group, clinical state, and occurrence or treatment of opportunistic infections. Severe anaemia developed only in patients who had normal platelet counts initially.



Mean platelet counts and 95% confidence intervals in patients with (○) and without (●) thrombocytopenia taking zidovudine

Comment

An immune thrombocytopenia related to HIV infection results from the reticuloendothelial removal from the circulation of platelets coated with immunoglobulin, but other causes of a fall in platelet count in HIV infection include clinical progression to AIDS, recovery from *Pneumocystis carinii* pneumonia,[3] and myelosuppression from drugs, or antibodies to glyco-