Transverse posterior cervicoisthmic rupture after gemeprost pessaries for termination

Drs S J VINE and A PRYS-DAVIES and Mr J F PEARSON (University Hospital of Wales, Cardiff CF4 4XN) write: A 16 year old woman was admitted for termination of her first pregnancy at 16 weeks' gestation. A gemeprost (16,16-dimethyl-trans- Δ^2 prostaglandin E₁ methyl ester) 1 mg pessary was inserted into the posterior fornix every three hours until a total dose of 5 mg had been given. The cervix remained closed and uneffaced, so a second identical course of gemeprost pessaries was started 24 hours later. Intermittent lower abdominal pain and vaginal bleeding began after the third pessary. Three hours later she aborted the single fetus, but not the placenta, at which time there was 400 ml blood loss. Examination under general anaesthesia showed the vagina to be full of clot, with a 4 cm transverse full thickness defect in the posterior wall of the cervix at the level of the isthmus, not affecting the vaginal skin. The placenta was removed through the defect, which was then sutured. The patient required two units of blood and antibiotics. She recovered uneventfully and was discharged after three days.

Uterine rupture has been described after a combination of gemeprost pessaries and intravenous oxytocin.¹ There has also been a report of uterine rupture after gemeprost alone, but this was in a multiparous woman in whom both an amniocentesis and chorionic villus sampling had been performed.² The Committee on Safety of Medicines has had three reports of uterine perforation after the use of gemeprost (personal communication), and the manufacturers (May and Baker) are aware of 12 cases of uterine-cervical rupture, nine of which occurred in association with either intravenous oxytocic agents, instrumentation of the cervix-uterus at evacuation, or multiparity. One of the remaining cases was a cervical tear in a 16 year old, which appears to be similar to this case, but there is no other information. Cervical rupture does not seem to have been described after gemeprost pessaries alone in a nulliparous woman with no risk factors.

Transverse rupture at the cervico-isthmic junction in induced second trimester abortion has been reported after the administration of other prostaglandin preparations (PgF_{2 α}),³ and following the use of both prostaglandins and intravenous oxytocin.⁴

The manufacturer's data sheet suggests a maximum dosage of 10 mg gemeprost. This patient received 8 mg, with the 12 hour rest period after 5 mg as recommended by Cameron *et al.*⁵ Gemeprost has now been in regular use for midtrimester abor-

tions since 1988. As it is seen to be more convenient and as effective as intra-amniotic or extra-amniotic prostaglandins more cases such as this may be reported.

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Peripheral neuropathy in a patient receiving enalapril

Drs A HORMIGO and MANUELA ALVES (Instituto Português de Oncologia, Lisbon, Portugal) write: We describe a patient in whom neuropathy developed while she was receiving enalapril and completely resolved after withdrawal of the drug.

A 56 year old woman with mild hypertension had taken captopril for two years before starting enalapril, 5 mg daily, which she had been taking for 18 months. She then complained of progressive paraesthesia of hands and feet and three months later she was referred to us. There were no risk factors for neuromuscular disorder. Neurological examination showed normal muscular strength; low sensation of pain, temperature, and vibration sense with glove and stocking distribution; and preserved tendon reflexes. She had a normal haematological, biochemical, and immunological profile. Needle electromyography of the extensor digitorum brevis showed no spontaneous activity and a moderately neurogenic pattern on volitional activity. Motor and sensory conduction velocities were reduced (peroneal nerve 47 m/s and proximal sural nerve 48 m/s). Sensory amplitude of the proximal sural nerve was reduced (5 μ V). Enalapril was then withdrawn. The symptoms progressively disappeared, and six months later she had normal responses on neurological and neurophysiological examination. The motor conduction velocity of the peroneal nerve was then 60 m/s and the sensory conduction velocity of the sural nerve 65 m/s. The sensory amplitude of the proximal sural nerve had also reverted to normal (15 μ V).

Paraesthesia has been reported during postmarketing surveillance

of enalapril,¹ and peripheral neuropathy and Guillain-Barré syndrome have been recognised as side effects of captopril.^{2,4} To our knowledge this case is the first report of peripheral neuropathy probably caused by enalapril.

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Hyponatraemia associated with paroxetine

Dr CAIT GODDARD and Ms CAROL PATON (Bexley Hospital, Kent DA5 2BW) write: Hyponatraemia has been associated with many psychotropic drugs, including carbamazepine,¹ neuroleptics,² tricyclic antidepressants³ and most recently, fluoxetine.^{4,5} We describe hyponatraemia associated with paroxetine.

A 69 year old woman was admitted to hospital with a preliminary diagnosis of agitated depression. On admission routine haematology and biochemistry screens gave normal results; her serum sodium concentration was 139 mmol/l. It soon became apparent that she had pronounced cognitive impairment and appearances on computerised tomography were consistent with multi-infarct dementia. She was very agitated and depressed with some insight into her memory problems and on 7 May was prescribed paroxetine 20 mg/day increasing to 40 mg after a week. On 23 May she was noted to be drowsy and was complaining of "feeling strange—as if I'm not here." These symptoms were initially attributed to a vascular event, although her plasma sodium value was noted to be 129 mmol/l. By 27 May she was very lethargic and unsteady on her feet. Plasma sodium concentration was 126 mmol/l. There was no obvious cause for her hyponatraemia, so paroxetine was discontinued (fluid was not restricted), and two days later her physical state was much improved. Plasma sodium concentration was 134 mmol/l. Serum and urine osmolality were not measured. Other biochemical variables were normal at all times. Her only other medications were aspirin 75 mg/day and lactulose 20 ml/day. Both were continued throughout. Rechallenge was thought to be unethical.

Although this case suggests a link between paroxetine and hyponatraemia, the clinical data are insufficient to confirm a causal relation. The manufacturers have,

however, received six reports and the Committee on Safety of Medicines 13 reports linking the two. The newer antidepressants may not be the answer to all the toxicity problems associated with tricyclic agents and should themselves be prescribed with care, particularly in elderly people.

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- Sandifer MG. Hyponatraemia due to psychotropic drugs. *J Clin Psychiatry* 1983;44:301-5.
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Bilateral hypopyon and streptokinase

Mr D KINSHUCK (Wolverhampton Eye Infirmary, Wolverhampton WV3 3QR) writes: A 69 year old white woman developed bilateral acute hypopyon (severe uveitis) two days after receiving intravenous streptokinase (Kabi Pharmacia) for a myocardial infarct. She made a complete recovery with periocular steroid injections and drops, and also had no noticeable long term cardiac problem. She had longstanding diabetes, well controlled by diet, and no history of allergies.

Such a severe uveitis after streptokinase has not been reported, although the Committee on Safety of Medicines has received one other report of uveitis. The timing strongly suggests that either the infarct or the drug were responsible. I have been unable to find any report of such a uveitis occurring related to myocardial infarct, although I have personally seen one patient with an acute unilateral hypopyon two weeks after a myocardial infarct (not treated with streptokinase). This patient also made a complete recovery. Acute bilateral hypopyon is unusual and occurs in patients with Behçet's disease, in HLA-B27 positive patients (usually with a history of ankylosing spondylitis or Crohn's disease), and in those with embolic infective endophthalmitis.^{1,2} This patient had no history of illness apart from diabetes. Her HLA status was not tested. Anterior uveitis is thought to have an immune basis, as are other reactions to streptokinase such as the Guillain-Barré syndrome.³

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