Letters to the Editor

Lower segment uterine scar rupture during induction of labour with vaginal prostaglandin E2

Sir,

A case of lower segment uterine scar rupture during induction of labour with prostaglandin E2 vaginal pessaries prompted us to review the literature with consideration to the relative safety of prostaglandin and oxytocin for induction of 'trial of scar' labour.

A healthy 30 year old previously had an elective lower segment Caesarean section for a breech presentation of a 3,317 g infant. Induction of labour at term was arranged in her second pregnancy because of reduced fetal movements. The cervix remained unfavourable (Bishop's score less than 5) despite the vaginal administration of three prostaglandin E2 3 mg pessaries over a 15 hour period. Painful tonic uterine contractions preceded sudden severe abdominal pain and persistent fetal bradycardia approximately 2 hours after insertion of the third pessary. Fifteen minutes later an emergency laparotomy was performed and a complete lower segment uterine scar rupture was discovered, extending posterolaterally on each side to extend through 270 degrees of the lower segment. A 3,756 g male infant was delivered through the rupture, with Apgar scores of 2 at 1 minute and 7 at 5 minutes. The tear was repaired with 600 ml total blood loss. The postoperative course was uncomplicated for mother and baby.

The perinatal mortality rate from vaginal birth after lower segment Caesarean section does not vary significantly from the general obstetric population.¹ The overall incidence of uterine scar rupture is only 1.7 per 1,000 'trial of scar' labours.² The safety of oxytocin with careful maternal and fetal monitoring in labour induction and augmentation in these patients is well established.^{3–6} However, few papers have assessed the safety of prostaglandins for cervical ripening and induction of labour in the presence of a lower segment uterine scar. MacKenzie *et al.*⁷ induced labour at term with vaginal prostaglandin E2 in 143 such patients without uterine rupture, and achieved vaginal delivery in the 68% of patients who initially had a very unfavourable cervix. The authors suggest the risk of rupture is small due to the low amplitude of the frequent uterine contractions promoted by prostaglandins.⁷ Sixteen of 19 'trial of scar' labours induced by Shepherd *et al.* with prostaglandin vaginal pessaries achieved vaginal delivery without augmentation, with no evidence of uterine scar rupture or dehiscence.⁸ However, Bromham and Anderson questioned the safety of vaginal prostaglandin E2 quoting one case of uterine scar rupture in the first stage of labour and two cases apparently following forceps delivery.⁹

The literature does not allow comparison of the relative safety or efficacy of vaginal prostaglandin E2 and oxytocin for the induction of labour in the presence of a lower segment uterine scar. The risk of uterine rupture with either method is low, but our case reinforces the need for appropriate precautions and close monitoring for signs of this potentially catastrophic event.

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Haemophilus influenzae meningitis in an elderly patient despite treatment with oral cefuroxime

Sir,

Haemophilus influenzae is a rare case of meningitis in the elderly.

An 87 year old woman presented with a short history of confusion, epigastric pain and vomiting but with no evidence of meningism. The epigastric pain and vomiting settled quickly. On day 2 her temperature rose to 38.9°C. She was started on oral ampicillin 250 mg four times daily for a presumed chest infection after blood cultures were taken. By day 5 her temperature came down but remained slightly elevated, and the blood culture grew a beta-lactamase producing *H. influenzae* sensitive to chloramphenicol, cefuroxime and trimethoprim. As her general condition had improved and the temperature was settling, she was started on oral cefuroxime 500 mg twice daily. Her temperature returned to normal but by day 7, having had five doses of cefuroxime, she became pyrexial again (39°C) with a marked deterioration in her condition. She developed intermittent focal motor seizures affecting her right hand side, and also marked neck stiffness. A

computed tomographic scan of her head was normal, but examination of the cerebrospinal fluid (CSF) revealed protein 1.8 g/l, a glucose level of 1.2 mmol/l, a significantly elevated white cell count of $750 \times 10^6/l$ (70% polymorphs). She was started empirically on intravenous chloramphenicol 1 g six hourly, and intravenous acyclovir 250 mg thrice daily. Cultures of the CSF yielded *H. influenzae* identical to that grown in the blood cultures. The acyclovir was stopped on day 9, and on day 11 her intravenous chloramphenicol was changed to oral before stopping on day 14.

H. influenzae meningitis is extremely rare in the elderly.¹ Although *H. influenzae* type b is traditionally considered more virulent as a cause of bacteraemia and meningitis in early life,² in the elderly an appreciable proportion of *H. influenzae* respiratory infections result from non-capsulated *H. influenzae* strains.³ Defects in the host's immune function that occur with age, along with a high incidence of chronic diseases, may render this part of the population more susceptible to invasive illnesses such as bacteraemia or meningitis as have been noted in children.⁴ Although our hospital antibiotic policy is to use parenteral antibiotics in patients with bacteraemia, the oral cefuroxime was used mainly due to improving condition of our patient on the oral therapy with ampicillin and partly due to the claims that oral cefuroxime is well absorbed through the gut and quickly achieves high serum levels.⁵ Our patient almost certainly had a defect in her humoral immune system and this may have been partly responsible for the apparent failure of oral cefuroxime to prevent progression of the disease despite the organisms apparent sensitivity to the drug *in vitro*.

In conclusion, uncapsulated *H. influenzae* may play a more pathogenic role in elderly patients with compromised immunity and, despite the improving clinical picture, it is essential to use parenteral antibiotics where bacteraemia is either confirmed or suspected.

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Reversible hyporeninaemic hypoaldosteronism and life-threatening cardiac dysrhythmias: the interaction of non-steroidal anti-inflammatory drugs and autonomic dysfunction

Sir,

Hypoaldosteronism has been described in association with postural hypotension accompanying autonomic neuropathy, as a consequence of inadequate renin stimulation.¹ Both hyperkalaemia and hyporeninaemic hypoaldosteronism have also been reported in patients with impaired renal prostaglandin biosynthesis, for example, during non-steroidal anti-inflammatory drug therapy.²

We describe a patient with autonomic dysfunction, who presented with life-threatening cardiac dysrhythmias associated with hyperkalaemia, following a short course of ibuprofen therapy, which may have precipitated hyporeninaemic hypoaldosteronism.

A 59 year old previously well man was admitted after an episode of loss of consciousness. He subsequently had a cardiac arrest associated with ventricular standstill. Successful cardiopulmonary resuscitation was performed. An initial serum potassium level was 8.0 mmol/l, and an electrocardiogram showed broad QRS complexes and peaked T waves. Serum creatinine and urea were within the normal range (creatinine 102 μ mol/l, urea 6.0 mmol/l). Treatment with intravenous calcium gluconate, dextrose and insulin brought the potassium level into the normal range. Subsequent enquiry revealed he had recently taken a course of ibuprofen for backache. There was no history of any other medication including potassium-sparing diuretics. Calcium resonium and fludrocortisone were prescribed on the basis that he may have developed hyporeninaemic hypoaldosteronism (and hyperkalaemia) secondary to the non-steroidal anti-inflammatory drug.

He was discharged home normokalaemic, but after 3 weeks was readmitted with palpitations and developed another episode of ventricular standstill requiring temporary intracardiac pacing. The initial potassium level on this occasion was 6.0 mmol/l. Autonomic nerve function tests were performed and these demonstrated a degree of autonomic neuropathy. Blood pressure was 160/90 mmHg supine and 130/90 mmHg erect. There was complete absence of the normal variation in heart rate during deep inspiration,³ and pupillary diameter in darkness was 32% (fifth centile for 59 year old men = 41.5%).⁴ Several random plasma blood glucose levels were in the normal range excluding diabetes mellitus, and there were no clinical features to suggest other specific causes of autonomic dysfunction such as the Shy–Drager's syndrome.

Supine and ambulant (erect) plasma aldosterone and plasma renin activity were measured after the fludrocortisone and calcium resonium had been stopped for over 3 weeks, and the results suggested hyporeninaemic hypoaldosteronism: resting supine aldosterone level was 119 pmol/l (reference range = 28–444 pmol/l). Ambulant aldosterone level was 105.5 pmol/l (reference range = 111–859 pmol/l). Supine plasma active renin was 2.9 pmol/l (reference range = 0.8–14.5 pmol/l). Ambulant plasma active renin was 2.7 pmol/l (reference range = 1.1–42.2 pmol/l). These results show the failure of the normal expected rise in supine aldosterone and