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CORRESPONDENCE

Bronchospasm following ergometrine in a non-asthmatic patient

A 28-year-old para 2 woman with singleton pregnancy at 41 + 4 weeks of gestation was admitted to hospital for induction of labour. She was given a 3-mg prostaglandin gel, which successfully stimulated labour. She had no background medical history but smoked 10 cigarettes per day (30–35 per day before pregnancy) and admitted having a 'smoker's cough'. For labour analgesia she initially used Entonox and intramuscular diamorphine. A lumbar epidural was subsequently sited with good effect. Intrapartum cardiotocograph findings were unremarkable. Artificial rupture of membranes was performed and an oxytocin infusion started. Labour progressed well and she had a spontaneous vaginal delivery of a healthy baby girl (Apgar scores of 8 and 9 at 1- and 5-min, respectively) 24 h after receiving the prostaglandin gel. Following delivery, intramuscular Syntometrine® 1 mL (oxytocin 5 IU, ergometrine maleate 0.5 mg) was given. Ten minutes later she complained of sudden severe shortness of breath and dizziness. On assessment she was cyanosed with oxygen saturations of 76%, tachypnoeic (respiratory rate of 45 breaths/min) and tachycardic (heart rate of 135 beats/min). Auscultation of her chest revealed widespread wheeze with reduced chest expansion. She had no petechiae, altered conscience or evidence of acute cardiac failure. Arterial blood gas showed a PaO₂ of 8.6 kPa and PaCO₂ of 4.1 kPa on oxygen 15 L/min. A 12-lead electrocardiogram showed a sinus tachycardia and chest radiograph was unremarkable. She was promptly treated with nebulised salbutamol 10 mg and intravenous magnesium sulphate 2 g resulting in improvement. Within 30 min her saturations and blood gases had returned to normal on room air. A provisional diagnosis of acute bronchospasm secondary to ergometrine was made.

Ergometrine maleate is an ergot alkaloid agent which is commonly used in obstetrics to stimulate contraction of uterine smooth muscle after second stage of labour. In our unit this drug is routinely given to aid completion of third stage of labour and is recommended in the treatment of atonic postpartum haemorrhage.¹ Smooth muscle contraction caused by ergometrine is not confined to the uterus. An in vitro study involving acetylcholine pre-treated canine tracheal smooth muscle exposed to ergometrine revealed increasing muscle contraction in the presence of higher ergometrine concentrations.² There is no specific receptor site on smooth muscle for ergometrine but contraction is triggered by release of

5-hydroxytryptamine (5-HT₂). An in vitro study showed that ergometrine-induced contraction of isolated coronary artery smooth muscle was offset by the addition of methysergide, a known 5-HT antagonist.³ Whilst there have been no in vivo controlled trials demonstrating the causal relationship between ergometrine and acute bronchospasm, there have been several case reports describing a similar clinical scenario as encountered above.^{4–7} In each case, ergometrine was administered and acute severe bronchospasm followed soon after. In each case the episode was effectively treated with bronchodilator therapy and symptoms eventually improved. Furthermore in each of these case reports, the patient was documented as having asthma requiring inhaled bronchodilators. Therefore, ergometrine triggered symptoms in those with susceptible airways.

In this case, the patient had no previous history of asthma or inhaler use. She may have had increased airway sensitivity because of regular cigarette smoke exposure, a known airway irritant. Cigarette smoke can trigger smooth muscle contraction via increasing afferent bronchopulmonary C-fibre nerve excitability from release of local inflammatory mediators, possibly histamine and prostaglandins PGE-2.⁸ This case highlights that bronchospasm following ergometrine may not be confined to asthmatics. A history of cigarette smoking during pregnancy predisposes to an irritable airway which could lower the threshold for bronchospasm following ergometrine administration. Careful monitoring of respiratory rate and work of breathing would provide early warning and treatment of impending bronchospasm in these patients.

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