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Rehan Khan, E. Anastasakis & R. A. Kadir

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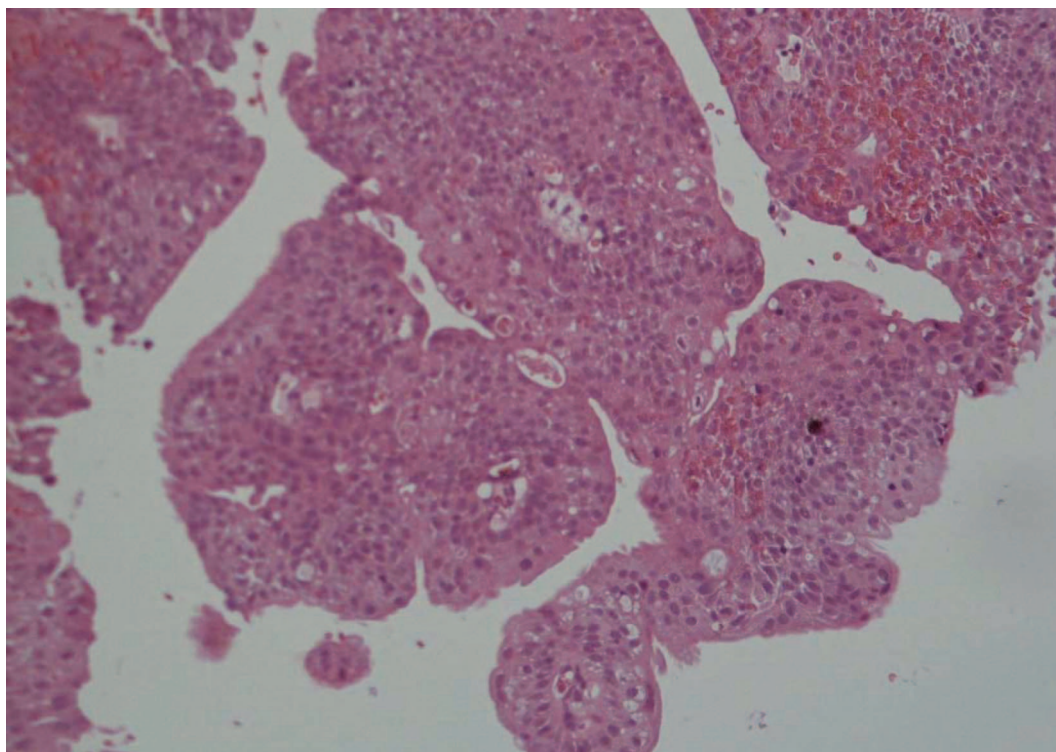


Figure 2. Photo-micrograph of Grade-I transitional cell carcinoma of bladder.

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Correspondence: Mrs. Keyuri N. Shrotri, Department of Obstetrics and Gynaecology, Queen Elizabeth Queen Mother Hospital, St. Peter's Road, Margate, Kent CT9 4AN, UK. E-mail: keyuri.shrotri@ekht.nhs.uk

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Anaphylactic reaction to ceftriaxone in labour. An emerging complication

REHAN KHAN, E. ANASTASAKIS & R. A. KADIR

Department of Obstetrics and Gynaecology, Royal Free Hospital, London, UK

Introduction

The incidence of anaphylactic reaction to antibiotics in labour is unknown. Reisner et al. (2000) investigated a cohort of 9,932 infants who received either ampicillin or erythromycin during labour in order to prevent transmission of group *B Streptococcus*. There were no cases of maternal anaphylaxis in this cohort. Thus, antibiotic – mediated anaphylaxis in labour is considered a rare

event. However, 0.7–10% of all pregnant women are allergic to penicillin, resulting in a fatality rate of 1 in 100,000 (Gilman et al. 1990).

We present the first case of a severe anaphylactic reaction to intravenous ceftriaxone in a 37-year-old primigravida with acute chorioamnionitis in labour. In addition, we performed a literature review using the Ovid Online in order to search the EMBASE and Medline.

Case report

A 37-year-old white Caucasian gravida 1, para 0, was admitted to the labour ward during the latent phase of labour at 38 weeks' gestation. She had a history of antibiotic treatment for a kidney infection 15 years prior to this pregnancy. The patient reported a 'mild allergy' to erythromycin and trimethoprim, but had no history of anaphylaxis, asthma, or eczema, and no history of penicillin allergy. It also emerged after her delivery that she had hayfever and contact sensitivity to metals. The booking appointment at 12 weeks' gestation revealed no other obstetric risk factors and routine antenatal investigations were normal. She had a normal anatomy scan at 20 weeks and a normal growth scan at 34 weeks' gestation.

She was admitted to hospital from home at 38 weeks' gestation with a history of pre-labour ruptured membranes which was confirmed on speculum examination. At initial assessment she was afebrile, draining clear liquor, and cardiotocography was normal. During the following hours, uterine contractions were mild and irregular. Expectant management with regular maternal and fetal assessments was therefore planned.

A total of 17 h after her membranes had ruptured, she complained of feeling cold and feverish. She was transferred to the labour ward for further assessment, and on arrival, she had a witnessed episode of rigor with temperature of 37.8°C and a tachycardia of 90 b.p.m. Uterine contractions were occurring every 3–4 min.

Cardiotocography recorded fetal tachycardia with decreased baseline variability. On examination, the abdomen was soft and non-tender, the head was not engaged. The cervix was 1 cm dilated and 2 cm long, with clear and non-offensive liquor draining. An intravenous cannula was inserted, blood and urine and vaginal swabs were sent for microbiological examination, and blood was sent for full blood count and CRP. Intravenous ceftriaxone 1 g was administered.

At 7 min after the ceftriaxone administration, the patient complained of feeling dizzy, faint and short of breath. She rapidly developed a generalised urticarial erythematous maculopapular rash, orbital oedema, together with a tachycardia of 160 b.p.m. Her blood pressure fell to 70/unrecordable mmHg. She was resuscitated by the obstetric and anaesthetic teams, who made an immediate presumptive diagnosis of antibiotic-related anaphylaxis. Her airway was secured and she was given 100% oxygen via facemask; oxygen saturation was 99% at this point and remained satisfactory throughout. A urinary in-dwelling catheter was inserted. Intravenous colloid and crystalloid solutions were administered rapidly. She was given intravenous chlorpheniramine 10 mg slowly within 3 min of the start of the anaphylactic episode, with no resulting improvement in her vital signs; 8 min later, she was given a slow intravenous bolus of hydrocortisone 100 mg, followed by intravenous adrenaline 10 µg. By 20 min after the onset of the hypotensive episode, she had received the above drugs plus 1 litre of crystalloid and 1 litre of colloid.

At this point, she started to feel slightly better, and her pulse fell to 120 b.p.m. with a blood pressure of 90/45 mmHg. However, the cardiotocograph had deteriorated during the above episode. Fetal tachycardia was recorded with decreased baseline variability and late decelerations. A decision for delivery was taken as early as 30 min after anaphylaxis began; however the patient was still being resuscitated at this time, and the vital signs improved only after another 30 min. An emergency caesarean section was then performed under general anaesthesia. The infant, a 3.052 kg girl, was delivered. Apgar scores were 5 at 1 min, 7 at 5 min and 8 at 10 min. The amniotic fluid was stained with blood, suggestive of placental abruption, but no retroplacental clot was identified. The operation was otherwise uncomplicated apart from an atonic postpartum haemorrhage of 800 ml. This was controlled with oxytocic agents.

The infant was apnoeic at birth with sporadic gasping, and a heart rate of 100 b.p.m., and resuscitation consisted of stimulation/bag and mask ventilation followed by endotracheal intubation and

IPPV. Cord blood samples showed an umbilical artery pH of 7.08 with base excess of –16.0, and umbilical vein pH of 7.14, with base excess of –13.9. The baby was admitted to the neonatal unit. One day after delivery, the baby was observed to be jittery with episodes of abnormal movements thought to represent fits. The infant was ultimately diagnosed with Grade 2 hypoxic ischaemic encephalopathy, both clinically and by the use of cranial ultrasound scan. At 1 week of age the abnormal movements became less, and the baby was discharged home after 1 further week in the neonatal unit. By 11 weeks of age, she had reached the appropriate milestones but was not yet smiling.

Postoperatively, the mother's blood pressure was much improved at 126/67 mmHg, and her rash quickly dissipated. The tachycardia steadily settled over the following 8 h, and the urine output remained satisfactory. She was given intravenous metronidazole without any further adverse effects. The laboratory subsequently reported a preoperative white cell count of 19.4 g/dl with a CRP of 1. The subsequent postoperative recovery was uncomplicated and she was discharged home after 6 days. Blood cultures proved to be negative, but both vaginal and placental swabs grew group *B beta-hemolytic streptococcus*. The retrospective diagnosis was therefore of acute chorioamnionitis in labour, followed by acute anaphylactic shock and presumed placental abruption. She was reviewed 11 weeks after delivery as an outpatient, and had made a good physical recovery. A plan was made for her to be reviewed by an immunologist in order to clarify the extent of her allergies and identify which antibiotics she could safely receive in the future.

Discussion

The intrapartum use of antibiotics is long established and widespread. Penicillin is given during labour to prevent transmission of group *B Streptococcus* to the newborn. Cephalosporins demonstrate broad spectrum cover and are used to treat pyrexia of unknown origin during labour. All antibiotics carry a potential risk of allergic and anaphylactic reactions. These reactions are IgE-mediated responses; usually following a sensitising event (Sogn 1984). Anaphylaxis includes a spectrum of clinical features, such as flushing, itching, urticaria, angioedema, bronchospasm, hypotension and ultimately coma and death (Matthews 1984). The anaphylactic reaction usually occurs within minutes of the drug administration. IgE does not cross the placental barrier, and the decidua can catalyse inflammatory mediators, but maternal hypotension can result in fetal hypoxia necessitating delivery (Baraka and Stein 1980).

There have been five case reports in the literature of anaphylaxis related to intrapartum antibiotic administration summarised in Table I (Gallagher 1988; Bloomberg 1988; Heim et al. 1991; Konno and Nagase 1995; Dunn et al. 1999). All cases, including ours, highlighted the acute maternal hypotension resulting in fetal bradycardia. In three of the reported cases, fetal bradycardia persisted, despite correction of the maternal blood pressure, necessitating emergency caesarean section, and subsequent admission to the neonatal unit (Heim et al. 1991; Konno and Nagase 1995; Dunn et al. 1999). Heim et al. (1991) reported a case where the infant presented profound neurological abnormalities at 6 months of age.

Anaphylaxis is managed by supportive measures, such as oxygen and intravenous fluids, and vasoactive medications, such as steroids, adrenaline, chlorpheniramine and adrenaline. It has been suggested that adrenaline (epinephrine) can cause vasoconstriction of the uterine vessels by acting on α -adrenergic receptors and can thus worsen fetal hypoxia (Konno and Nagase 1995; Dunn et al. 1999; Entman and Moise 1984). Ephedrine causes an increase in the presynaptic noradrenaline release. Thus, it would be less likely to cause uterine arteriole vasoconstriction (Ralston et al. 1974). In managing this emergency, we considered the safe resuscitation of the mother to be of paramount importance, and adrenaline a more effective drug in acute anaphylactic shock.

Table I. Previous reported cases of anaphylactic reactions to antibiotics in labour.

Author	Antibiotic	CTG changes	Mode of delivery	Maternal outcome	Neonatal outcome
Gallagher 1988	Ampicillin	Bradycardia	Vaginal delivery with fundal pressure	Subendocardial ischemia; normal on discharge	Normal development at 9 months
Bloomberg 1988*	Cefoxitin	Given post-delivery	Caesarean section	Normal recovery	Normal on discharge
Heim et al. 1991	Ampicillin	Bradycardia	Caesarean section	Normal recovery	Pronounced neurological abnormalities at 6 months
Konno and Nagase 1995	Cefazolin	Bradycardia	Caesarean section	DIC	Normal development at 12 months
Dunn et al. 1999	Penicillin	Bradycardia	Caesarean section	DIC and acute tubular necrosis needing dialysis	Normal on discharge

*Three separate cases are reported in this paper.

The American College of Obstetricians and Gynecologists and the Centers for Disease Control and Prevention have developed strategies for the prevention of neonatal group *B Streptococcus* infection (ACOG 1992; AAPC 1992). One strategy is to administer antibiotics in labour to all women with prolonged rupture of membranes, pre-term labour or pyrexia during labour. This strategy is also widely used in the UK. A second strategy in the USA is to screen all pregnant women at 35–37 weeks' gestation for group *B Streptococcus* and administer intrapartum antibiotics to those women who test screen positive. It has been estimated that a 'screen-and-treat' approach would lead to 26.7% of women being given treatment in labour, with a resulting prevention of 86% of neonatal group *B Streptococcus* cases. As this infection causes significant neonatal mortality, antenatal screening has become increasingly attractive. However, it is inevitable that if more women receive intrapartum antibiotics than currently do so, the incidence of serious side-effects, including anaphylaxis, will rise. The clinician has to be aware of the signs and symptoms that would lead to prompt recognition and management of this severe condition.

In conclusion, there is an increasing need for awareness of the symptoms and signs of anaphylaxis in labour. Even if actions are prompt, there can be serious maternal and fetal sequelae. All personnel delivering obstetric care needs to be aware of this potential complication arising from the effort to prevent group *B Streptococcus* infection to the newborn.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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Correspondence: R. A Kadir, Department of Obstetrics and Gynaecology, 5th floor, Aldrich Blake Ward, Royal Free Hospital, Pond Street, London NW3 2QG, UK. E-mail: rezan.abdul-kadir@royalfree.nhs.uk

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