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CORRESPONDENCE

Detecting pulmonary edema in multiple pregnancy through point-of-care lung ultrasonography

We managed a 29-year-old, gravida 1 para 0, woman at 32 weeks-of-twin-gestation who underwent emergent cesarean section for arrest of descent. She had been treated with ritodrine and dexamethasone for two days. At the operating room her body temperature was 38.5°C; blood pressure 130/85 mmHg; heart rate 125 beats-per-minute; respiratory rate 23 breaths-per-minute and oxygen saturation 95% on room air. General anesthesia was chosen because of the lack of a routine blood examination and our suspicion of systemic infection. Rapid sequence induction was with remifentanil, propofol and succinylcholine. After intubation her oxygen saturations decreased to as low as 82%, ranging from 89–95% with manual ventilation. The baby was delivered while the maternal inspired oxygen was 100%. Auscultation of her lungs revealed wheezing. The surgery lasted 40 minutes, the blood loss was 800 mL and the patient received one litre of crystalloid solution, associated with a urine output of 400 mL. After extubation, her lowest oxygen saturation was 88% on room air. Five hours later, in the intensive care unit, lung auscultation revealed bilateral rales and her pro-brain natriuretic peptide concentration was 1020 pg/mL. She was discharged after 10 days of treatment for infection and heart failure.

Another 28-year-old, gravida 1 para 0, woman was scheduled for cesarean section at 35 weeks-of-twin-gestation for breech presentation. She had received tocolytic therapy with ritodrine and dexamethasone for 20 days. When she arrived in the operating room, lung ultrasonography revealed multiple B-lines, despite no abnormalities being heard on lung auscultation. Combined spinal-epidural anesthesia was performed and intravenous fluid volumes were restricted. Nevertheless, she complained of dyspnea after delivery and lung ultrasound showed the "rockets" sign in the lower lung fields. Intravenous furosemide was administered and her symptoms rapidly improved. She was sent to the ward and discharged two days later.

Multiple pregnancy is a risk factor for pulmonary edema, which can be life-threatening and is a common reason for intensive care admission. Point-of-care lung ultrasonography is beneficial, as the anesthesiologist may be able to detect pulmonary edema early and make appropriate decisions. The maternal blood volume in twin pregnancy is about 400 mL more than a single pregnancy.[1] Volume overload contributes to the development of pulmonary edema and after delivery, blood volume increases further following uterine contraction, potentially worsening pulmonary edema. These processes appeared apparent in our first patient, although ritodrine used for tocolysis may have been an additional contributor.[2] Both our patients were treated with ritodrine.

The diagnosis of pulmonary edema is primarily confirmed on chest radiography: lung auscultation has low sensitivity. In contrast, lung ultrasonography is a safe, effective and sensitive method of diagnosing pulmonary edema. In multiple pregnancy, a finding of three or more B-lines in a lung field is 86–93% sensitive and 93–98% specific for pulmonary alveolar interstitial syndrome.[3,4] Spinal anesthesia may be preferable when multiple B-lines are detected, as this may reduce venous return, but fluid restriction is also recommended. After delivery, lung ultrasonography can help in the early recognition and treatment of pulmonary edema. In our second patient, point-of-care lung ultrasonography proved valuable, so that pulmonary edema was not exacerbated. The patient avoided intensive care unit admission.

In conclusion, multiple pregnancy with tocolytic treatment is a well-known risk for pulmonary edema. Point-of-care lung ultrasonography may a help detect pulmonary edema early and assist in making timely clinical decisions. Lung ultrasonography has the potential to improve the quality of care of women with multiple pregnancy.

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Coagulopathy in obstetric cholestasis in Wessex Deanery

Obstetric cholestasis affects approximately 0.7% of pregnancies in England.[1] The condition is defined as pruritus with an onset in pregnancy, abnormal liver function in the absence of other liver disease, and resolution following delivery.[2] Obstetric cholestasis is of relevance to anaesthetists due to concern regarding the development of coagulopathy and the implications for regional anaesthetic and analgesic techniques. Coagulopathy is hypothesised to occur from the malabsorption of vitamin K, secondary to reduced bile acid secretion in the gastrointestinal tract. However, this concern is based upon limited evidence from small, retrospective studies.[3,4] Bacq et al. demonstrated an incidence of abnormal prothrombin time of 8% in 49 cases of obstetric cholestasis.[3] This rate has not been found in subsequent studies, and most recently DeLeon et al. found no cases of abnormal clotting in 319 parturients.[2,5,6] In the presence of conflicting data, and given that the consequences of an epidural haematoma secondary to coagulopathy are extreme, we sought to add to the current literature and understanding of obstetric cholestasis by conducting a multicentre observational study.

We performed a retrospective cross-sectional study across three hospital trusts in Wessex Deanery, co-ordinated by the local trainee-led research network. Approval for the study was obtained (South Central – Hampshire A Research Ethics Committee (14/SC/1456)). Inclusion criteria were parturients diagnosed with obstetric cholestasis, defined as at least one serum bile acid result greater than 14 μmol/L, who delivered between January 2010 and December 2014 and had a coagulation test result during their pregnancy. Electronic maternity databases were interrogated to identify women diagnosed with obstetric cholestasis. Biochemistry departments were contacted to identify all women of reproductive age who had a bile acid assay during this time period, to mitigate against incomplete records. Patient notes were reviewed to identify obstetric cholestasis treatment initiation and the presence of co-morbidities that could account for deranged biochemistry or coagulation. The primary study outcome was the prevalence of deranged coagulation studies within the study population (International Normalised Ratio (INR) greater than 1.4).[7] Sample size analysis determined that at least 113 subjects would be required to detect an 8% incidence of coagulopathy, based upon previous studies, with a 5% precision error.[8]

During the study period 745 parturients were diagnosed with obstetric cholestasis. With a combined number across trusts of 15 000 deliveries per year, this equates to a prevalence of 1%. Of those, 290 (39%) were excluded because they had no coagulation study result. In total, the 455 women included had 596 coagulation studies analysed. We identified no abnormal coagulation results, giving an incidence of coagulopathy of 0.0% (95% confidence interval 0.0% to 0.84%, calculated using a Poisson distribution). The mean INR (median [range]) was 0.9 (0.9 [0.8–1.2], n=303). The majority of coagulation studies were taken within 24 hours of delivery (75%, n=261). Serum bile acid and alanine aminotransferase (ALT) levels sampled at the same time as coagulation studies were severely deranged (bile acids >100 μmol/L; ALT >200 IU/L) in 9% and 15% of women, respectively.

None of the 455 parturients with obstetric cholestasis developed a coagulopathy. This is despite co-existing and significantly deranged serum bile acid and ALT levels, with the bile acid pathway being implicated in the mechanism for developing coagulopathy in obstetric cholestasis. As the largest sample to date, our results add, in the form of patient numbers, to the findings of similar more recent studies, such as that by DeLeon et al.[6] Older studies with smaller patient numbers have reported a higher incidence of coagulopathy and the reasons for this are not clear.[3,4] Applying the 'rule of three' to our sample, we can be 95% confident that fewer than 1 in 152 parturients with obstetric cholestasis will have deranged clotting, but it is impossible to quantify how many of those would go on to develop a spinal or epidural haematoma if neuraxial blockade were performed in the presence of an undiagnosed coagulopathy. Risk is a continuum and the alternative anaesthetic options are not without their own risk of serious complications.

There are limitations to our data. It is a retrospective study, and there were challenges collating complete data sets and identifying treatment initiation. Determination of the true incidence of coagulopathy in obstetric cholestasis, and the subsequent demonstration of an acceptably low level of risk for a complication, would require a large prospective study. However, we believe that our results strengthen those of recent studies and that this information will add to the clinician's decision-making when weighing-up the risks and benefits