Postpartum pulmonary edema in twin parturient: beyond the fluids
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Pulmonary edema (PE) after postpartum hemorrhage (PPH) resuscitation is mainly due to fluid overload or transfusion-related acute lung injury. Here we present the case of a 30-year-old primigravida having uncomplicated twin pregnancy. She developed PPH and PE simultaneously during the early postpartum period. Chest radiography was inconclusive to exclude fluid overload. Echocardiography and lung ultrasound ruled out volume overload. PE could be due to adverse effects of drugs, or negative pressure from laryngospasm originating from incisional pain during uterine manipulation. Caution should be exercised while administering methylergometrine or carboprost for atonic PPH, with continued vigilance for detection of signs of PE, especially in high-risk pregnancy. Moreover, the risk versus benefit ratio should be considered for performing abdominal uterine massage as it may be more harmful than beneficial in such subset of patients.

Introduction
Postpartum pulmonary edema (PE) is a rare entity in healthy women, but it carries significant morbidity and mortality. Early detection and identification of the etiologies can lead to treatment in right direction and save the life. Cardiogenic PE is usually due to a chronic condition but can be acute in presentation. The onset of acute PE in a young, previously healthy postpartum patient, mainly directs towards a noncardiogenic origin. Here, we report a rare case of noncardiogenic PE during the postpartum hemorrhage (PPH) management. Written informed consent was taken from the patient before reporting the case.

Case presentation
A 30-year-old primigravida having a supervised and otherwise uneventful pregnancy was referred to us at term for management of twin pregnancy. She had no comorbidities. Her investigations were unremarkable, except for a platelet count of 49,300/mm³ (no bleeding) since the last 2 days without any significant cause. The patient then exhibited nonreassuring fetal heart rate and an emergency cesarean section was planned. Her airway examination revealed adequate mouth opening with modified Mallampati grade II. She was premedicated with intravenous metoclopramide 10 mg and ranitidine 50 mg before shifting to the operating theater. General anesthesia was chosen in view of obstetric emergency. Rapid sequence induction with cricoid pressure was conducted using intravenous thiopental 250 mg and succinylcholine 100 mg. The chest was clear on auscultation. Anesthesia was maintained with a combination of intravenous and inhaled anesthetics, including Fentanyl 75 mcg was administered intravenously after delivery of the second fetus. Oxytocin 40 U in the form of intravenous infusion was started as titration dosage. The 1.5 h surgery was uneventful with an expected blood loss of 1 l. A total volume of only 1.5 l of 0.9% saline was administered intraoperatively, without transfusion of blood products. Perioperatively, monitoring parameters, including urine output, were within normal limits. Neuromuscular paralysis was fully reversed with neostigmine–glycopyrrolate and the patient was uneventfully extubated with full consciousness without any residual anesthetic effect and 100% oxygen saturation in pulse oximetry. The patient had normal breathing pattern with regular chest movements during the immediate postoperative period on facemask oxygen. On performing vaginal toileting, fresh bleeding was observed and atonic PPH was suspected. The uterus felt flabby and per abdominal uterine massage was instituted. Fluids and blood products were again started. Intramuscular methylergometrine and intramuscular carboprost were administered.

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injected. A repeat dose of carboprost was injected and oxytocin infusion rate was increased. The uterus became harder. The patient then acutely developed a respiratory distress with hypotension (86/44 mmHg). On auscultation, air entry was reduced bilaterally with basal crepitations. Arterial blood gas analysis showed the following results: pH 7.36, PaCO₂ 42 mmHg, PaO₂ 55 mmHg, and HCO₃ 26 mmol/l on 100% oxygen mask. Radiograph of the chest revealed bilateral alveolar infiltrates suggestive of PE (Fig. 1). Fluids were then restricted, with a total infusion volume of 1 l colloids only (postoperatively). The patient was reintubated with the help of succinylcholine and midazolam. Immediately after intubation, endotracheal suctioning revealed pink frothy secretions. The secretions were so copious that they repeatedly filled up the endotracheal tube and were hard to clear by tracheal suctioning. Intravenous frusemide 20 mg was injected and the patient was shifted to ICU. She was put on mechanical ventilation. Central venous cannulation showed a central venous pressure of 10 mmHg. Echocardiography was performed and revealed the left ventricular end-diastolic diameter to be less than 5 cm and that of the inferior vena cava to be 1.3–1.8 cm (five readings) with normal hepatic veins (Fig. 2). Ejection fraction was 45–50%, with no organic lesions, or significant right ventricular dysfunction. Ultrasound of the chest revealed multiple interstitial B lines (Fig. 3). After 4 h of ventilation, the tracheal secretions remarkably reduced and the chest was clear on auscultation. A repeated chest radiography and ultrasound of the chest showed markedly cleared lung fields (Fig. 4). Arterial blood gas parameters improved. Postpartum urine output was adequate. The patient was extubated 2 h later and discharged from ICU after 24 h of observation. On follow-up consultations with cardiologist and pulmonologist, no organic cause was found.
Discussion
The incidence of acute PE in a parturient is 0.08% [1]. There is a long list for differential diagnosis of postpartum PE [2]. Negative pressure PE is known to occur in patients who generate enough inspiratory pressures against a closed glottis. Most cases are seen secondary to laryngospasm, which is common in pediatrics [3]. In our case, the etiology for PE could be due to laryngospasm provoked by pain [4]. Uterine massage and vigorous compression of freshly sutured anterior abdominal wall incites pain, which may lead to laryngospasm through vagus nerve stimulation (abdominolaryngeal reflex). This coupled with exaggerated inspiratory efforts might end up precipitating frank PE. Other possibilities may have been caused by the use of carboprost or methylergometrine (uterotonic) [5,6]. Administration of prostaglandin F$_2$α (PGF$_2$α) is known to increase pulmonary arterial pressure, which predisposes to fluid transudation across the pulmonary capillaries. Tocolytic drugs are more common offender for PE compared with uterotonic [7]. There are many drugs that cause PE, but drugs we used in this patient did not produce PE, except uterolytic agents. Twin pregnancy imposes greater physiological challenges to the mother when compared with single pregnancy. There is a greater expansion of plasma volume leading to reduced plasma oncotic pressure predisposing to edema. Interestingly, both etiologies of edema formation dramatically subside within 4–6 h of elective ventilation as was the case in our patient. Negative pressure PE due to painful stimulation in twin parturient has never been described in the literature. The authors hence suggest that caution be exercised while administering methylergometrine and carboprost for atonic PPH with continued vigilance for detection of signs of PE, especially in multiple gestation females. In addition, the risk versus benefit ratio should be considered before applying abdominal uterine massage as it may cause more harm than good in such subset of patients. Perioperative as well as postoperative ultrasound monitoring is a good idea for detection of PE with its underlying etiologies [8].

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Conflicts of interest
There are no conflicts of interest.

References