



Diagnosing Excessive Irrigation Fluid Intravasation During Operative Hysteroscopy: A Case Report

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Abstract

Excessive irrigation fluid intravasation is a rare but potentially life-threatening complication of operative hysteroscopy.

A 46-year-old woman underwent hysteroscopic endometrial polypectomy under general anesthesia using normal saline as the uterine distension medium. During surgery, an abrupt decrease in end-tidal carbon dioxide accompanied by progressive oxygen desaturation prompted a broad differential diagnosis, and subsequent arterial blood gas analysis revealed severe hyperchloremic metabolic acidosis and marked hemodilution. Integration of physiological changes, arterial blood gas abnormalities, and intraoperative findings led to the diagnosis of massive irrigation fluid intravasation, resulting in prompt termination of the hysteroscopic procedure. The patient recovered without major complications.

This case highlights the value of integrating physiological monitoring, arterial blood gas analysis, and intraoperative findings for timely diagnosis of excessive irrigation fluid intravasation during operative hysteroscopy.

Keywords: Hysteroscopy; Fluid Intravasation; Normal Saline; Pulmonary Edema

Introduction

Operative hysteroscopy is a widely performed minimally invasive procedure, but excessive irrigation fluid intravasation remains a potentially life-threatening complication [1]. Because its early physiological manifestations may overlap with those of other causes of acute intraoperative cardiopulmonary deterioration, early recognition may be delayed [2]. To date, the diagnostic approach to excessive fluid intravasation has received little attention.

Herein, we report a case of massive irrigation fluid intravasation during operative hysteroscopy in which physiological monitoring, arterial blood gas analysis, and intraoperative findings prompted diagnostic reassessment and appropriate management. Written informed consent for publication of this case report and the accompanying images was obtained from the patient.

Case Presentation

A 46-year-old woman (height, 162 cm; weight, 53.2 kg) with no significant medical history was scheduled for elective hysteroscopic endometrial polypectomy under general anesthesia. She was classified as American Society of Anesthesiologists (ASA) physical status I.

Anesthetic induction was performed using intravenous lidocaine (20 mg), propofol (70 mg), and rocuronium (30 mg). A size 3 laryngeal mask airway (LMA) was inserted uneventfully. Anesthesia was maintained with sevoflurane and a continuous remifentanyl infusion. Mechanical ventilation was initiated with a fraction of inspired oxygen (FiO₂) of 0.5, a tidal volume of 420 mL, a respiratory rate of 14 breaths/min, and a positive end-expiratory pressure (PEEP) of 0 cmH₂O. Initial vital signs remained stable, with an oxygen saturation of 100% and an end-tidal carbon dioxide (ETCO₂) of approximately 30 mmHg.

The hysteroscopic procedure was performed using normal saline as the uterine distension medium. Approximately 20 minutes after the procedure began, ETCO₂ began to decrease below 30 mmHg. Despite reducing the respiratory rate to 10 breaths/min, ETCO₂ continued to decline to 20 mmHg. During the same period, the peak airway pressure gradually increased from 11 to 21 cmH₂O, while the delivered tidal volume remained unchanged. Simultaneously, oxygen saturation gradually declined from 100% to 94% despite increasing the FiO₂ from 0.5 to 0.8. Heart rate and



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Table 1: Serial ABGAs and laboratory findings during intraoperative deterioration and early postoperative management.

	Initial Deterioration	During Resuscitation	Before Angiography	ICU admission	30 minutes after ICU admission	POD 1
pH	7.179	7.109	7.200	7.14	7.29	7.34
PaCO ₂ (mmHg)	27.9	32.4	34.9	58	36	33
HCO ₃ ⁻ (mmol/L)	10.5	10.4	13.8	19.7	17.3	17.8
Base Excess (mmol/L)	-15.6	-16.8	-12.4	-9.8	-8.5	-7.1
Hemoglobin (g/dL)	5.5	5.7	7.7	13.6	15.0	10.9
Sodium (mmol/L)	137	138	137	137	135	132
Potassium (mmol/L)	2.6	2.5	3.2	4.5	4.8	4.1
Chloride (mmol/L)	126	122	119	111*	not determined	106*

*Serum chloride values at ICU admission and POD 1 were obtained from concurrent serum chemistry testing.

blood pressure remained within normal limits throughout this period.

An immediate arterial blood gas analysis (ABGA) revealed severe hyperchloremic metabolic acidosis (pH 7.179, HCO₃⁻ 10.5 mmol/L, Cl⁻ 126 mmol/L) and a marked reduction in hemoglobin concentration (5.5 g/dL) (Table 1). Although pulse oximetry demonstrated progressive oxygen desaturation (SpO₂ decreased from 100% to 94%), arterial oxygenation remained preserved (PaO₂ 181.7 mmHg). The lactate concentration was within the normal range.

Given the abrupt decrease in ETCO₂ and progressive oxygen desaturation, venous gas embolism or pulmonary thromboembolism was initially suspected. The LMA was replaced with a 7.0-mm cuffed endotracheal tube using a video laryngoscope without difficulty. A central venous catheter was inserted into the right internal jugular vein under ultrasound guidance, and an arterial catheter was placed in the left radial artery for continuous hemodynamic monitoring and serial arterial blood gas analysis. Sodium bicarbonate infusion was initiated to correct the severe metabolic acidosis, a single intravenous bolus of phenylephrine (100 µg) was administered, and two units of packed red blood cells were transfused after review of the ABGA findings.

Despite these interventions, serial ABGAs demonstrated persistent metabolic derangement, prompting further discussion with the surgical team regarding the underlying cause. As the marked hemodilution raised concern for significant hemorrhage, hysteroscopic reassessment of the operative field was performed. Moderate active bleeding was observed. Pelvic ultrasonography demonstrated no massive intraperitoneal fluid collection, and subsequent laparoscopic exploration revealed only a small amount of ascites without significant hemoperitoneum. At this point, the irrigation fluid deficit was found to be approximately 7 L after a total of 10 L of normal saline had been used as the uterine distension medium. The marked irrigation fluid deficit, together with the absence of significant intraperitoneal bleeding, confirmed the diagnosis of massive irrigation fluid intravasation.

After intraoperative reassessment with transvaginal ultrasonography and laparoscopy, the patient was transferred to the angiography suite for empirical uterine artery embolization. Angiography demonstrated hypertrophied bilateral uterine arteries, and uterine artery embolization was performed (Figure 1). She was then admitted to the surgical intensive care unit for postoperative monitoring and supportive care.

The patient was successfully extubated approximately one hour after ICU admission once her vital signs and respiratory status

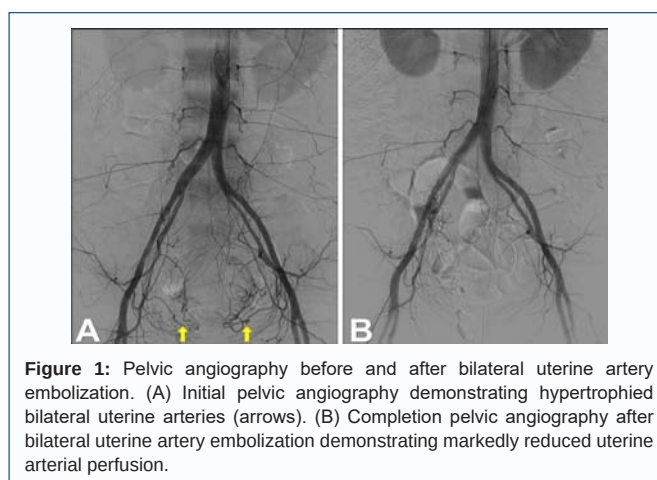


Figure 1: Pelvic angiography before and after bilateral uterine artery embolization. (A) Initial pelvic angiography demonstrating hypertrophied bilateral uterine arteries (arrows). (B) Completion pelvic angiography after bilateral uterine artery embolization demonstrating markedly reduced uterine arterial perfusion.

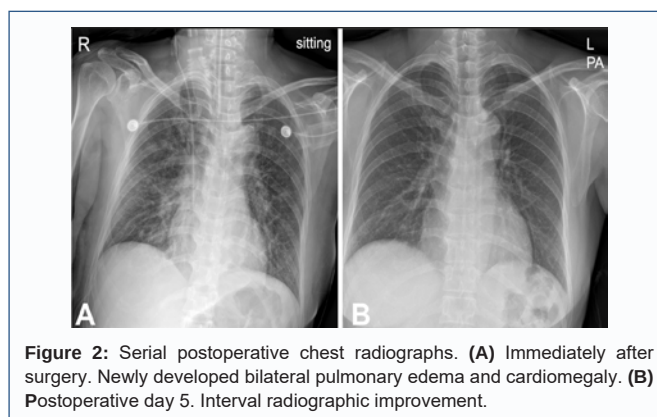


Figure 2: Serial postoperative chest radiographs. (A) Immediately after surgery. Newly developed bilateral pulmonary edema and cardiomegaly. (B) Postoperative day 5. Interval radiographic improvement.

had stabilized. Postoperative chest radiography demonstrated bilateral pulmonary edema, which resolved with supportive management during the postoperative course (Figure 2). Serial ABGAs demonstrated progressive correction of the metabolic acidosis and electrolyte abnormalities, accompanied by recovery of hemoglobin concentration. She was transferred to the general ward on postoperative day 1 and was discharged home on postoperative day 8 without further complications.

Discussion

Operative hysteroscopy is a safe and widely performed procedure for the diagnosis and treatment of intrauterine pathology. However, excessive fluid intravasation remains a rare but potentially life-threatening complication of operative hysteroscopy. Distension

media may enter the systemic circulation through disrupted endometrial or myometrial vessels and exposed venous sinuses when the intrauterine distension pressure exceeds the pressure within the venous circulation [1]. Clinical manifestations depend on the type and volume of the absorbed distension medium [3]. When isotonic distension media are used, fluid absorption primarily results in acute intravascular volume overload, which, in severe cases, may progress to pulmonary edema [4]. Risk factors for excessive fluid intravasation include high intrauterine distension pressure, prolonged operative time, deep myometrial penetration, low mean arterial pressure, and large uterine cavities. Accordingly, careful monitoring of fluid deficit throughout the procedure is essential, and current guidelines recommend terminating the procedure when the fluid deficit exceeds 2,500 mL for isotonic distension media in healthy women [1, 5]. Subsequent management is primarily supportive and includes respiratory care and correction of acute volume overload with diuretics [3, 6].

In the present case, unexpected intraoperative physiological deterioration prompted a stepwise differential diagnostic evaluation to identify its underlying cause. During general anesthesia, a sudden decrease in ETCO_2 accompanied by oxygen desaturation requires immediate evaluation because it may indicate a life-threatening intraoperative event. The differential diagnosis includes venous gas embolism, pulmonary thromboembolism, massive hemorrhage, anaphylaxis, and other causes of acute intraoperative deterioration [7]. Among these, both venous gas embolism and significant fluid intravasation should be considered in patients undergoing operative hysteroscopy who develop acute intraoperative cardiopulmonary deterioration [3, 8]. Venous gas embolism is one of the most serious complications, and its early manifestations include a sudden decrease in ETCO_2 , hypoxemia, and cardiovascular instability [9]. Although venous gas embolism and pulmonary thromboembolism were initially included in the differential diagnosis, the absence of significant cardiovascular instability made clinically significant embolic events less likely. Meanwhile, the progressive increase in airway pressure raised concern for evolving pulmonary pathology, prompting further diagnostic evaluation.

Further diagnostic evaluation with ABGA proved pivotal in establishing the diagnosis in our patient. The initial ABGA revealed a marked decrease in hemoglobin concentration, initially raising concern for significant hemorrhage. However, the degree of intraoperative bleeding was insufficient to explain this hemodilution. Concomitant severe hyperchloremic metabolic acidosis provided an additional clue. The Stewart approach provides a physicochemical framework for understanding metabolic acid–base disorders [10]. According to this approach, rapid intravascular absorption of large volumes of isotonic saline decreases the plasma strong ion difference, resulting in hyperchloremic metabolic acidosis. These laboratory abnormalities led to reassessment of the irrigation fluid deficit. Collectively, these findings were more consistent with excessive irrigation fluid intravasation than with hemorrhage alone.

Previous case reports have described pulmonary edema, hypoxemia, and hyperchloremic metabolic acidosis following excessive absorption of normal saline during operative hysteroscopy [11–13]. However, these reports mainly focused on the clinical manifestations and supportive management after diagnosis. Diagnosis may be challenging because its initial presentation can mimic other causes of intraoperative respiratory deterioration [14,

15]. Our case underscores the importance of careful correlation of physiological changes with laboratory results and intraoperative findings in recognizing excessive irrigation fluid intravasation. Timely communication between the anesthesiologist and surgeon is an important component of this diagnostic process and may facilitate timely interruption of the procedure, thereby limiting further irrigation fluid absorption and subsequent physiological deterioration.

Conclusion

This case shows that massive irrigation fluid intravasation during operative hysteroscopy may present as abrupt intraoperative cardiopulmonary deterioration under general anesthesia. Diagnosis depends on the integration of physiological monitoring, arterial blood gas analysis, and assessment of operative findings. Timely diagnostic reassessment and close communication between the anesthesiologist and surgeon facilitate appropriate management.

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