Retroperitoneal Hemorrhage following Ultrasound-guided Transvaginal Oocyte Retrieval: A Case Report

Raymond Pla¹, K. Gage Parr¹, Katharine L. Bumbarger¹, Eric Heinz¹, Geoffrey Ho¹, Sasmira Lalwani², David Frankfurter²

¹Department of Anesthesiology and Critical Care Medicine, School of Medicine and Health Sciences, George Washington University, Washington, DC, USA, ²Division of Reproductive Endocrinology, Fertility and IVF, School of Medicine and Health Sciences, George Washington University, Washington, DC, USA

ABSTRACT

Ultrasound-guided transvaginal retrieval of oocytes is a common and uncomplicated procedure done in ambulatory care centers as part of in vitro fertilization. However, it has a potential for serious, life-threatening complications. In our case report, we detail the case of a 39-year-old G1P0 undergoing ultrasound-guided transvaginal oocyte retrieval in an ambulatory care center who developed hypotension mid-procedure. As she was persistently hemodynamically unstable, she was transferred to the emergency department for further evaluation and ultimately had an exploratory laparotomy which revealed a large retroperitoneal hematoma. She required surgical control of hemostasis as well as a blood transfusion and was eventually discharged from hospital after a short convalescence.

Key words: Ambulatory surgery, hemorrhagic shock, in vitro fertilization, retroperitoneal hematoma

INTRODUCTION

Ultrasound-guided transvaginal oocyte retrieval is the preferred means for obtaining oocytes for in vitro fertilization (IVF). Although it is regarded as a reasonably safe procedure, complications including hemorrhage, pelvic organ injury, and infection have been reported. We present and discuss a rare case of a retroperitoneal hemorrhage following an ultrasound-guided transvaginal oocyte retrieval.

MATERIALS AND METHODS

A 38-year-old G1P0 with an obstetric history of an elective abortion at age 22 and an 18-month history of infertility presented to our center for evaluation and treatment. Her medical history was significant for beta-thalassemia trait, migraines, vertigo, and she was an ex-smoker. Her male partner was noted to have a surgically corrected urethral stricture and erectile dysfunction, and asthenox-terato-hypospermia. Infertility evaluation was otherwise unremarkable (normal ovarian reserve testing and hysterosalpingogram). She underwent four cycles of clomiphene citrate controlled ovarian hyperstimulation (COH) and intrauterine insemination without a pregnancy following which, they proceeded with IVF.

She was placed on a 2-week course of oral contraceptive pill before initiation of a GnRH antagonist COH protocol using 225 IU follitropin beta (Follistim, Merck Inc., Kenilworth, New Jersey) and 150 IU human menopausal gonadotropin (Menopur, Ferring Pharmaceuticals, Parsippany, New Jersey). Cetorelix acetate (Cetrotide, Merck Inc., Kenilworth, New Jersey) was used to prevent ovulation. Stimulation lasted 10 days, with a peak estradiol of 1832 and lead follicle of 19 mm, and recombinant ß-hCG (Ovidrel, Merck Inc.,
Kenilworth, New Jersey) was administered 36 h before oocyte retrieval to induce final oocyte maturity. Ultrasound-guided transvaginal oocyte retrieval was scheduled to be performed at an ambulatory care center.

Anesthesia was induced with midazolam 2 mg, propofol 80 mg, and fentanyl 75 mcg. She was kept on 100% FiO₂, and anesthesia was maintained with a propofol infusion during the procedure. Her pre-procedure blood pressure was 130/85 with a heart rate (HR) of 83. During the procedure, her blood pressure declined to the 80 s/40 s, prompting an increase in her intravenous fluid infusion rate and discontinuation of the propofol infusion. Multiple doses of phenylephrine boluses were given in increments of 100–200 mcg. As a result of these measures, her blood pressure modestly improved. She was examined for signs of an allergic reaction (i.e., chest rash and wheezes), but none were noted. A speculum examination revealed no cervical or vaginal bleeding and a transabdominal ultrasound demonstrated no free fluid. During the entirety of their course, no electrocardiogram changes were noted and the patient did not become tachycardic. A finger-stick glucose obtained at that time was 385, so Solu-Cortef was withheld given low degree of suspicion for Addison’s crisis.

As the patient began to awaken from sedation, she was noticeably lethargic but was able to move all four extremities and respond appropriately to commands. On arrival in the post-procedure care area, the patient’s blood pressure was 84/43 and her HR was 77. At that time, a second peripheral IV was inserted and the patient received a fluid bolus. After 3 L of crystalloid, the patient’s blood pressure improved to 100/57, but then subsequently declined to systolic pressures in the 70 s. At this point, the decision was made to transfer the patient to the emergency department (ED) for further work-up and treatment.

Our institution did not require informed consent for this case report as the case is not so unique as to be identifiable with reference to other public sources and does not contain protected health information.

RESULTS

On arrival in the ED, her blood pressure was 80/47 with a HR of 73. Her physical examination was notable for moderate distress, pallor, diaphoresis, with cold, clammy skin, and piloerection. Her abdomen was soft and non-distended. There was mild tenderness to palpation in the periumbilical region but no guarding, rigidity or rebound. Laboratories at that time showed a white cell count of 18,000, hemoglobin of 5.6, and hematocrit of 18.7. Abdominal ultrasound was repeated and showed free fluid in Morrison’s pouch, bilateral lower quadrants, and right upper quadrants estimated to be >500 cc.

A presumptive diagnosis of an acute hemoperitoneum led to an emergent laparoscopy which was converted into exploratory laparotomy. Intraoperatively, iSTAT showed a hemoglobin of 6.1. The patient subsequently received 2 U packed red blood cells and an additional 1.5 L crystalloid, which raised their hemoglobin to 8.5. She was found to have a large right pelvic retroperitoneal hematoma without signs of active hemorrhage as well as a small left-sided retroperitoneal hematoma. The right ovary was noted to be enlarged and friable with a surface laceration that was actively oozing. Once hemostasis was obtained and it was visually confirmed that neither retroperitoneal hematoma was expanding, the abdomen was closed, and the patient was admitted for further observation and convalescence. During the remainder of her hospitalization, she did not require any further operative intervention or blood transfusion.

DISCUSSION

Transvaginal ultrasound-guided oocyte retrieval for IVF was initially described in 1985. Since then, it has become a commonly performed ambulatory surgical procedure. It is considered safe and relatively simple to perform, safe, and patients tend to be generally young and healthy. However, the literature mentions several incidences of life-threatening complications that may arise.

Acute hemoperitoneum is rarely seen following an ultrasound-guided transvaginal oocyte retrieval. Aragona et al.[1] observed that over 12 years and 7098 transvaginal oocyte retrievals, only four patients (incidence of 0.06%) required a surgical intervention for peritoneal bleeding following oocyte retrieval. Each of the five cases of peritoneal bleeding was associated with abdominal pain and hemodynamic instability. In two of the patients, hemorrhage was observed 12 h post-retrieval; the other two, at 2 h post-retrieval. A 20-year retrospective analysis of 23,827 patients who underwent transvaginal ultrasound-guided oocyte retrieval was performed by Levi-Setti et al.[2] between 1996 and 2016. The authors reported a complication rate of 0.76% (96 patients). Seventy-one patients (0.56%) required hospitalization. A 2014 retrospective case series and pooled analysis of 32 published cases by Nouri et al.[3] estimated that the incidence of hemoperitoneum following transvaginal oocyte retrieval was 0.08%. They found that most patients with this complication were not overweight and concluded that lean patients with polycystic ovarian syndrome and, perhaps, coagulopathy were the only identifiable risk factors. The risk factors for post-retrieval hemorrhage postulated by Aragona et al.[1] were factor IX deficiency, ovarian necrotizing vasculitis, and anticoagulant therapy. Our case was notable for none of these risk factors.

Despite all this, it is not unusual to encounter some intra-abdominal bleeding following transvaginal oocyte retrieval. Dessole et al.[4] estimated that in non-coagulopathic, normal weight women who were not taking anticoagulants,
the normal blood loss following an uncomplicated ultrasound-guided transvaginal oocyte retrieval was 230 ml at 24 h after procedure. Further, the authors found no correlation between the amount of blood loss and the number of follicles aspirated, the number of oocytes collected, the duration of the procedure, or pre-ovulatory E2 levels. Finally, they went on to conclude that when a complication such as acute abdomen occurs after transvaginal oocyte retrieval and the blood loss is not greater than normal, hemoperitoneum may be reasonably excluded. Such diagnoses as adnexal torsion, tubo-ovarian abscess, or ruptured endometrioma should be considered more likely.

A 2007 case report by Moayeri et al.[5] described a 34-year-old woman that underwent successive ultrasound-guided oocyte retrievals; each complicated by a 1500–2200 ml blood loss. The authors noted that she had screened positive for anticardiolipin IgG during her pre-IVF evaluation. Before her first retrieval, she was treated with a combination of heparin, low-dose aspirin, steroids, and intravenous immunoglobulin. Heparin and baby aspirin were stopped 2 days before the oocyte retrieval. Before the second egg retrieval, her coagulation studies were normal and pre-treatment was similar to the first cycle but lacked heparin. The second procedure was marked by post-procedure syncope, hypotension, and tachycardia. Emergent laparotomy revealed 1500 ml hemoperitoneum. Subsequent complex work-up for coagulopathy revealed Von Willebrand disease which was likely exacerbated by the use of aspirin. Again, the present case was not noted for any coagulopathy.

Azem et al.[6] reported a case of massive retroperitoneal hemorrhage following ultrasound-guided transvaginal oocyte retrieval requiring emergent exploratory laparotomy. In that case, no hypotension was noted during or immediately following the procedure. However, the patient complained of severe abdominal pain and tenesmus. Physical examination revealed no abnormal findings. She was prescribed diclofenac suppositories, and discharged home once her pain subsided. She represented to the hospital 10 h following oocyte retrieval with abdominal pain, vomiting, skin pallor, tenderness, and rebound. Physical examination revealed vaginal tenderness but no hypotension. Transvaginal ultrasound revealed a small collection of fluid. A retroperitoneal hematoma was discovered on laparotomy and described as “massive” and originating from the mid-sacral vein.

A review of the literature conducted by El-Shawar by et al.[7] noted that the risk of injury can be minimized by modifying technical aspects of the procedure, such as ensuring precise visualization of the vessels with ultrasound before advancement of the needle. In addition, minimizing lateral movement of the needle in the vaginal wall prevents vaginal tearing. Other technical aspects of the procedure such as aspirating all follicles with a single puncture of the ovary appear to reduce the risk of vaginal hemorrhage, as well. They commented that direct injury to such pelvic structures as the iliac vessels, the uterus, bladder, or colon can be avoided by visualizing structures in both the longitudinal and transverse axis before puncture to distinguish between follicles and iliac vessels.

Massive retroperitoneal bleeding can occur as a result from injury to the mid-sacral vein. Battaglia et al.[8] reported a case of severe hemoperitoneum following transvaginal oocyte retrieval requiring exploratory laparotomy. However, no injury to surrounding vessels was noted. Retrospectively, the patient was found to have a preexisting factor XI deficit. While the current patient had no coagulopathy and was not receiving anticoagulants, it is possible that an unrecognized mid-sacral vein injury occurred at the time of the retrieval. A timely recognition of a hemorrhagic complication lead to a full recovery. The lack of sonographic evidence of a hemoperitoneum in a case of post transvaginal oocyte retrieval hemorrhage should raise an alarm for a sacral vein injury and a retroperitoneal hemorrhage. Timely recognition of such a complication could allow for treatment through interventional radiology instead of surgery.

On review of the published literature, it seems clear that severe hemoperitoneum after transvaginal oocyte retrieval is a rare event. However, because of its life-threatening presentation, providers should be aware of the potential for such a complication. The conflicting studies make definitive establishment of risk factors, perhaps with the exception of coagulopathy, difficult. What seems reasonably clear is that patients who undergo transvaginal ultrasound-guided oocyte retrieval and develop a combination of abdominal discomfort, hypotension, tachycardia, or vomiting within 12–24 h of the procedure should undergo a prompt evaluation including complete blood count and ultrasound imaging to determine the appropriate course of action. This may include resuscitation/observation, surgery, or interventional radiologic embolization. It should also be stressed that evidence of hemorrhage in the absence of a hemoperitoneum should prompt evaluation for a retroperitoneal bleed.

CONCLUSION

Although hemorrhage after ultrasound-guided transvaginal oocyte retrieval is rare, anesthesiology providers should be aware that it may present with life-threatening complications. Further, evidence of hemorrhage in the absence of hemoperitoneum should prompt evaluation for a retroperitoneal bleed.

REFERENCES
