Anesthetic Considerations for Severe Ovarian Hyperstimulation Syndrome

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Severe ovarian hyperstimulation syndrome (OHSS) due to exogenous gonadotropin administration is a life-threatening condition associated with ascites, pleural effusions, oliguria, electrolyte abnormalities, hemocencentration, hypercoagulability, and hypotension.1

We present the first recorded case of anesthetic management for the patient with severe ovarian hyperstimulation syndrome.

CASE REPORT

A 39-yr-old woman who had previously been in good general health and who was diagnosed with primary infertility underwent ovarian stimulation, in vitro fertilization, and embryo transfer. After follicle-stimulating hormone and luteinizing hormone administration, serum estradiol concentrations reached 7,280 pg/ml, a very high level compared to the 1,500-2,000 pg/ml level that is frequently sought. Ten thousand international units of human chorionic gonadotropin (HCG) was given and 40 oocytes were retrieved, of which 28 were fertilized and six were transferred to the patient. Development of nausea, vomiting, and abdominal pain on the day of embryo transfer (3 days after HCG administration) prompted admission to the hospital (table 1). Initial treatment consisted of bed rest and 50 g albumin intravenously (iv) every 12 h. On hospital day 2, the patient developed abdominal distension and polythecymia. Fluid intake was restricted to intravenous fluids administered at 50 ml/h, and 25 mg indomethacin by mouth was begun. The next day she complained of dyspnea, worsening of her abdominal pain, and vomiting. Physical examination demonstrated increased abdominal girth, absent breath sounds over the lower half of both posterior lung fields, and an increase in hematocrit to 68%. Hematocrit decreased to 43%, and furosemide was added to the regimen.

Over the next 8 days, abdominal distension and dyspnea worsened; her weight increased by 9 kg; and pitting edema developed over the sacrum and the lower extremities. Beta HCG levels increased, indicating pregnancy. Renal failure developed as BUN and creatinine increased to 65 mg/dl and 2.0 mg/dl, respectively. Dopamine was started at a dose of 2 μg·kg⁻¹·min⁻¹ in an attempt to increase renal blood flow. Despite this, urine output decreased to less than 10 ml/h, and creatinine clearance decreased to 10 ml/min. Chest x-rays had not been obtained, but arterial oxygen tension during breathing of supplemental oxygen was 119 mmHg.

To prevent irreversible renal damage, termination of pregnancy by suction dilatation and curettage was deemed necessary. On hospital day 12, she was brought to the operating room. By this point, her normal weight of 54.5 kg had increased to 63.6 kg. Blood pressure was 110/70 mmHg, pulse was 80 beats per min, and respiratory rate was 20 breaths per min. Serum sodium was 128 mEq/l, potassium was 4.1 mEq/l, BUN was 65 mg/dl, creatinine was 2.0 mg/dl, hemoglobin was 11.5 g/dl, and hematocrit was 32%. On arrival in the operating room, an iv catheter was in place and working well. Monitoring with an automated blood pressure cuff, ECG, apical stethoscope, pulse oximeter (SpO₂), and mass spectrometer was established. While the patient breathed 100% oxygen via mask, anesthesia was induced with 1 mg midazolam and 100 mg ketamine, and maintained with 1 mg midazolam and 50 mg ketamine. She remained hemodynamically stable with an SpO₂ of 99-100%. Spontaneous ventilation was continued throughout the procedure, and blood loss was estimated at 200 ml. After the 10-min operation, she was taken to the recovery room fully conscious but disoriented. After an uncomplicated 2-h recovery room stay, she was fully alert and oriented. On return to her hospital room, spontaneous diuresis of large volumes of urine ensued. Over the next few days, serum potassium decreased to a nadir of 2.9 mEq/l, and she was treated with oral potassium supplements. Otherwise, the electrolyte abnormalities, ascites, and pleural effusions resolved uneventfully. All parameters returned to normal by the sixth postoperative week.

DISCUSSION

Ovarian hyperstimulation syndrome following ovulation induction with HCG is clinically evident in 11% of cases and identifiable by ultrasound in 44% of cases.2 Between 0.4 and 4% of treatment cycles will result in development of severe OHSS.3,4 The pathophysiology of OHSS represents an entire spectrum of abnormalities. The most common form is called mild OHSS. It consists solely of elevated estrogen and progesterone concentrations but lacks significant clinical signs and symptoms. This form is self-limiting and usually resolves with the onset of menses. Moderate OHSS is recognized clinically by abdominal distension, nausea, vomiting, diarrhea, ovarian

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enlargement of less than 12 cm, and weight gain. In the absence of conception, symptoms respond well to bed rest for 2 or 3 weeks. Those who become pregnant may progress rapidly to severe OHSS that consists of the above symptoms as well as massive ovarian enlargement beyond 12 cm, ascites, pleural effusions, hemococoncentration, coagulation abnormalities, electrolyte imbalances, hypovolemia, oliguria, or shock. Signs and symptoms frequently become manifest between 5 and 10 days following HCG administration.¹

Augmented endogenous HCG production from successful conception precipitates further deterioration. Hyperstimulated patients demonstrate progesterone metabolite concentrations that are usually found in the second or third trimester of pregnancy. The basic pathophysiology has been ascribed to ovarian follicle production of prostaglandins² and the local ovarian influence of the renin–angiotensin system.³ Both prostaglandins and the renin–angiotensin system increase capillary permeability. These processes allow for edema and fluid accumulations resulting in ovarian enlargement, ascites, hydrothorax, and anasarca.¹ An associated case of adult respiratory distress syndrome, in which capillary leak occurred, was also reported.² The rapidity of fluid shifts may result in intravascular hypovolemia, hemococoncentration, oliguria, renal damage, and electrolyte abnormalities. Hemoconcentration predisposes to increased blood viscosity, thrombosis, and thromboembolic phenomena. There also exists at least one well-documented case of profound liver dysfunction accompanying severe OHSS.⁶

Nonsteroidal antiinflammatory agents such as indomethacin,² which block prostaglandin synthesis, and histamine (H-1) receptor blockers⁵ may help the underlying problem of increased capillary permeability.

Intraoperatively, it was assumed that ascites, pleural effusions, and the lithotomy position would predispose to hypoxemia. To prevent this potential problem, an anesthetic technique allowing for high inspired oxygen concentrations was used.

The basic pathophysiologic abnormality is one of capillary leak in which large amounts of intravascular volume are lost to the third spaces. Diuretic therapy further exacerbates the intravascular hypovolemia. Consequently, ketamine was selected to minimize the risk of hypotension on induction. The brevity of her surgical procedure also allowed the use of ketamine as a maintenance agent. Fluid fluxes represent relative indications for invasive intravascular volume monitoring of central venous pressures (CVP) or pulmonary artery pressures. In this case, fluid changes were anticipated to be minimal in the immediate perioperative period, thereby obviating the need for CVP monitoring. In the absence of congestive heart failure, cardiac valvular disease, and pulmonary hypertension, there was no need for a pulmonary artery catheter.

A history of recent food ingestion, vomiting, uremia, or ascites are indications for protecting the airway with an endotracheal tube during general anesthesia. Despite the emergency nature of this case, the patient had received nothing by mouth over the preceding 8 h. Nausea and vomiting had dissipated 5 days before anesthesia. Although renal failure was progressive, function had not yet deteriorated to a state of uremia. Ascites represents a controversial indication for endotracheal intubation. Because this patient’s ascites was neither massive nor tense, two of the authors (H.T. and H.R.) believed that the risk of vomiting and aspiration was not increased. Consequently, without a specific requirement for tracheal intubation, the patient was allowed to breath spontaneously via a mask.

The rapidly expanding use of in vitro fertilization and gamete intrafallopian transfer will predictably result in large numbers of women receiving HCG to induce ovu-
lation. Between 0.4 and 4% of treatment cycles will result in severe OHSS. Some of these patients will require surgery and anesthetic management for termination of pregnancy or laparotomy. Therefore, it becomes important for the anesthesiologist to understand the related pathophysiology and potential treatment modalities for severe OHSS.

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Pneumoencephalos after Inadvertent Intrathecal Air Injection during Epidural Block

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The “loss-of-resistance” technique is routinely employed for identification of the epidural space. During performance of this test, air may be injected along the epidural needle tract and into the epidural space.1 We report a case in which a loss-of-resistance test used in the presence of unnoticed dural puncture resulted in a severe transient neurologic event. If a computerized tomographic (CT) brain scan had not been performed, the neurologic symptoms might have been attributed to another complication—prolonged neurotoxicity of bupivacaine hydrochloride following total spinal anesthesia—which also occurred during the administration of this epidural block.

CASE REPORT

A healthy, 25-yr-old primigravida was anesthetized with epidural block for cesarean section. Using a 16-G Tuohy needle, the epidural space was identified, after many attempts, by a loss-of-resistance test and subsequent repeated injections of air (total amount, approximately 20 ml) for further confirmation of needle localization. Sixteen milliliters of bupivacaine hydrochloride 0.5% with epinephrine 1:200,000 were slowly injected and were followed immediately by cessation of respiration and a decrease in blood pressure. After tracheal intubation, the patient’s lungs were mechanically ventilated with 100% oxygen. 2,000 ml saline supplemented with 50 mg epinephrine were administered intravenously, and blood pressure rapidly increased to preanesthetic values. An infant with normal Apgar scores was delivered, after which anesthesia was maintained using 10 mg diazepam, 0.3 mg fentanyl, and a mixture of 66% N2O in oxygen. At the end of the operation, the patient was transferred to the recovery room, and mechanical ventilation was continued until the effects of the total spinal anesthesia were expected to subside. Four hours later, spontaneous breathing resumed and involuntary movements began. The patient, however, remained drowsy and stuporous. Since her condition did not improve over the following hours, she was referred to our hospital for neurologic evaluation.

A CT brain scan revealed a large subarachnoid air-filled cavity located mainly at the parietofrontal cerebral cortex region with an estimated volume of 25 ml. The ventricles were not displaced and appeared normal (fig. I). The patient was admitted to the neurosurgical intensive care unit (ICU) for continuous monitoring. The next day, her neurologic status improved, and she was able to sit, talk, and move freely. A repeat CT scan did not show any residual air within the subarachnoid space, and the patient was discharged from the neurologic ICU on the third day postpartum.

DISCUSSION

The clinical symptoms presented by the patient could have been attributed, if not correctly diagnosed, to pro-