THE JOURNAL OF OBSTETRICS AND GYNECOLOGY OF INDIA

The Journal of Obstetrics and Gynecology of India (March–April 2016) 66(2):131–133
DOI 10.1007/s13224-015-0715-8

CASE REPORT

Critical Ovarian Hyperstimulation Syndrome and Management

Shruti Sharma[1] · Parshotam Lal Gautam[1] · Tarandeep Singh[1] · Rabinder Singh Sobti[1] · Jaswinder Singh Sandhu[2] · Suman Puri[3]

Received: 11 May 2015 / Accepted: 18 May 2015 / Published online: 7 October 2015

About the Author

Dr. Shruti Sharma is working as full time intensivist at the reputed tertiary care teaching institution since 10 years. She completed her DNB in Anaesthesia and later successfully completed Indian Diploma of Critical Care Medicine. Intensive care is her chief area of interest. She has been involved in many research projects. She also completed her observership in Infection Control at Detroit Medical Center, USA. She is always keen to learn new skills and improve her knowledge in fields related to intensive care

Introduction

Ovarian hyperstimulation syndrome (OHSS) is a complication that occurs in the luteal phase of an induced hormonal cycle. In most cases, the symptoms are self-limited, and spontaneous regression occurs [1]. Mild manifestations of OHSS include transient lower abdominal discomfort, nausea, vomiting, diarrhea, and abdominal distension. Life-threatening OHSS can progress to adult respiratory distress syndrome, renal failure, hepatic injury, hemostatic imbalances, and thromboembolic episodes [1, 2]. We report here a challenging case of critical OHSS and its management in a critical care unit.

Case Report

A 27-year-old female was admitted to emergency with history of abdominal distension, breathlessness, and decreased urine output. Patient was undergoing in vitro fertilization (IVF) at a private center where she had received first cycle of controlled ovarian stimulation and oocyte retrieval. Patient had already been started on gonadotropin

Dr. Shruti Sharma is a Intensivist in the Department of Critical Care; Dr. Parshotam Lal Gautam is Professor and Head in the Department of Critical Care; Dr. Tarandeep Singh is Assistant Professor in the Department of Critical Care; Dr. Rabinder Singh Sobti is Assistant Professor in the Department of Critical Care; Dr. Jaswinder Singh Sandhu is Professor in the Department of Nephrology; Dr. Suman Puri is Professor in the Department of Obstetrics and Gynaecology, Dayanand Medical College and Hospital.

✉ Shruti Sharma
shrutisharmadr@yahoo.co.in

[1] Department of Critical Care, Dayanand Medical College and Hospital, Ludhiana, Punjab, India

[2] Department of Nephrology, Dayanand Medical College and Hospital, Ludhiana, Punjab, India

[3] Department of Obstetrics and Gynaecology, Dayanand Medical College and Hospital, Ludhiana, Punjab, India

RH (GnRH) antagonist Cetrolix 0.25 mg subcutaneously OD, cabergoline 0.5 mg BD, and intravenous albumin therapy to suppress the symptoms of OHSS. At the time of presentation to emergency unit, the patient was conscious, oriented, and able to communicate. Her vitals were respiratory rate: 20–26/min, heart rate: 110/min, and blood pressure: 80/50 mmHg. Laboratory investigations showed deranged renal function tests (S. creatinine: 9.0 mg/dl, blood urea: 158 mg/dl, Serum potassium: 5.5 meq/l), WBC: 12,200/mm^3, hemoglobin: 12.0 gm %, hematocrit: 38 %, and platelet count: 271,000/mm^3 at admission. Arterial blood gases (ABGs) showed compensated metabolic acidosis with pH: 7.38, pCO_2: 20 mmHg, pO_2: 73 mmHg, lactate: 2.1 mmol/L, and bicarbonate: 11.8 mmol/L. Coagulation profile, erythrocyte sedimentation rate (ESR), and liver function tests were normal. Blood estradiol was 11,000 pg/ml and βHCG: 9.81mIU/ml. X-ray chest showed bilateral pulmonary infiltrates suggestive of pulmonary edema and bilateral costo-phrenic angle blunting. Ultrasound abdomen and lung showed large multilobulated, multicystic ovaries with moderate ascitis and bilateral mild pleural effusion. Her central venous pressure (CVP) was 20 cm of normal saline (NS). She was dialyzed in emergency unit and was kept on noninvasive ventilation to improve her oxygenation and ventilation. Her mean arterial pressure was maintained above 65 mmHg on low dose of vasopressors (nor-epinephrine). Patient was shifted to medical intensive care unit. Sustained low efficiency dialysis (SLED) was done daily over the next 3 days. Her serum creatinine improved from the initial 9 to 4.6 mg/dl, and CVP was around 15 cm of normal saline. Abdominal paracentesis was done twice to relieve the symptoms of respiratory distress, and the fluid was transudative. On the fifth day of ICU admission, she had respiratory distress and desaturation. She was intubated and put on volume control ventilatory settings. Her CVP was 28 cm NS at this time. It was difficult to maintain her saturation above 75 % despite the best ventilatory settings. A jugular dialysis catheter was inserted, and SLED was started. Her cardiac work-up in the form of shortness of breath profile (SOB), echocardiography and ECG were normal. Thirty minutes into the SLED, patient had a cardiac arrest, from which she was revived after about 45 min of cardiopulmonary resuscitation (CPR). Femoral arterial invasive BP monitoring (IBP) was started. Further plan was to prevent capillary leak and pulmonary edema through SLED and to maintain intravascular volume. For achieving this, we were aided by CVP, IBP, stroke volume variation, and cardiac output monitoring through a Flotrac device—EV 1000. Anti-HCG treatment, gonadotropin antagonists, and other supportive measures were continued. Some of the ABGs are shown in Table 1.

Patient had a low Glasgow Coma Scale (GCS) score and 2D MRI showed findings consistent with mild hypoxic damage. Over the next few days, pO_2 improved, CVP decreased, and Flotrac readings were consistent with good cardiac index and other derived parameters. Bedside percutaneous tracheostomy was done to facilitate weaning. The patient started producing good urine output after 2 weeks of intermittent dialysis. GCS gradually improved, and patient was eventually weaned off the ventilator after 3 weeks. She suffered from nosocomial infection in the form of ventilator-associated pneumonia (VAP) in the second week of admission to ICU. Tracheal culture grew *pseudomonas aeruginosa* for which she was treated with Carbapenems according to sensitivity reports. After 6 weeks of ICU stay, patient had fever and loose stools. Stool examination was positive for *Clostridium difficile*. Antibiotics were stopped, and she was started on Tab Metronidazole for 10 days through Ryle's tube. At 8 weeks, patient was fully conscious, and after tracheal decanulation, patient was discharged in satisfactory condition. Patient is on regular follow-up in our outpatient department and does not have any neurological deficit or cognitive impairment except for a little time lag while speaking.

Discussion

First fatal case of OHSS with renal failure was described in 1951. Although the incidence of severe OHSS may be underreported at 0.5–0.96 %, it can be life threatening [2]. OHSS usually results from stimulation of the ovaries by gonadotropins and the administration of exogenous hCG. Vasoactive substances that are secreted upon ovarian stimulation cause an increase of vascular permeability and third spacing, leading to hemoconcentration and inadequate end organ perfusion [2]. OHSS can be classified as mild (abdominal distension and discomfort), moderate (ascites revealed only by ultrasound), severe (ascites revealed by physical examination, or the presence of pleural or pericardial effusion associated with hemoconcentration), and critical (the symptoms described above are accompanied by hypotension, acute renal failure, and thromboembolic disorders) [3]. Preventive measures to decrease the risk of OHSS are coasting (withholding gonadotropin stimulation and continuing the agonist suppression until estrogen levels declines to acceptable levels), GnRH antagonists, and intravenous albumin therapy, etc. A dopamine agonist cabergoline started on the day of hCG injection has also been shown to reduce the risk of severe OHSS [4]. A meta-analysis of five trials demonstrated a significant reduction in severe OHSS on administration of albumin at the time of oocyte retrieval in high-risk patients [5]. Renal failure, thromboembolism, pericardial effusion, and adult respiratory distress syndrome are potential life-threatening

Table 1 ABGs of the patient at the time of arrest

Time (h)	pH	pCO (mmHg)	pO_2 (mmHg)	Lactate (mmol/L)	Bicarb (mmol/L)	SpO_2 %	Situation
09.00	7.15	45	50	10	12.4	68	Post intubation
09.55	6.94	73	52	10.7	15.7	70	Post arrest
17.21	7.22	35	58	4.6	14.3	83	Post SLED
22.08	7.21	40	62	3.4	16	85	

complications of severe OHSS. Management of our patient was challenging because the patient had renal failure, respiratory failure, fluid overload, ascitis, pleural effusion, nosocomial infections, hypoxic brain injury, and poor GCS. The patient had life-threatening OHSS which required aggressive treatment including cardiac output and stroke volume variation monitoring to decide about the intravascular volume status. Our case report also raises a commonly asked question that how long should the duration of CPR be. Goldberger et al. found that resuscitation efforts were terminated within 10 min in 15.8 % of the patients, and within 30 min in 76.6 % of the nonsurvivors. The author concluded that generally continuing CPR for a longer period may increase the likelihood of survival [6]. There are a few case reports where patients had neurological recovery after prolonged CPR [7, 8]. The physicians and intensivists should be familiar with the different clinical manifestations of OHSS and their management.

Compliance with Ethical Standards and Conflict of Interest This article does not contain any studies with human participants or animals performed by any of the authors. The authors declare no conflict of interest.

References

1. Junqueira JJM, Bammann RH, Terra RM, et al. Pleural effusion following ovarian hyperstimulation. J Bras Pneumol. 2012;38(3):400–3.
2. Mullin CM, Fino ME, Reh A, et al. Symptomatic isolated pleural effusion as an atypical presentation of ovarian hyperstimulation syndrome. Case Rep Obstet Gynecol. 2011;011:967849. doi: 10.1155/2011/967849. Epub 2011 Aug 7. http://www.hindawi.com/journals/criog/2011/967849/. Accessed 28 April 2015.
3. Roden S, Juvin K, Homasson JP, et al. An uncommon etiology of isolated pleural effusion. The ovarian hyperstimulation syndrome. Chest. 2000;118(1):256–8.
4. Alvarez C, Martí-Bonmatí L, Novella-Maestre E, et al. Dopamine agonist cabergoline reduces hemoconcentration and ascites in hyperstimulated women undergoing assisted reproduction. J Clin Endocrinol Metab. 2007;92:2931–7.
5. Venetis CA, Kolibianakis EM, Toulis KA, et al. Intravenous albumin administration for the prevention of severe ovarian hyperstimulation syndrome: a systematic review and metaanalysis. Fertil Steril. 2011;95(1):188–96.
6. Goldberger ZD, Chan PS, Berg RA, et al. Duration of resuscitation efforts and survival after in-hospital cardiac arrest: an observational study. Lancet. 2012;380(9852):1473–81.
7. Brown RE, Surapaneni S, Chevli P, et al. Survival with complete neurological recovery after prolonged resuscitation. Internet J Cardiol. 2015;11(1). https://ispub.com/IJC/11/1/1532. Accessed 28 April 2015.
8. Cohen L, Kaur S. Duration of CPR: how long is too long? A positive outcome after 90 minutes of CPR. (2012). Anesthesiology Publications and Presentations. Paper 137. http://escholarship.umassmed.edu/anesthesiology_pubs/. Accessed 28 April 2015.