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Case Report

Fatal cardiac tamponade following cytoreductive surgery for serous carcinoma of the ovary

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1. Introduction

There were an estimated 21,980 women who received a new diagnosis of ovarian cancer in 2014, over half of whom had distant metastases at presentation (http://www.cancer.gov). Ovarian carcinoma has a well-described pattern of metastasis, including intraperitoneal spread through epithelial sloughing of tumor cells as well as retroperitoneal spread through the lymphatics. It is uncommon for female genital malignancies to spread above the diaphragm, especially early in the disease process. In particular, it has been estimated that only 2.4% of patients with epithelial ovarian carcinoma will develop pericardial involvement (Dauplat et al., 1987). A PubMed search revealed 20 reports of pericardial effusion associated with ovarian carcinoma, most commonly presenting as recurrent disease and much more rarely as part of initial diagnosis (Blich et al., 2007). There were no reports, however, of immediate postoperative pericardial effusion associated with gynecologic malignancy. We report a case of cardiac tamponade diagnosed postmortem on postoperative day one after primary debulking for serous ovarian cancer.

2. Case

A 44-year-old G3P3 with no family history of malignancy presented to the emergency room with the complaint of two weeks of abdominal pain and distension. She had no significant past medical or surgical history. Computed tomography of the chest, abdomen, and pelvis showed marked abdominal ascites, omental caking, bilateral adnexal masses measuring up to 13 cm in diameter, and a large right-sided pleural effusion. There was no evidence of pleural effusion on the left, chest lymphadenopathy, or pericardial effusion. At her preoperative appointment several days later, vital signs were within normal limits and she reported no chest pain or shortness of breath. Given the absence of lung metastases or liver parenchymal disease on imaging, it was felt she could be optimally cytoreduced. Neoadjuvant chemotherapy was considered as an alternative option, but given her young age, high functional status, and pre-operative imaging suggesting optimal cytoreduction was possible, after discussion with the patient, the decision was made to proceed with surgery.

Six days later, she underwent exploratory laparotomy, total abdominal hysterectomy, bilateral salpingo-oophorectomy, bilateral pelvic and para-aortic lymphadenectomy, omentectomy, appendectomy, and intra-abdominal tumor debulking. Intraoperatively, 7.5 l of straw-colored ascites were drained upon entry into the peritoneal cavity. There were bilateral adnexal masses (15 cm on right, 7 cm on left) and the uterus was mildly enlarged. Tumor extended into the cul-de-sac and onto the serosa of the sigmoid colon, and this was completely excised followed by oversewing of an area of denuded sigmoid serosa. The omentum was noted to have mild nodularity but no large caking. There was a questionable palpable nodule on the surface of the right diaphragm but no gross disease, and the remainder of the upper abdomen appeared to be free of disease. The patient was considered to be optimally cytoreduced at the completion of the procedure, with no gross residual disease. The patient received 6 l of cystalloid, 1 l of albumin, and 1 unit of packed red cells during the surgery. Estimated blood loss of 500 ml, and urine output for the case was 300 ml. She remained hemodynamically stable throughout the surgery and immediate recovery period, and she required no pharmacologic pressor support.

Pathologic examination revealed high-grade serous carcinoma with involvement of both ovaries, uterine serosa, omentum, and cul-de-sac, consistent with FIGO stage IIIB. Five para-aortic and 21 pelvic lymph nodes were all negative for carcinoma.

The patient was extubated immediately postoperatively and taken to the ICU for recovery as is standard at our hospital for extensive abdominal surgery. Postoperative chest x-ray showed pulmonary venous congestion, interstitial edema, and a right pleural effusion. On the morning of postoperative day one, the patient appeared stable with no chest

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pain or shortness of breath; however her heart rate began to increase from the 100 s to 130 s with a supraventricular tachycardia rhythm. Her blood pressure also gradually declined, oxygen saturation decreased, and respiratory rate increased. Her heart rate increased to 160 s, and oxygen saturation failed to improve beyond 93% despite non-rebreather mask of 100% oxygen. An EKG showed supraventricular tachycardia with an S in lead I, Q in lead III, and T in lead III with incomplete right bundle branch block. Adenosine, a calcium channel blocker, and digoxin were administered in an attempt to decrease heart rate without effect. CT pulmonary angiogram was ordered to assess for pulmonary embolism, and albumin was given for intravascular fluid resuscitation. Before the CT could be performed, the patient developed bradycardia and then asystole. Cardiopulmonary resuscitation was immediately initiated, but she never resumed cardiac activity and she was declared dead 20 min later.

Autopsy showed a pericardial effusion of 400 ml of serosanguinous fluid, with no metastatic carcinoma identified in the fluid or pericardium. Also seen were bilateral pleural effusions (450 ml on right, 250 ml on left) and a 0.2 cm deposit of metastatic carcinoma on the peritoneum overlying the right diaphragm. There was no evidence of pulmonary embolism, and autopsy was otherwise unremarkable.

3. Discussion

Our case represents a rare postoperative complication following primary debulking surgery for ovarian cancer. A PubMed literature review found 20 reports describing pericardial effusion in patients with ovarian carcinoma, with only 4/20 with effusion apparent at initial cancer diagnosis (Forslund et al., 1991; Arbol Linde et al., 1998). Pericardial effusion has also been described in patients with vaginal, endometrial, and cervical cancer (Rudoff et al., 1989), most commonly diagnosed remote from initial cancer diagnosis as a manifestation of recurrent disease. Postoperative cardiac tamponade has been reported in other fields, particularly following interventions with close proximity to the pericardium (Paz et al., 2011), but to the best of our knowledge, there has been no prior report of cardiac tamponade presenting immediately following cytoreductive surgery for ovarian carcinoma.

Though rare, cardiac tamponade must be considered in the differential diagnosis of hemodynamic instability in patients with gynecologic malignancy. Timely diagnosis can facilitate a relatively simple, life-saving intervention. Fluid accumulation in the pericardial space may be rapid or indolent, and may become clinically apparent only when a critical amount of fluid is reached, overwhelming the body's compensatory mechanisms (Spodick, 2003). The patient may complain of dyspnea, chest pain, orthopnea, or weakness, however most are asymptomatic, and “classic” findings of pulsus paradoxus, Kussmaul's sign, and pericardial rub are often absent (Press & Livingston, 1987). The patient will most commonly demonstrate tachycardia (the body's response to a limited stroke volume), hypotension, and venous distension of the jugular and scalp veins (Pawlak Cieślik et al., 2012). Echocardiography is then essential for diagnosis and can also guide treatment. If the patient is hypovolemic, infusion of intravenous fluids can temporarily help. However, the definitive treatment for acute cardiac tamponade is prompt drainage of the fluid by pericardiocentesis or subxiphoid pericardiotomy, with a success rate of 90% or greater with minimal complications (Donato et al., 1986).

Our patient was asymptomatic, demonstrating tachycardia followed by hypotension and hypoxia within 24 h of surgery. The etiologies of primary concern were pulmonary embolism, infection, hemorrhage, and pulmonary compromise secondary to known pleural effusion. Her hypotension was unresponsive to fluid resuscitation and she quickly developed cardiac failure. In retrospect, bedside echocardiogram would have likely been diagnostic; indeed it has been suggested that echocardiography be used routinely in critically ill patients with hemodynamic instability (Beaulieu, 2007).

Further suggesting the diagnosis of cardiac tamponade in this patient was the known presence of pleural effusions. It has been shown that pleural effusions with or without associated pericardial effusion can cause cardiac tamponade. Adam et al. note that even a small pericardial effusion may become clinically significant with the added extrinsic pressure of a large pleural effusion (500–2000 ml in this case series) (Traylor et al., 2002). In this patient, pleural effusions measured 250 ml on the left and 450 ml on the right, perhaps large enough to cause significant extrapericardial stress and impaired intracardiac filling. It is possible that pre- or post-operative thoracentesis would have prevented or resolved the tamponade, but this has been demonstrated in the literature only with much larger pleural effusions (900–1500 ml) (Traylor et al., 2002).

The differential diagnosis of pericardial effusion is broad. Our patient was previously healthy with no past medical or surgical history. She had no predisposing factors such as history of radiation, cardiac, renal, or liver disease, or chronic inflammatory disease such as lupus. In this case, no malignant cells were seen in the pericardial space; however, the sensitivity of pericardial fluid cytology has been shown to be as low as 46% (Pawlak Cieślik et al., 2012). It has been reported that up to 40% of pericardial effusions in patients with metastatic cancer may have a non-malignant cause, thus alternate etiologies must be considered as outlined by Donato et al. (1986). Although there are significant fluid shifts that take place during and after cytoreductive surgery for ovarian cancer (Eisner et al., 1990), it would be rare for this to lead to fluid accumulation in a body cavity not already affected, making this an unlikely cause of acute cardiac tamponade.

The histologic type of cancer may play an important role in predicting the risk of developing pericardial involvement. Patel et al. (1999) reported a trend of supradiaphragmatic manifestations of serous ovarian cancer, suggesting that this histologic type is more likely to appear in the chest (Patel et al., 1999). Pericardial effusion has also been reported as the presenting symptom in a 43-year-old with serous adenocarcinoma of the ovary (Winter et al., 2002), suggesting that involvement of the heart may occur even very early in the disease process for this histologic type. Indeed, our patient's pathologic diagnosis was consistent with high-grade serous carcinoma. Though there were no malignant cells found in either the pericardium or the pericardial effusion perhaps the serous histopathology can play a unique role in affecting the pericardium early in the disease process.

Pericardial effusion is a rare but dangerous complication of ovarian cancer. Though more common in recurrent disease, it may be present upon initial diagnosis and can be life threatening, especially when coupled with the hemodynamic stress of debulking surgery. Cardiac manifestations of ovarian carcinoma, such as pericardial effusion and tamponade, should be considered in the differential diagnosis of postoperative hemodynamic instability so that life-saving pericardiocentesis can be performed.

Conflict of interest

The authors declare that they have no conflict of interest.

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