Ovarian Carcinoma Presenting With Isolated Contralateral Inguinal Lymph Node Metastasis: A Case Report

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Introduction

Ovarian carcinoma is the most frequent cause of death from gynaecological malignancies in the United States. Rates for ovarian carcinoma in Singapore fall between those of Western Europe and those of the rest of Asia, accounting for 5.4% of all female cancers diagnosed between 1998 and 2002 in Singapore.1 A major contributing factor to the high mortality is the lack of symptoms associated with the disease at an early stage. This is in part due to the intra-abdominal location of the ovaries, and that symptoms do not occur until malignancy is widespread. Isolated inguinal lymph node metastasis is an uncommon manifestation of ovarian carcinoma. To date, there have been 3 case reports that bear similarities to our case.2-4 We describe a patient seen at the Singapore General Hospital who presented with an enlarged metastatic (contralateral) right inguinal lymph node from a primary left ovarian adenocarcinoma without evidence of disseminated intra-abdominal disease or gross evidence of pelvic or para-aortic lymph node disease. To our knowledge, such a rare presentation of primary ovarian cancer has not been reported before.

Case Report

A 59-year-old gravida 2 para 2 Chinese patient with the Eastern Cooperative Oncology Group (ECOG) performance status0 presented in July 2005 with a painless right groin swelling. She had a medical history of uterine fibroids, which had regressed after menopause at age 54 years. Her most recent pelvic ultrasound performed in July 2001 was normal. Papanicolaou smear examination performed in April 2002 was negative for malignancy. The patient had...
never received hormone replacement therapy, and had no personal or family history of malignancy. Clinically, an enlarged, non-tender right inguinal lymph node (3 x 2 cm) was palpable. There was no evidence of other lymphadenopathy, ascites or abdominal masses. Breast examination was normal, the thyroid gland was not palpable and there were no skin lesions. Pelvic and rectal examination did not reveal any abnormality. Chest X-ray (CXR) did not show any lung lesions. An excision biopsy of the right inguinal lymph node showed metastatic adenocarcinoma (Fig. 1). Computed tomography (CT) scans of the abdomen and pelvis revealed a large, complex left ovarian mass of 9.0 x 6.4 cm (Fig. 2). There was no CT evidence of ascites, intra-abdominal lymphadenopathy or metastatic involvement. The full blood count, renal function tests and liver function tests were normal. CA-125 was elevated at 215 U/mL (normal range, <35 U/mL). The serum beta-2-microglobulin was 1189 µg/L (normal range, 878 to 2000 µg/L) and the CEA level was 0.7 µg/L (normal range, 0.5 to 3.5 µg/L).

An exploratory laparotomy was performed on 19 August 2005. Intraoperative findings were that of a 10-cm left ovarian mass with an intact capsule. There was no ascites. The liver, diaphragmatic and subdiaphragmatic surfaces, stomach, small and large bowels, omentum and peritoneal surfaces were all normal by inspection and palpation. There were no palpable pelvic or para-aortic lymph nodes. Frozen section of the left ovary showed adenocarcinoma, similar to the findings from the earlier right inguinal lymph node excision biopsy. Total hysterectomy, bilateral salpingo-oophorectomy (THBSO) and partial omentectomy were performed. Histopathological examination revealed a moderately differentiated papillary serous adenocarcinoma of the left ovary, with the presence of intra-ovarian vascular tumour emboli, but the ovarian capsule was not breached by tumour (Fig. 3). The cervix, uterus, right ovary and omentum were negative for malignancy histologically. Postoperative recovery was uneventful, and the patient was commenced on adjuvant chemotherapy with paclitaxel and carboplatin for treatment of Stage IIIc ovarian carcinoma based on the International Federation of Gynaecology and Obstetrics (FIGO) classification system. The CA-125 level decreased from a preoperative value of 215 U/mL to 43.3 U/mL postoperatively, with a steady decline to a normal value of 10.4 U/mL after the first cycle of chemotherapy. Following completion of 6 cycles of chemotherapy in February 2006, the CA 125 level recorded was 2.7 U/mL. Follow-up abdominal and pelvic CT at the end of chemotherapy in February 2006 showed no evidence of recurrent or residual lesions in the pelvis. In addition, no
free fluid or adenopathy was demonstrated in the pelvis. The CA-125 level recorded at follow-up on 7 September 2006 was <2.0 U/mL.

Discussion

The FIGO defined lymph node involvement into the definition of Stage III ovarian carcinoma in 1985. Ovarian carcinoma presenting with isolated inguinal lymph node enlargement is unusual. Scholz et al described a 43-year-old patient who had an undifferentiated serous adenocarcinoma of both ovaries with metastasis to the right fallopian tube and left inguinal lymph node diagnosed intraoperatively after she presented with left inguinal swelling. In a similar case, McGonigle and Dudzinski highlighted the case of a 59-year-old gravida 0 post-menopausal white female whose initial presentation of an enlarged left inguinal lymph node was attributed to secondary involvement from metastatic endometrioid carcinoma involving both ovaries and the right fallopian tube. Metastatic adenocarcinoma of the left (ipsilateral) inguinal lymph node preceded any evidence of left ovarian carcinoma by 33 months in a 66-year-old Caucasian female reported by Kehoe et al.

The most common routes of spread in epithelial ovarian carcinoma are lymphatic dissemination and transcoelomic spread to adjacent visceras. Lymphatic drainage occurs mainly via the infundibulopelvic ligament to the para-aortic lymph nodes; hence, these nodes are at highest risk of involvement through lymphatics that run parallel to ovarian vessels. Less commonly, lymphatics traverse the subovarian plexus in the broad ligament to the obturator and pelvic lymph nodes. In addition, lymph vessels that follow the round ligament of the uterus pass through the inguinal canal and drain into the superficial inguinal lymph nodes and can also reach the contralateral obturator by traversing across the uterine fundus. We postulate that this may account for inguinal lymph node involvement in the absence of para-aortic or pelvic lymphadenopathy.

The incidence of inguinal lymphadenopathy from ovarian cancer at presentation is rare. In a study by Musumeci et al, preoperative lymphangiogram in 349 patients with ovarian masses identified lymph node metastases in 88 patients. Only 3 of these 88 patients had inguinal lymph node involvement. Following surgical exploration, the radiological-histological correlation was 100%. In a large retrospective analysis of 2232 patients with inguinal lymph node metastases by Zaren and Copeland, an ovarian primary accounted for 5% of all cases. Low rates of inguinal lymph node involvement have similarly been reported in autopsy studies. Whilst autopsy studies provide an important source of data in cancer patients regarding extent of metastases, they often do not correlate with the initial clinical presentation, as they reflect patients who are likely to have significant tumour burden and advanced disease with multiple areas of lymph node disease. An autopsy study by Dvoretsky et al of 100 patients who succumbed to ovarian carcinoma identified inguinal lymph node involvement in 3% of cases. None of these studies provided information on whether inguinal lymph node disease occurred in isolation or concurrently with other areas of metastases.

The third and least common route of spread of ovarian carcinoma occurs by the haematogenous route, occurring in approximately 2% to 3% of patients. The presence of vascular tumor emboli in the histological specimen in our patient may account for haematogenous dissemination to distant sites, such as the contralateral inguinal lymph node illustrated in our case and the involvement of right axillary lymph nodes described by Hockstein et al.

Whilst most patients with FIGO Stage IIIc present with advanced clinical manifestations, our case is unusual as the only abnormality on clinical examination was an enlarged contralateral inguinal lymph node. The impact of such a clinical presentation on prognosis is unknown, with no previous studies reported on long-term survival. Similar to the case described by Kehoe et al, where metastatic adenocarcinoma involving the ipsilateral inguinal lymph node preceded any evidence of ovarian carcinoma, the diagnosis of inguinal lymph node metastases in our patient occurred before any clinical manifestation of intra-abdominal disease. Our case is unique as the only site of metastasis was the contralateral inguinal lymph node. This is contrary to the findings of Benedetti-Panici et al, who observed that when ovarian cancer is localised to one ovary and in the absence of intraperitoneal spread, lymphatic involvement occurs only in the ipsilateral lymph nodes.

We present a patient with left ovarian adenocarcinoma with isolated metastasis to the right inguinal lymph node. This rare and unusual presentation highlights the need to consider ovarian carcinoma in the differential diagnosis of women presenting with inguinal lymphadenopathy.

REFERENCES

4. Kehoe S, Luesley D, Rollason T. Ovarian carcinoma presenting with