Fludeoxyglucose F 18 PET/CT Assessment of Ovarian Cancer

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KEYWORDS

• Ovarian cancer • FDG PET/CT • Staging • Biomarkers • Prognosis • Response to treatment

KEY POINTS

- Fludeoxyglucose F 18 (FDG) PET/MR imaging is an emerging hybrid modality that may be useful in the assessment of many malignant disease conditions, including ovarian and others pelvic tumors.
- Thanks to its morphologic high soft tissue contrast and the use of diffusion-weighted imaging–MR imaging with intravenous contrast administration, PET/MR imaging is able to overcome some limitations of FDG PET/CT in the detection of the primary tumor in patients with a high suspicion of disease and also in the delineation of the tumor mass for pretreatment planning, which may have an impact on the therapeutic approach.
- The reason for the high death rate is the late presentation in most cases, due to its silent nature earlier in the course of disease.

INTRODUCTION

Ovarian cancer is one of the most common gynecologic malignancies, is the most fatal gynecologic malignancy, and is the fifth most common cause of female cancer–related death.[1] The reason for the high death rate is the late presentation in most cases, due to its silent nature earlier in the course of disease. Thus, patients often present with advanced disease, widely spread within the abdomen (75% of cases), in the absence of specific signs or symptoms. One of the most important factors influencing survival is represented by the disease stage at diagnosis.[2] Despite advances in medicine over the past decades, only minor improvement in 5-year survival has been achieved in patients diagnosed with advanced epithelial ovarian cancer.[3]

EPIDEMIOLOGY OF OVARIAN CANCER

Ovarian cancer is one of the most commonly diagnosed cancers and one of the leading causes of cancer death in women worldwide, accounting for approximately 3.6% (238,719) of total new cancer cases and 4.3% (151,917) of total cancer deaths among women in 2012.[4] Published epidemiologic data estimate approximately 22,440 new cases in the United States in 2017, accounting for approximately 2.6% of all new malignancies in women. According to the same data, ovarian cancer is the leading cause of gynecologic cancer–related deaths (14,080 deaths estimated for 2017) and the fifth most frequent cause of cancer mortality in women (5% of all cancer deaths) after lung and bronchus (25%), colon-rectum (14%), breast (8%), and pancreas (7%) cancers.[5]

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According to the 2017 Surveillance, Epidemiology, and End Results program statistics of the National Cancer Institute, the number of new cases of ovarian cancer was 11.7 per 100,000 women per year, and the number of deaths was 7.4 per 100,000 women per year (rates age adjusted and based on the 2010–2014 period). According to the same data for patients with disease limited to the ovaries, the 5-year survival rate is approximately 92.5%, whereas for patients presenting with advanced disease, the 5-year survival rate is approximately 30%.[5] Unfortunately, approximately 70% of patients are diagnosed with advanced ovarian cancer.

SPREAD

Ovarian cancer spreads via 3 different routes: (1) the peritoneum, (2) the lymphatic system, and (3) the bloodstream.

1. The peritoneum is the most frequent target, due to the distribution of cells within the normal peritoneal fluid circulation. Tumor cells from the primary tumor spread into the peritoneal cavity and then are transported by peritoneal fluid toward the upper abdominal quadrants. During breathing movements, the negative pressure at the subphrenic level increases to a positive one, so fluid moves from the paracolic gutters up to the right subhepatic space and the right subdiaphragmatic space.[6] The more common sites of tumor disseminations include greater omentum, paracolic gutters, pouch of Douglas, liver (especially glissonian capsule), diaphragmatic and bowel surface, and, less frequently, mesentery, splenic surface, porta hepatis, and gastrosplenic ligament. Peritoneal involvement may appear as nodular soft tissue lesions, linear or plaque-like thickening of the parietal or visceral peritoneum, or, in histologically serous tumors, tiny calcifications,[3]
2. Lymph node dissemination can follow 3 different routes: (a) along the ovarian vessels (more frequently), reaching the upper common iliac and para-aortic lymph nodes; (b) along the broad ligament and parametrium, reaching the external iliac and obturator lymph nodes; (c) rarely, along the round ligaments, toward the external iliac and inguinal lymph nodes.[7–9]
3. Through the bloodstream, tumor dissemination usually occurs to the liver, lung, spleen, and skeleton.[7]

STAGING: INTERNATIONAL FEDERATION OF GYNECOLOGY AND OBSTETRICS, TNM, AND WORLD HEALTH ORGANIZATION CLASSIFICATIONS

International Federation of Gynecology and Obstetrics Classification

After the diagnosis of cancer, staging is an essential step in disease management, with the aim to define, with standard terminology, tumor characteristics and extension and to assign patients to prognostic groups to optimize/personalize treatment options to achieve a more favorable outcome.

At present, the staging system worldwide used for ovarian cancer is represented by the International Federation of Gynecology and Obstetrics (FIGO) staging classification,[7] which was first published in 1973, then revised in 1988 and, more recently, in 2014. Numerous publications in recent years have enlightened various controversial aspects of the disease. It is recognized that "ovarian cancer" is not a homogeneous disease but is instead a group of diseases, each with different etiology, pathogenesis, morphology, and prognosis, which can present in the ovaries, fallopian tubes, and peritoneum.[10] In the new FIGO staging classification of 2014, ovarian, fallopian tube, and primary peritoneal cancers are considered collectively as 1 entity. Ovarian cancers differ primarily based on histologic type. Approximately 90% of ovarian, fallopian tubes, and primary peritoneum tumors are carcinomas, with at least 5 main types accounting for 98% of them (based on histopathology, immunohistochemistry, and molecular genetic analysis):

1. High-grade serous carcinoma (HGSC [70%])
2. Endometrioid carcinoma (EC [10%])
3. Clear cell carcinoma (10%)
4. Mucinous carcinoma (3%)
5. Low-grade serous carcinoma (<5%)[11]

Much less common types are malignant germ cell tumors (dysgerminomas, yolk sac tumors, and immature teratomas [3% of ovarian cancers]) and potentially malignant sex cord–stromal tumors [1%–2%, mainly granulosa cell tumors].[10] Despite differences in tumor biology, dissemination pattern, response to chemotherapy, and outcome, the FIGO staging classification is the same for each type of ovarian carcinoma for the sake of simplicity, considering the most relevant prognostic parameters shared by all tumor types.[11]

Stage I

Stage I includes tumors limited to the ovaries or fallopian tubes, although tumor cells may be