

suppressor gene likely serve as secondary genetic “hits” in the carcinogenesis related to estrogenic excess. The molecular events that underlie less common endometrial cancers such as clear cell and papillary serous tumors of the uterine corpus are not well understood.

■ PATHOLOGY

Approximately 75–80% of endometrial cancers are adenocarcinomas and have been characterized as type 1 (estrogen-linked) endometrial cancers and type 2 cancers that have less clear associations with estrogens (clear cell cancers, serous cancers, and mucinous cancers). Endometrial serous cancers show *TP53* loss of function and behave clinically more like ovarian cancers with high risk for systemic recurrence. Prognosis for endometrial cancer depends on stage, histologic grade, and depth of myometrial invasion.

■ CLINICAL PRESENTATION

The majority of women with tumors of the uterine corpus present with postmenopausal vaginal bleeding due to shedding of the malignant endometrial lining. Premenopausal women often will present with atypical bleeding between typical menstrual cycles. These signs typically bring a woman to the attention of a health care professional, and the majority of women present with early-stage disease in which the tumor is confined to the uterine corpus and, consequently, have a high cure rate. Diagnosis is typically established by endometrial biopsy. Type 1 tumors may spread to pelvic or para-aortic lymph nodes and are generally subjected to sentinel lymph node biopsy at the time of primary surgery. Serous tumors tend to have patterns of spread much more reminiscent of ovarian cancer, and patients may present with omental/peritoneal disease and sometimes ascites. Some women with endometrial cancer have a history of endometriosis. Some women presenting with uterine sarcomas will present with pelvic pain. Uterine sarcomas (carcinosarcomas and leiomyosarcomas) commonly are found by