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ScienceDirect
Annals of Diagnostic Pathology 11 (2007) 297–311
Annals of DIAGNOSTIC PATHOLOGY

Review Article

Histopathology of endometrial hyperplasia and endometrial carcinoma An update

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Abstract Endometrial cancer (EC) is the most common malignancy of the female genital tract in the western world. Conceptually, a dualistic model of endometrial carcinogenesis exists for sporadic EC, based on molecular findings with a good correlation to the morphologic phenotype and clinical behavior. Type 1 endometrial carcinoma represents an estrogen-related tumor, which usually arises in the setting of endometrial hyperplasia, has endometrioid histology with low grade, and tends to be biologically indolent. Grade 3 endometrioid cancers, which constitute a minority of EC, also behave aggressively. The type 2 cancers are not estrogen-driven and have a higher grade, various histologies, particularly serous carcinomas and clear-cell carcinomas, and a poorer prognosis. The diagnostic criteria of endometrial hyperplasia, endometrial in situ carcinoma, and of the different histologic types of EC, according to the most recent World Health Organization classification, are given in detail. In addition, the risk of progression of endometrial hyperplasia into endometrioid type EC and their treatment modalities are discussed. Endometrial pathologies in patients with breast cancer, receiving tamoxifen, and women affected by hereditary nonpolyposis colorectal cancer syndrome are described, including their pathogenetic aspects. Finally, a short practical description for the handling of surgical specimens from fractional curetting and hysterctomies is given.
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Keywords: Endometrial carcinoma; Endometrial hyperplasia; Type 1 and type 2 carcinoma; Microscopy; Surgical pathology

1. Introduction

During the last decades, a change of demographics has occurred. The number of women more than 60 years of age is increasing worldwide, with increasing life span. In addition, women in the 21st century will also experience menarche at a younger age than earlier generations, will typically (especially in countries of the western world) have fewer children, and, as a consequence, spend less time breast-feeding. In addition, they will enter the menopause stage later as some decades before. As one consequence, the focus for the aging woman will be on lifestyle support to counteract various features of degenerative changes. This will be accompanied by an increasing demand for medical expertise on lifestyle drugs, for example, the use of hormone replacement therapy. In addition, there is an increase in malignant diseases worldwide, also in women [1]. All these factors might also associate with a potential increase in endometrial malignancies and their precursors. Endometrial cancer (EC) is the most common invasive malignancy of the female genital tract with an estimated incidence of 39.080 new cases in the United States of America for 2007 [2]. In Germany, there are about 11,300 estimated cases each year [3], representing the fourth most common cancer in women and the number one in cancers of the female genital tract.

Historically, more than 23 years ago, Bokhman [4] classified endometrial carcinomas into 2 types. Although this classification is an oversimplification, it is of use conceptually. The so-called type 1 endometrial carcinoma represents an estrogen-related carcinoma, usually arises in the setting of endometrial hyperplasia and have endometrioid

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doi:10.1016/j.anndiagpath.2007.05.002

Table 1
Principal types of endometrial carcinoma

	Type I	Type II
Menopausal status	Premenopausal and perimenopausal	Postmenopausal
Estrogen-related	Yes	No
Estrogen or progesterone receptors	Present	Absent
Histology of adjacent endometrium	Hyperplastic	Atrophic/cystic polyp
Precursor lesion	Atypical hyperplasia	EIC
Obesity	Yes	No
Parity	Nulliparous	Multiparous
Grade	Low	High
Histologic subtype	Endometrioid	Serous carcinoma clear-cell carcinoma
Clinical behavior	Indolent	Aggressive

histology with low grade, and tends to be biologically indolent. However, grade 3 endometrioid cancers, which constitute a minority of EC, also behave aggressively.

The type 2 cancers are not estrogen-driven and have a higher grade, various histologies, particularly serous carcinomas and clear-cell carcinomas, and a poorer prognosis. Table 1 lists the classic features of the 2 categories [5,6]. Black women have substantially more aggressive tumor types than do white non-Hispanic women, including serous carcinoma and clear-cell carcinoma, and have a worse overall survival for all tumor types [7].

Based on these features, a dualistic model of endometrial carcinogenesis for sporadic EC has been proposed [8,9].

The present review will summarize the morphologic features of the different types of endometrial carcinomas, their precursors, and of rare ECs in recognition experiences and recent published updates [10-16]. In addition, a short update on handling and histopathologic reporting of curettings and hysterectomy specimens in endometrial hyperplasia and carcinoma will be given at the end of this article.

Malignant mixed mullerian tumors are not included in this article and have been reviewed recently [15].

2. Endometrial hyperplasia

Endometrial hyperplasia represents a nonphysiological, noninvasive proliferation of the endometrium that results in a morphologic pattern of glands with irregular shapes and varying size [9,12]. There are 2 forms of hyperplasia, one (atypical) that is closely related to adenocarcinoma, being an apparent precursor lesion, and another (nonatypical) that is largely self-limited with little apparent relationship to carcinoma [17-19]. The World Health Organization (WHO) classification of endometrial hyperplasia is summarized in Table 2. Correct identification and classification of hyperplasia are especially important in endometrial biopsy and curettage specimens because proper diagnosis guides clinical therapy [20], which can be very different depending on the type of hyperplasia found.

Table 2
World Health Organization classification of endometrial hyperplasia (Silverberg et al [14])

Nonatypical hyperplasias (typical)
Simple hyperplasia without atypia
Complex hyperplasia without atypia (syn. adenomatous hyperplasia without atypia)
Atypical hyperplasias
Simple atypical hyperplasia
Complex atypical hyperplasia (syn. atypical adenomatous hyperplasia)

3. Histopathology of endometrial hyperplasia associated with type 1 carcinoma

All forms of hyperplasia share certain morphologic features, showing an increase in the gland-stroma ratio, irregularities in gland shape, and variation in gland size [13,14]. The leading clinical symptom is irregular bleeding. The amount of tissue obtained at curettage may be considerable, sometimes yielding enough to fill 3 or more tissue cassettes.

Simple and complex forms of hyperplasia are distinguished by architectural alterations characterized by

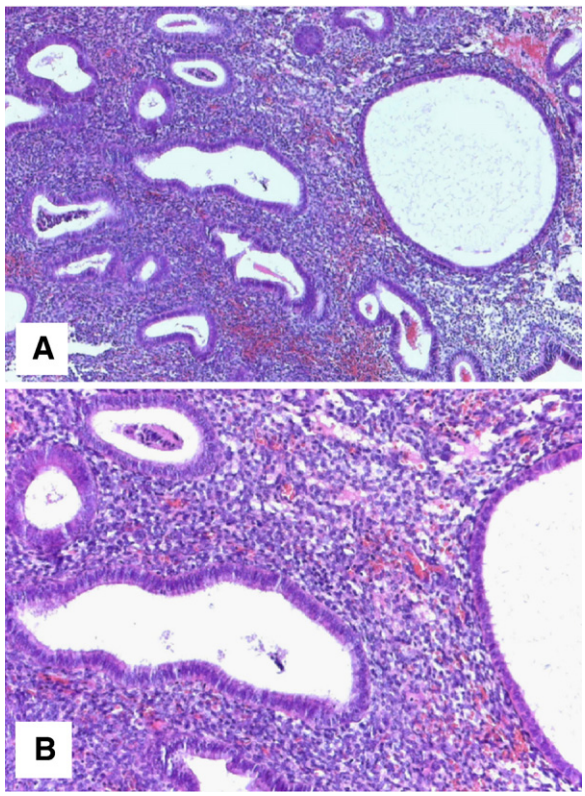

Fig. 1. Simple hyperplasia without atypia: proliferated endometrial glands with irregular distribution and cystic dilatation, but widely separated by endometrial stroma, which is also hyperplastic (A). Glands are lined by proliferative-type endometrial epithelium without atypia (B).

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