Cancer Prone Disease Section
Short Communication

Uterus: Endometrial carcinoma

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Classification

The modified WHO classification distinguishes:
- endometrioid adenocarcinoma
- serous carcinoma
- clear cell carcinoma
- mucinous carcinoma
- serous carcinoma
- mixed types of carcinoma
- undifferentiated carcinoma

Clinics and pathology

Disease

It is an heterogeneous entity, comprising at least two types:
- type I: endometrioid carcinoma: pre- and perimenopausal, estrogen dependent, associated to endometrial hyperplasia, low grade, indolent behaviour, representing about 80 % of the cases
- type II: serous carcinoma: post-menopausal, estrogen independent, associated to atrophic endometrium, high grade, aggressive behaviour, representing about 10 % of the cases.
- among other histologic types, type I includes mucinous and secretory carcinomas, whereas type II includes clear-cell carcinomas and adenosquamous carcinomas.

Embryonic origin

Tumour developed from the epithelium of the endometrial mucosa.

Etiology

A strong association between unopposed estrogen stimulation and the development of endometrial carcinoma has been demonstrated; this may be related to replacement estrogen therapy, obesity, or chronic anovulation.

Epidemiology

It is the most common malignancy of the female genital tract in developed countries (33,000 new cases per year in the USA); the incidence is 4 to 5 times lower in developing countries.

Genetics

Note

Besides the common sporadic form, there are two forms of hereditary endometrial carcinoma:
- a predisposition to endometrial carcinoma;
- Lynch syndrome, which associates a risk of colon cancer, and ovarian carcinoma in women.

Cytogenetics

Cytogenetics Morphological

The results of conventional cytogenetic techniques and Comparative Genomic Hybridisation (CGH) show that the two types of endometrial carcinoma differ by their karyotypic features: endometrioid carcinomas are characterised by relatively simple chromosome aberrations whereas serous carcinomas show complex abnormalities.

- Endometrioid carcinomas are generally slightly hyperdiploid; chromosome gains concern mainly the long arm of chromosome 1 (70 % of the cases) through isochromosomes or unbalanced translocations; it can be observed as sole abnormality; trisomies 10 (40 % of the cases), 2, 7, and 12 are, in decreasing order, the most frequently associated abnormalities; trisomy 10 can exist as sole imbalance; loss of chromosome 22 has also been recurrently observed; Comparative Genomic Hybridization (CGH) confirms the low rate of copy number changes (mean of 1,5 aberrations per tumour); it roughly shows the same imbalances, and, in addition, gains of 8q (18 % of cases), and of the region 13q21--qter (3 cases); loss of chromosome 22 has not be
detected using this method; high level amplification was found in 3 cases, in 1q and in 6p.
- Due to their low incidence and to the complexity of their chromosome abnormalities serous carcinomas are less documented; a CGH study, carried out on 24 case showed a higher rate of chromosome imbalances (mean of 5.7 aberrations per tumour); the most frequent regions of gain were 3q26.1-->qter (50 % of cases), 8q (33 %), and 1q, 2q, 5p, 6p; high level amplifications were found in 30 % of the cases in 2q, 3q, 5p, 6p, 8q, 15q, 18 p and 18q, 20.
- The distinct patterns of chromosome changes observed in serous and endometrioid carcinomas suggest that these two types belong to two genetic entities. A comparison of these results with those of a CGH study of serous ovarian carcinoma (cited in 5) shows that the serous endometrial carcinoma should be genetically related to this tumour.

References


This article should be referenced as such: