Solid Tumour Section
Mini Review

Bladder: Squamous cell carcinoma

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Classification

Existence of different histologic types of bladder cancer:
- Squamous cell carcinoma: herein described,
- Transitional cell carcinoma,
- Adenocarcinoma: rare,
- Poorly differentiacted carcinoma/small cell carcinoma, exceptional.

Clinics and pathology

Disease
Cancer of the urothelium.

Etiology
Most often secondary to bilharzial infection (schistosoma haematobium), may be associated with other types of long term irritations: chronic infections, calculi, treatment with cyclophosphamid.

Epidemiology
Geographic areas of high incidence: represents 70 to 80% of the cases of bladder cancer in the Middle East and in Africa, in particular in Egypt, were it is the most common adult cancer; only 5% in Europe and in the USA, where the transitional cell carcinoma represents 90-95 % of cases.

Pathology
Graded and staging: tumours are:
Graded by the degree of cellular atypia (G0->G3), and staged: pTIS carcinoma in situ (but high grade), and pTa papillary carcinoma, both mucosally confined; pT1 lamina propria invasive; pT2 infiltrates the superficial muscle, and pT3a, the deep muscle; pT3b invasion into perivesical fat; pT4 extends into neighbouring structures and organs.
**Prognosis**
Considered to have a poorer prognosis than the transitional cell carcinoma.

**Cytogenetics**

**Cytogenetics Morphological**
Highly complex karyotypes, yet poorly known. Allelic losses are frequent; the most frequent regions involved in loss of heterozygosity (LOH) are 3p, 8p, 9p, 9q, 17p; the karyotype is more complex in advanced grades/stages, as in transitional cell carcinoma.

Chromosome 7: trisomy 7 seems to be more frequent than in transitional cell carcinoma, and is found more often in advanced stages; unknown significance as +7 may also be found in normal tissues.

Chromosome 9: monosomy 9 is an early event and might even occur at dysplastic stages; allelic losses are frequent, mainly in 9p (65%), more often than for transitional cell carcinoma; LOH are found in particular in the locus where CDKN2/P16 sits; homozygous deletion of P16 is frequent (50%) and may also be found in squamous metaplasias from cancerous patients (but not in squamous metaplasias from non cancerous patients); trisomy 9, on the other hand, would be frequent in advance diseases.

Chromosome 17: P53 is often implicated, especially in high grades/stages; the profile of mutations of P53 is different from what is found in transitional cell carcinoma.

**Cytogenetics Molecular**
Comparative genomic hybridization (CGH) and multi-FISH (M-FISH) are complementary tools to determine respectively unbalanced segments and structural rearrangements in these complex karyotypes.

**Genes involved and proteins**

**Note**
Multistep process; largely unknown.

**References**

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