sufficient for a diagnosis of PCA whereas in some cases the application of immunohistochemistry is necessary.

Architectural features are assessed at low to medium power magnification. Malignant acini usually have an irregular, haphazard arrangement. The suspicious acini are usually small. In the experience of many pathologists, low grade PCA and Gleason grade 3 PCA are particularly difficult to separate from benign acini in needle biopsies due to the relatively uniform size and spacing of acini. The basal cell layer is completely absent in PCA, an important feature which sometimes needs immunohistochemical verification.

Concerning cytologic features, the most important in the majority of cases are nuclear and nucleolar enlargement. Since practically every single cell reveals a nucleolus, one looks for larger ones (1.25-1.50 microns). The presence of two or more nucleoli in a single cell is virtually diagnostic for PCA (19). Nucleoli are found in virtually all cells. Nucleolar size is a good indicator of cancer when compared with benign lesions (20). Nuclear size is an important finding used to differentiate AAH from PCA. Crystalloids and luminal mucin are often seen in larger quantities in PCA. Crystalloids are needle-like eosinophilic structures found in well and moderately differentiated PCA and they are not specific for PCA (21, 22). The pathogenesis of crystalloids is not clear. They are considered to be the result of abnormal protein and mineral metabolism within benign and malignant acini. Mucin (acidic sulfated and non sulfated) mucin is often seen in the acini of PCA. This mucin can stain with alcian blue, whereas the normal prostatic epithelium contains acid mucin which sometimes needs immunohistochemical verification.

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Molecular diagnosis of prostate adenocarcinoma with emphasis on screening programs

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Introduction

The use of sextant prostate biopsy associated with serum prostate-specific antigen levels has become the most common way of diagnosing prostate adenocarcinoma. However, early diagnosis in nonsymptomatic men introduced some practical problems related with the biology of the neoplasia and with the unexpected prognosis of this tumor in what concerns aggressive versus nonaggressive behavior. In spite of the prognostic information given by conventional histology, many questions remain to be answered. Consequently, research is also underway in the fields of molecular biology and genetics in order to better understand such matters as premalignant lesions, aggressive and nonaggressive tumors, mechanisms of metastasis, hormonal response and nonresponse tumors. In the present study we will present our results in the following fields: susceptibility to prostate carcinoma; diagnosis; immunexpression of oncoproteins and adhesion molecules; immunexpression of MUC1; and detection of circulating prostate cells in peripheral blood.

Susceptibility to prostate carcinoma

Epidemiological studies suggest a correlation between prostate carcinoma and certain geographical, racial and environmental risk factors. The incidence and mortality from prostate cancer is highest in the Afro-American population (1) and lowest in the Far East, namely in Japan and China (1, 2). Japanese men have significantly reduced hormone-binding globulin and reduced 5α-reductase activity and this can explain, at least in part, the recognized low incidence of prostate adenocarcinoma (3).

We studied the role of glutathione S-transferase polymorphism in several neoplasias, including prostate. The presence of a GSTM1 null genotype was analyzed by polymerase chain reaction (PCR) in DNA isolated from the blood samples of 67 patients with neoplastic progression as well as to induce the regression of cancers and their precursors. The rapid pace of research in this field suggests that this aspect of tumor biology will be soon have increasingly important roles in evaluation and treatment.

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