

Commentary on Prostate Carcinoma with Squamous Differentiation

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COMMENTARY

We mention a commentary involving the prostate and urinary bladder of pure squamous cell carcinoma and explain the diagnostic dilemmas we encountered in trying to ascertain its origin. Ten years ago, a woman was diagnosed with prostatic adenocarcinoma and treated with radioactive seed implantation. He also underwent a TURP procedure over the past year for urinary obstruction complicated by multiple infections. The patient developed colo-urethral fistula after surgery, and the decision to perform a cystprostatectomy was made. A tumor mass replacing the entire prostate that microscopically proved to be squamous cell carcinoma was demonstrated by Excision. The problem we encountered was to determine its origin, the possibilities being divergent differentiation with substantial squamous differentiation from post-radiation therapy adenocarcinoma, de novo neoplasm or urothelial carcinoma.

Prostate squamous cell carcinoma is a rare individual, responsible for less than 1 percent of all prostate carcinomas. During endocrine or radiation treatment for adenocarcinoma, about half of the cases occur. However, "de novo" cases have also been identified in patients with no history of prostatic disease. This discovery points to different etiologies for this disease. Squamous distinction of prostate cancer is thought to derive from the urothelial lining of the prostatic urethra or periurethral ducts. It is also speculated that pluripotent stem cells capable of multidirectional differentiation could be derived from it. Morphologically, in pure form or associated with adenocarcinoma, urothelial carcinoma or sarcoma, squamous differentiation in prostate cancer may be observed. Given its many potential causes, it can be very difficult to determine if the squamous portion evolves after treatment by divergent differentiation from adenocarcinoma, whether it reflects squamous differentiation of transitional cell carcinoma, or if it is pure second prostatic malignancy. Understanding this tumor's biology could help to establish more effective therapies with a poor prognosis for this aggressive malignancy. Prostate cancer screening is based on a sensitive cancer test, as far as no identifiable precursor has been found for which a test could be developed. This is equivalent to screening for breast cancer, but not, of course, for either cervix or colorectal cancer, although it seems likely that the identification of early cancers, and not only adenomatous polyps, is the key advantage of screening for colorectal cancer through the faecal occult blood test. Of the available prostate cancer screening tests, automated rectal examination (DRE) and prostate-specific antigen (PSA) blood tests, the latter is the one with the greatest chance of reducing the mortality rate of prostate cancer. DRE and PSA screening, the European Randomized Screening Trial for Prostate Cancer (ERSPC) in seven European countries, and the Prostate, Colon, Lung and Ovary Trial (PLCO) in 10 centres in the United States are underway in two large trials. From both, it is already clear that DRE lacks sufficient sensitivity. The fact that occult prostate carcinomas can be demonstrated at autopsy, as first recorded in Arnold Rice Rich's classic paper replicated in this IJE edition, and subsequently verified by other writers, complicates considerably the problem of prostate cancer screening and raises some interesting aspects of its epidemiology. Just to quickly consider these aspects of epidemiology, the data we have seems to indicate that occult (latent) prostate carcinomas can be demonstrated on a roughly similar scale in all populations of older men. Therefore, the major variations that occur epidemiologically in the incidence of prostate cancer may be attributed to factors that influence the progression of occult carcinomas to clinically important cancers, some of which are apparently dietary, especially animal fat.

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