When pelvic arterial bleeding exists, arteriography and trans-catheter embolization of the bleeding arteries are often required. Pelvic arterial injuries from pelvic fracture are in decreasing frequency, to the internal pudendals, superior gluteal, obturator and lateral sacral arteries. Arteriography is indicated in the presence of ongoing blood loss after intra-abdominal sources have been eliminated and the pelvis, at least temporarily, is stabilized. In stable patients, contrast blush on CT imaging indicates a high likelihood of arterial injury and angiography and embolization should be pursued.

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PATHOLOGY

Propionibacterium acnes associated with inflammation in radical prostatectomy specimens: a possible link to cancer evolution?

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Purpose: Inflammation is commonly observed in the prostate gland and has been implicated in the development of prostate cancer. The etiology of prostatic inflammation is unknown. However, the involvement of a carcinogenic infectious agent has been suggested.

Materials and Methods: Prostatic tissue from 34 consecutive patients with prostate cancer was cultured to detect the presence of bacterial agents. Prostatic inflammation was assessed by histological examination of wholemount tissue sections.

Results: The predominant microorganism detected was Propionibacterium acnes, found in 35% of prostate samples. A significantly higher degree of prostatic inflammation was observed in cases culture positive for P. acnes ($p = 0.007$). P. acnes was separated into 3 groups based on cell surface properties, phenotype and genetic grouping. All skin control isolates were classified as group 1 whereas most prostatic isolates were classified as groups 2 and 3.

Conclusions: P. acnes has been isolated from prostatic tissues in men who underwent radical prostatectomy for localized cancer and has been shown to be positively associated with prostatic inflammation. This inflammation may then be linked to the evolution of carcinoma. Furthermore, organisms infecting these patients with prostate cancer differ genetically and phenotypically from the commonly identified cutaneous P. acnes isolates, suggesting that specific subtypes may be involved in development of prostatic inflammation.

Editorial Comment

This is a very exciting article considering that recently the authors that implicated Helicobacter pylori to the pathogenesis of both peptic ulcer and gastric carcinoma were awarded the Nobel Prize.

Chronic inflammation of longstanding duration has been linked to the development of carcinoma in several organ systems (1-3). In the prostate, chronic inflammation is associated with both postatrophic hyper-

plasia and focal simple atrophy (4). De Marzo et al. (5) propose combining these lesions into a category called proliferative inflammatory atrophy (PIA). The authors suggest that PIA may be a precursor to prostatic adenocarcinoma. They also suggest that there are morphological transitions within the same acinar/duct unit, between high-grade prostatic intraepithelial neoplasia (HGPIN) and PIA that occur frequently (5). This finding supports a model whereby the proliferative epithelium in PIA may progress to HGPIN.

This hypothesis is challenged by others. Anton et al. (6) studying radical prostatectomies concluded that postatrophic hyperplasia is a relatively common lesion present in about one-third of prostates, either with or without prostate carcinoma. The authors found no association between the presence of prostatrophic hyperplasia and the likelihood of cancer and no topographic association between postatrophic hyperplasia and prostate carcinoma foci. This held for both clinical cancer in a radical prostatectomy specimen and incidental cancer in a cystoprostatectomy specimen (6). Bakshi et al. (7) studied 79 consecutive prostate biopsies. 54% of initial biopsies were benign, 42% of the cases showed cancer, and 4% HGPIN or atypia. Postatrophic hyperplasia was seen in 17% of benign initial biopsies with available follow-up. Of these, 75% had associated inflammation. There was no significant difference in the subsequent diagnosis of prostate cancer for groups with postatrophic hyperplasia, partial atrophy, atrophy, or no specific abnormality. The authors conclude that the subcategories of atrophy do not appear to be associated with a significant increase in the risk of diagnosis of prostate cancer subsequently. Billis & Magna (8) studied 100 consecutive autopsies of men older than 40 years. Prostatic atrophy without (group A) and with inflammation (group B) was correlated with age, race, histologic (incidental) carcinoma, HGPIN, and extent of both these lesions. No statistically significant difference was found between the groups. Furthermore, neither a topographical relation nor a morphologic transition was seen between prostatic atrophy and histologic carcinoma or HGPIN. In a recent paper, Postma et al. (9) evaluated whether the incidence of atrophy on sextant biopsies is associated with subsequent prostate cancer detection and did not find a greater prostate cancer or HGPIN incidence during subsequent screening rounds.

The authors of the article surveyed found a positive association between P. acnes and prostatic inflammation, which may be implicated in the development of prostate cancer. However, they comment that it is possible that prostatic inflammation will also be caused by other microorganisms which could not be identified by the study, for example obligate anaerobes or species which are difficult to culture under laboratory conditions. They also comment on a second important limitation of the study related to the lack of appropriate negative controls such as prostate tissue from patients without inflammation, atrophy and cancer.

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Serum PSA level correlates with the needle biopsy extent of atrophy and chronic inflammation, but not with high grade PIN and prostate cancer

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Background: Serum prostate specific antigen (PSA) is the most common marker used to follow men with and without prostate cancer (PCa) and is used as a guide to initiate prostate biopsies. Recently, the value of serum PSA level in predicting the presence or absence of PCa has been questioned.

Design: One hundred consecutive first time saturation prostate biopsies (PBx) performed by a single urologist between February 2003 and May 2004 and reviewed by two pathologists were included in the study. Biopsy criteria were defined as serum total PSA of 2.5 ng/mL or greater and/or abnormal findings on digital rectal examination. All patients underwent a 24-core biopsy protocol. Patient age, PSA, extent (% of tissue involved) of high grade prostatic intraepithelial neoplasia (PIN), % cancer, % atrophy, % chronic inflammation, and presence of acute inflammation were recorded.

Results: The patients were divided in 3 groups: group A (n = 34 with atrophy only), group B (n = 29 with PIN and/or atypical glands), group C (n = 37 with PCa). Atrophy was detected in all cases, ranging from 1.4 to 62.2% of the tissue (mean 22.5%). Chronic inflammation (CI) was present in 98% of the cases, ranging from 0.2 to 44.6% of the tissue (mean 4.5%). Acute inflammation was present in 61% of the cases. The mean PSA and age of the patients for each group were: 7.4 ng/mL and 60.8 years (A); 5.2 ng/mL and 61.7 years (B); 6.0 ng/mL and 65.4 years (C). The difference in mean age between group A (atrophy) and C (PCa) was statistically significant ($p = 0.045$). No correlation was found between PSA and presence or extent of PIN and/or PCa either in the general population (A + B + C) or in the PCa and PIN group (B + C). The presence of PIN was associated with concurrent prostate cancer ($p = 0.003$). Serum PSA level in the general population correlated with the extent of atrophy ($p = 0.022$) and CI ($p = 0.009$).

Conclusions: In this group of patients, preoperative serum PSA level does not correlate with the presence or absence, and extent of PCa and PIN. Atrophy and chronic inflammation are strong contenders for the PSA released into the serum at an increased level. PSA is a marker useful to follow up men with cancer, although its value as screening and staging tool is questionable.

Editorial Comment

The hypothesis that atrophy is a strong contender for the PSA release into the serum is challenging. We have just finished a paper dealing with this subject.

There is evidence that age associated prostatic atrophy may be a manifestation of chronic ischemia due to local arteriosclerosis (1-4). In autopsies, there is a positive and statistically significant association between intense local arteriosclerosis and presence and extent of atrophy (1). The aim of our study was to find any

association between extent of atrophy in prostate needle biopsies and serum prostate-specific antigen (PSA) levels (total, free or free/total ratio).

The study was based on 136 needle prostatic biopsies corresponding to 123 patients. The only diagnosis in all biopsies was focal prostatic atrophy without presence of cancer, high-grade prostatic intraepithelial neoplasia (HGPIN), suspicious for cancer, or prostatitis. The data were analyzed subdividing the patients into 2 groups: with free/total serum PSA ≥ 0.15 (Group 1, 61 biopsies), and with free/total PSA < 0.15 (Group 2, 75 biopsies). The extent of atrophy was evaluated considering either the absolute number or the percentage of cores showing the lesion. Polynomial regression or simple correlation were applied using in each analysis the most suitable function that best fitted to the distribution of the data.

Group 1: there was a positive and statistically significant correlation between extent of atrophy and either free ($p = 0.0076$ and $p = 0.0210$, respectively, for parabolic and linear functions) or free/total PSA ($p = 0.0068$ and $p = 0.0085$, respectively, for 4th degree and parabolic functions); no correlation was found for total PSA. Group 2: no significant correlation was found between extent of atrophy and free, total or free/total PSA.

Considering that age associated prostatic atrophy may be a manifestation of chronic ischemia due to local arteriosclerosis, the results suggest that chronic ischemia may be involved in free PSA serum level elevation in patients with several needle biopsies showing only prostatic atrophy and free/total PSA ≥ 0.15.

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INVESTIGATIVE UROLOGY

A dose-dependent dual effect of oestrogen on voiding in the male mouse?

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Objectives: To explore the effect of different degrees of oestrogenization on male voiding, by treating adult castrated and 5alpha-dihydrotestosterone (DHT)-maintained male mice with different doses of oestrogens, as exposure of male mice to excessive amounts of oestrogens can cause bladder outlet obstruction (BOO); in addition, male mice lacking oestrogen receptor (ER)alpha (ERKO) or ERbeta (BERKO) were studied to assess the importance of ER subtypes.