Short Report

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Prostate Carcinoma, what would be the best to do ?

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Abstract

Diagnosis and treatment of prostate cancer is a controversial topic. An ideal clarity has not been found to date. This concerns screening, risks, genetics as well as therapy. Another issue is the differential diagnosis to prostatitis. The aim should be freedom from schemata and instead strict individualization. to this end, it is important not to set a standard in terms of lowering testosterone to zero. There is a lack of age-related normal ranges for this purpose. If these exist, the real free testosterone can be individually brought into the normal range, which would eliminate an important risk factor.

Introduction

The world`s men are suffering. Among them, prostate cancer is the most common form of cancer and the third most frequent cause of cancer death. Many older men experience going to the urologist as the start of a career of illness with a significantly reduced quality of life. Particularly problematic: In the early stages, those affected notice little. Now doctors are increasingly looking for risk factors and sensible and gentle therapies.

Mass screening ?

Tracking down prostate carcinomas: tests for prostate-specific antigen (PSA) are offered nationwide - not without controversy. The German Society of Urology (DGU) took a stand. According to an updated S3 treatment guideline on prostate cancer, urologists are against mass screening [1]. Instead, physicians should provide men aged 45 and older with detailed information about screening examinations so that patients can make a conscious decision for or against testing. The American Urological Association (AUA) also opposes widespread screening of healthy men.

Fritz H. Schröder of Erasmus University in Rotterdam has published another study on this sensitive topic [2]. He evaluated data from the European Randomised Study of Screening for Prostate Cancer (ERSPC) involving 162,388 men between the ages of 50 and 74. All subjects were randomly assigned to a screening group or a control group. Physicians performed biopsies starting at PSA levels of 3.0 ng/ml. As expected, there was an increase in diagnoses (7,408 versus 6,107) and fewer prostate Ca-related deaths (427 versus 610). With a total of 18,251 deaths in the screening arm and 21,992 in the control group, this cancer hardly mattered numerically, Schröder writes, mass screening has not succeeded in significantly reducing overall mortality.

Genes

Commenting on the study, Keith A. Ashcraft of the University of Texas and Cathee Till of the Fred Hutchinson Cancer Research Center advise combining PSA testing with other risk factors. So far, human geneticists know that there is a clustering of prostate Ca in the family for one in ten people with the disease. Patients are particularly at risk should first-degree relatives develop the disease before the age of 60. In order to be able to provide more precise information, scientists from an international consortium studied the genes of 43,000 men with prostate cancer [3-6]. A similarly sized group of healthy study participants was used for comparison. As a result of their genome-wide association study, researchers identified a total of 100 risk genes. Depending on the pattern, the probability of developing prostate cancer increases by a factor of 2.9 to 5.7.

Risks - down to the hair

But that's not all: Michael B. Cook of the National Cancer Institute, Bethesda, found a risk factor of a special kind. He

evaluated data from 39,070 men. They had participated in the Prostate, Lung, Colorectal and Ovarian Cancer Screening Trial. Cook used questionnaires to find out a not insignificant detail, namely possible hair loss. For follow-up: within three years, physicians recorded 1,138 cases of cancer of the prostate; 572 had tumors with a Gleason score of at least seven, with clinical stage III or had even died. In fact, study participants up to age 45 with bald patches on the back of their heads or receding hairline were 39 percent more likely to develop aggressive prostate cancer [7, 8]. According to Cook, this shows the possibility of shared pathophysiological mechanisms between tumors and androgenetic alopecia. Hair roots are hypersensitive to dihydrotestosterone- and testosterone may accelerate the growth of prostate cancers. Androgen deprivation is usually considered as a therapeutic option for highly malignant carcinomas. In Cook`s study, men who received Testosteron supplementation did not have a higher rate of prostate cancer compared with the unexposed groups. Could it be that elevated testosterone is not a risk factor for prostate cancer ?

Shooting at sparrows with cannons ?

Grace Lu-Yao of Rutgers Cancer Institute in Brunswick proved that anti-androgens or castration do not make sense in low-malignant variants. Her work was based on data from more than 66,000 men over 65 with localized prostate tumors. Physicians treated 22 to 39 percent of their patients via hormone deprivation - especially those with a poorer prognosis. These included higher PSA levels, a higher Gleason score and comorbidities. Regional preferences were also present. To identify potential added value, Lu-Yao looked at areas where physicians used androgen deprivation frequently and rarely, respectively [9].

Her finding was that when patients suffered from moderately differentiated tumors, their overall survival rate after 15 years was 20 versus 21 percent - not a significant difference. Even in poorly differentiated carcinomas, data from the cohort did not suggest added value for radical therapies. Here, the researchers report an overall survival rate of 8.6 versus 9.2 percent. Grace Lu-Yao therefore advises that antiandrogenic treatment options should be reserved for patients with advanced prostate cancer.

Risk factors

The question arises : What are important risk factors for getting prostate carcinoma? First : It could be the xeno-estrogens [10]. They are found in drinking water and are the result of degradation products of birth control pills and various agricultural herbicides. To reduce this factor, a general distribution of drinking water purification devices would be appropriate. Of course, these are indicated not only with regard to harmful hormones, but also for many other pollutants in tap water. Wastewater treatment plants are not able to remove all potentially harmful substances, many of which end up in the final consumer.

Inflammation of the prostate is another risk factor. A large percentage of men over the age of 50 have prostatitis, which is predominantly caused by trichomonads or chlamydia. These venereal diseases, which used to be rather rare, have become common diseases. Prostatitis is characterized by moderately elevated PSA levels, which raise the suspicion of carcinoma, but only partially cause it. The therapy of choice is metronidazole. The sexual partner(s) should also be treated. In addition to determinations of specific antibodies in the blood, sonography of the prostate is significant. If it is predominantly echo-poor, it is likely to be an inflammation. In such cases, the urologist should not exert pressure on the organ, nor should he bother it with a biopsy. It is malpractice to consider any elevated PSA level as cancer.

Conclusion

In conventional medicine, anti-androgenic treatment, known as chemical castration, is predominantly used for proven cancer. This treatment assumes that testosterone is an important cause of the carcinoma. Several experts and naturopaths, on the other hand, dispute this. What is the solution to the problem? It includes both: any abnormal concentration of free testosterone is in need of correction. Because for every age there is an age related normal range. Laboratory physicians are required to define this normal range for each age group based on the examination of healthy men. Elevated concentrations should be lowered (low dose anti-androgens), and lowered concentrations should be increased. Prohormones such as DHEA, 7-keto-DHEA or pregnenolone serve this purpose. So, there is no scheme, only individualization.

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