Screening for Prostate Cancer

It works, but it’s not as simple as we would like it to be

Every year in the United States, roughly 250,000 men are diagnosed with prostate cancer. The American Cancer Society estimates that about 30,000 men will succumb to prostate cancer-related deaths in 2013. Other than skin cancer, prostate cancer is the most common cancer in American men and is the second leading cause of male cancer death behind lung cancer. Yet, most men with prostate cancer will neither die prematurely nor have a reduced quality of life should it remain untreated. The issue facing the medical profession today is that there is no definitive test to identify the subset of men who will suffer or die from their disease.

For the last 20 years, the serum Prostate Specific Antigens (PSA) measurement has served as the foundation for prostate cancer screening throughout North America and much of Western Europe, but recently its usefulness has been called into question.

PSA is an enzyme secreted by cells in the prostate gland and is measurable in a blood sample. For all practical purposes, it is only made by the prostate gland and is elevated during times of prostatic cellular stress when the enzyme can escape into the bloodstream. Prostate cancer usually causes an elevation in the serum PSA and therefore is useful as a screening marker. The confounding issue is that PSA can also be elevated due to benign prostatic growth (common as a man ages), prostatic inflammation (also called prostatitis) or prostatic trauma (due to various causes). In fact, the majority of men with PSA elevations are not found to have prostate cancer. Nationwide the prostate cancer detection rate based on PSA alone is only 30 percent, and much of that cancer is not clinically significant. In other words, PSA is prostate specific, not prostate cancer specific.

The plethora of potential explanations for an elevated PSA has led to criticism of the non-specific nature of the test. A race to develop a more specific prostate cancer screening test is ongoing and shows great promise but as of 2013 the serum PSA, its velocity (a misnomer for the rate of PSA increase), along with a patient’s age, race, family history and the results of a digital rectal exam is used in combination to calculate the risk of clinically significant prostate cancer.

There was consternation in some of the medical community when in 2012 the U.S. Preventative Services Task Force recommended against a PSA screening because, currently, most patients with detectable prostate cancer die of another cause. The Task Force noted that the test may result in "overdiagnosis" and "overtreatment" because "most prostate cancer is asymptomatic for life." They also noted that prostate cancer treatments involve risks of complications including erectile dysfunction and incontinence.

Most of the Task Force concerns are valid, but it is widely recognized and statistically undeniable that since the widespread use of PSA testing (introduced in 1987) the mortality rate secondary to prostate cancer has declined substantially. Also significant, albeit much less frequently measured or reported, the number of men suffering physically, financially or emotionally from metastatic prostate cancer that will not directly result in their demise has been greatly reduced. Given the long natural history of the disease, the number of men who could suffer from metastatic prostate cancer 40 years ago was painless metastatic disease. Today it is asymptomatic organ continued disease initially detached with a PSA. It is clear that not all the disease being detached currently requires treatment, but having the option to monitor the disease is preferable to discovering it when it can no longer be cured.

A recent national cancer registry study from Sweden suggests that if a PSA value at the age of 45 is below a certain level (less than .7 ng/ml), the lifetime risk of developing clinically significant prostate cancer is less than 4 percent, whereas if that value is above 1 ng/ml, the risk rises to greater than 30 percent. These numbers are certainly useful in aiding a patient and his physician in delineating a lifelong screening schedule. The same registry data indicates that a low PSA at the age of 60 confers essentially no lifetime risk of developing clinically significant prostate cancer. Discontinuing screening in this subgroup would save money, angst and the potential complications of any future biopsy.

It is clear that PSA can be a very useful screening tool if applied appropriately, but it is not as simple as we would like it to be.

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