OncoLog
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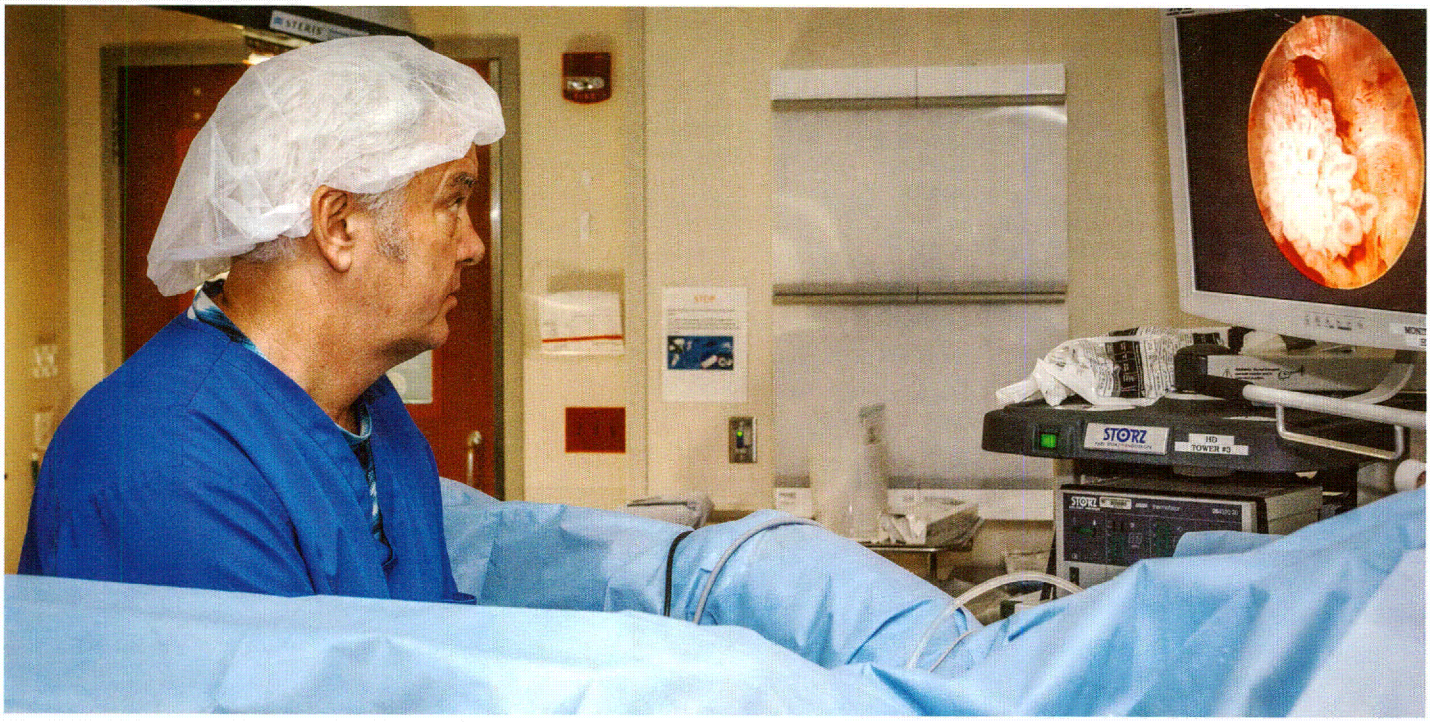
New Gene Therapy for Bladder Cancer Shows Promise

By Stephanie Deming

A gene therapy that stimulates interferon production within the body may prove to be effective against high-risk, early-stage bladder cancer when standard treatment fails.

For patients with high-risk non–muscle-invasive bladder cancer (i.e., Tis and high-grade Ta and T1 tumors) that persists or recurs despite standard treatment with bacille Calmette-Guérin (BCG), known as "BCG-unresponsive disease," treatment options are currently limited. Because of the high risk of progression to muscle-invasive disease, the safest option is cystectomy; however, many patients are understandably reluctant to undergo removal of their bladder. An alternative is second-line medical therapy, but the only U.S. Food and Drug Administration (FDA)–approved drug for BCG-unresponsive disease, valrubicin, results in a



Dr. Colin Dinney *evaluates a bladder tumor during a cystoscopy.*

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[Continued from **page 1**]

durable complete response for only 8% of patients.

"There is currently no effective salvage therapy for patients with BCG-unresponsive, high-risk non–muscle-invasive bladder cancer that can safely delay cystectomy or direct patients away from cystectomy," said Colin Dinney, M.D., a professor in and chair of the Department of Urology at The University of Texas MD Anderson Cancer Center. "So we and others are launching studies to try to find alternatives to cystectomy that are safe for patients."

Within the next few years, patients may have a more effective second-line medical treatment option: a new adenovirus-mediated gene therapy that causes cells lining the bladder to produce interferon alfa-2b.

A long road to a new treatment

Although interferon alfa-2b is an established anticancer agent, instillation of interferon into the bladder has had only limited efficacy against bladder cancer because the drug is rapidly cleared from the body in urine. Gene therapy, in contrast, causes the cells lining the bladder to produce interferon, resulting in prolonged exposure.

A group that includes Dr. Dinney and William Benedict, M.D., a professor in the Departments of Genitourinary Medical Oncology and Urology, has been working for years to develop an effective gene therapy for bladder cancer. The researchers expected bladder tumors to be ideal targets for gene therapy because the vector containing the gene can be instilled easily into the bladder and retained in the bladder for several minutes, which should allow for good contact between the vector and the bladder wall, good transfer of the vector, and good gene activity. However, the researchers discovered that the bladder's antibacterial and antiviral layer, which protects against infections, also prevents gene transfer.

A solution to this problem came in the form of Syn3, a surfactant that enhances the ability of the adenoviral vector to transduce cells in the bladder wall. Critical preclinical studies in mouse models developed by Dr. Benedict showed that delivering the gene vector together with Syn3 resulted in effective vector uptake and interferon production by both tumor cells and normal cells. "The bladder was working like a bioreactor to produce interferon, which it did for about a week," Dr. Dinney said.

> **"There is a real unmet need for new treatments for BCG-unresponsive, high-risk non–muscle-invasive bladder cancer."**
>
> **– Dr. Colin Dinney**

In the preclinical studies, the new gene therapy facilitated by Syn3 caused marked shrinkage of established bladder tumors, and three mechanisms of action were identified. First was a direct effect: some tumor cells that incorporated the gene became overwhelmed by the resulting production of interferon protein and died as a result. Second, the interferon-sensitive tumor cells—approximately 20%–25% of all tumor cells—underwent apoptosis because of their prolonged exposure to interferon in the bladder. Third and most important was a so-called bystander effect, in which a protein excreted into the urine was effective even against interferon-resistant tumor cells. The normal urothelial cells, although transduced by the vector and producing interferon, were not harmed by exposure to interferon.

The new gene therapy is the latest advance in an ongoing line of research, much of which is supported by MD Anderson's Bladder Cancer Specialized Programs of Research Excellence (SPORE) grant from the National Cancer Institute.

Early clinical trials show promise

Building on the promising preclinical findings, researchers from several institutions conducted a phase I trial of recombinant adenovirus–mediated gene therapy with interferon alfa-2b plus Syn3 for patients with non–muscle-invasive bladder cancer that recurred after treatment with BCG. The 17 patients enrolled in this dose-escalation trial received a single intravesical instillation of the gene vector plus Syn3, which was retained in the bladder for 1 hour. Patients were monitored for adverse effects for 5 days after treatment and underwent cystoscopy at 3 months. Patients with a complete response at 3 months were allowed to receive 1 more dose of the gene therapy.

No dose-limiting toxic effects were observed in the trial. All adverse effects were grade 1 or 2, and the most common adverse effect, urinary urgency, was minimized by pretreatment administration of anticholinergics in all cases.

In all patients except those treated at the lowest dose, interferon alfa-2b was detectable in urine for up to 10 days after treatment, confirming effective gene transfer. The peak levels of interferon in the urine corresponded with the number of viral particles administered. And most exciting, cystoscopy 3 months after gene therapy demonstrated a complete response in seven of the 17 patients, including six of the 13 patients who were treated at a dose of at least 1×10^{10} viral particles/mL.

Encouraged by the results of the phase I trial, Dr. Dinney and others designed an industry-sponsored multi-institutional phase II trial. In this trial, patients with high-grade, non–muscle-invasive bladder cancer were randomly assigned to receive treatment with Syn3 plus the highest or second-highest dose of the interferon alfa-2b gene therapy vector from the phase I trial (3×10^{11} or 1×10^{11} viral particles/mL). Patients underwent an initial 1-hour intravesical

instillation of the therapy, and patients who exhibited a complete response could receive up to 3 additional treatments at the same dose level at 3-month intervals, for a total of 4 doses. The trial was launched in November 2012, and accrual was completed in just over 1 year.

Of the 40 patients enrolled in the phase II trial, 23 had had disease that persisted despite BCG therapy, and 17 had been rendered disease free with BCG therapy but then had a recurrence. Unaudited interim results were reported in May at the American Urological Association's annual meeting, and results are now available for all 40 patients.

The rate of freedom from high-grade recurrence, calculated on an intent-to-treat basis, was 56% at 6 months and 35% at 12 months and was similar in the two dose groups. The study also showed that the 12-month recurrence-free survival rate of the nine patients with papillary (Ta or T1) disease alone (55%) was higher than that of the 29 patients with a component of carcinoma in situ (30%). There were only three adverse events related to the treatment: a grade 2 uncomplicated urinary tract infection, a case of grade 3 diarrhea in a patient with a history of diarrhea, and a case of grade 3 acute renal failure resulting from dehydration due to a urinary tract infection.

Larger trial in the works

As a result of the phase II trial's success, Dr. Dinney and others are currently working with the FDA to design a phase III registration trial of recombinant adenovirus–mediated interferon alfa-2b plus Syn3 in patients with BCG-unresponsive high-risk non–muscle-invasive bladder cancer. The trial, slated to begin in early 2016, is expected to enroll approximately 100 patients at 35 centers. The trial will have two unconventional features. First, it will have no control arm because outcomes with the currently approved second-line drug therapy, valrubicin, are relatively poor. Instead, all patients will receive gene therapy, and results will be compared with benchmarks approved by urologic oncologists and the FDA. Second, the trial will include a mixed population of patients with carcinoma in situ and patients with high-grade papillary disease, a patient combination the FDA has not allowed in previous phase III bladder cancer trials.

"The bladder was working like a bioreactor to produce interferon."

– Dr. Colin Dinney

The criterion for determining whether the new gene therapy should be approved remains to be identified. Dr. Dinney expects this criterion to be a high-grade recurrence–free survival rate of at least 25% at 12 months. "That would be meaningful," he said, "because valrubicin had a complete response rate of about 20% at 3 months and a durable response rate of about 8%."

The planned phase III trial will also identify biomarkers linked to response to the new gene therapy. Currently, David McConkey, Ph.D., a professor in the Department of Urology, and Xifeng Wu, M.D., Ph.D., a professor in and chair of the Department of Epidemiology, are analyzing pretreatment and posttreatment urine and tissue samples from the phase II trial to identify potential biomarkers. "One of the possible biomarkers we're looking at in the clinical trial specimens is whether or not adenoviral interferon caused upregulation of TRAIL (a cytotoxic cytokine) in urine or tumors or maybe both and whether the urine levels of TRAIL or the length of time TRAIL was detectable in urine correlates with clinical responses," Dr. McConkey said. Any promising markers will be further studied in the phase III trial.

Also, work in Dr. McConkey's laboratory has shown that interferon alfa upregulates the immune mediator programmed cell death ligand 1 (PD-L1) in human bladder cancer cells, so samples obtained before and after treatment will be analyzed to determine whether there is a change in the level of PD-L1 or its binding partner, programmed cell death 1 (PD-1). "We hypothesize that we might be able to get even better clinical activity if we combine adenoviral interferon with antibodies that block PD-L1 or PD-1, which might limit the immune response," Dr. McConkey said.

"There is a real unmet need for new treatments for BCG-unresponsive, high-risk non–muscle-invasive bladder cancer," Dr. Dinney said. "The research we're doing could change the field." ■

"We hypothesize that we might be able to get even better clinical activity if we combine adenoviral interferon with antibodies that block PD-L1 or PD-1."

– Dr. David McConkey

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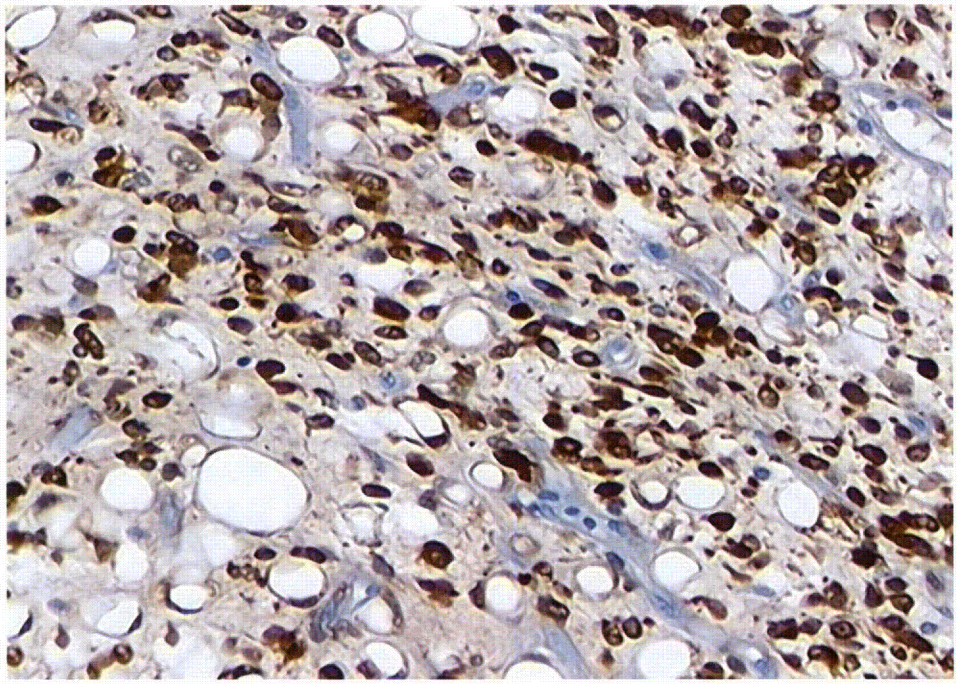
Vaccine Helps T Cells Target Sarcomas, Mela

By Bryan Tutt

A vaccine that delivers an antigen to dendritic cells, in turn activating killer T cells that can target specific cancers, is the subject of two ongoing clinical trials.

The vaccine, LV305, is a lentiviral gene vector that specifically binds to dendritic cells in a patient's body via surface receptor CD209 (also called DC-SIGN) and introduces the full length of the NY-ESO-1 antigen into these cells. The dendritic cells then present the antigen to CD8-positive T lymphocytes via the major histocompatibility (MHC) class I molecules on the cell surface. The activated CD8+ cells can then recognize and attack cancer cells that express NY-ESO-1.



Most myxoid liposarcoma cells show strong nuclear immunoreactivity for the cancer/testis antigen NY-ESO-1. Reprinted with permission from Endo M, et al. Mod Pathol. *2015;28:587–595. © 2015 Macmillan Publishers Ltd.*

A target for immunotherapy

The cancer/testis antigen NY-ESO-1 is highly expressed in spermatogonia during embryogenesis, but in adults the antigen is expressed only in testicular germ cells and in several types of cancer. The highest rates of NY-ESO-1 have been seen in certain sarcoma subtypes: 80%–100% of myxoid/round cell liposarcomas and synovial sarcomas express NY-ESO-1. The antigen is also expressed in about 40% of melanomas and up to 20% of breast, ovarian, and non–small cell lung cancers.

NY-ESO-1 is considered a good target for immunotherapy because NY-ESO-1–expressing cancer cells—but not healthy germ cells—express MHC restriction elements that are recognized by cytotoxic T lymphocytes. "If you have a T cell response against NY-ESO-1, there should be no negative immunological effects in the body except against the tumor," said Neeta Somaiah, M.D., an assistant professor in the Department of Sarcoma Medical Oncology at The University of Texas MD Anderson Cancer Center. LV305 is one of several new treatments aimed at inducing such a response.

Dr. Somaiah said that LV305 compares favorably with other technologies that target NY-ESO-1–positive tumors. One such technology is adoptive T cell therapy, in which T cells that have been genetically modified to recognize a specific peptide of NY-ESO-1 are infused into the patient. Although the adoptive T cell therapy has shown promising results, it is human leukocyte antigen (HLA)–specific. Most adoptive T cell therapy approaches target the NY-ESO-1 peptide presented by HLA-A*02:01 and therefore are limited to patients with that specific HLA type. The technique also requires specialized centers for the expansion and administration of T cells. In contrast, the LV305 vaccine could be administered at any center and, since the vaccine induces in T cells the expression of full-length NY-ESO-1, is not restricted by HLA type.

First-in-human trial

Last year, an ongoing multi-institutional, first-in-human trial of LV305 began enrolling patients with locally advanced or metastatic sarcoma, melanoma, ovarian cancer, non–small cell lung cancer, or breast cancer whose biopsy specimens show NY-ESO-1 expression in at least 5% of the tumor cells. Another eligibility requirement is low tumor burden. "Patients with bulky or rapidly progressing disease might be immunosuppressed and might not be able to generate an immune response fast enough to see a benefit from LV305 as a single agent," said Dr. Somaiah, MD Anderson's principal investigator for the trial.

Patients in the open-label study receive 3 or 4 intradermal injections of LV305 given at 3-week intervals. In the

nomas, and Other Tumors

"If you have a T cell response against NY-ESO-1, there should be no negative immunological effects in the body except against the tumor."

– Dr. Neeta Somaiah

trial's dose escalation arm, which has completed enrollment, patients received doses of 1×10^8, 1×10^9, or 1×10^{10} vector genomes per injection.

All 12 patients in the dose escalation arm had sarcoma with NY-ESO-1 expression levels ranging from 6% to 100%. Eleven of these patients completed the full course of LV305 treatment. One patient had progressive disease after his second LV305 injection and discontinued the trial to begin a different therapy.

Dr. Somaiah, who presented the trial's preliminary results at the 2015 American Society of Clinical Oncology Annual Meeting, said that eight of the 11 patients for whom immunological data were available had a doubling in the number of CD4+ cells and/or $CD8^+$ cells against NY-ESO-1 (five had a $CD4^+$ cell response and six had a $CD8^+$ cell response). Four of the six patients with a $CD8^+$ cell response received the middle or high dose of LV305, indicating a possible dose-response relationship similar to that seen in preclinical models. Additional immunological studies in one patient revealed an increase not only in the number of NY-ESO-1–specific $CD8^+$ cells but also in their binding affinity for NY-ESO-1 and their ability to recognize multiple NY-ESO-1 epitopes.

Eight of the 12 patients had stable disease at last follow-up, and one patient had tumor regression of around 14%. "The clinical and immunological response data are encouraging and warrant further study," Dr. Somaiah said.

As expected, side effects were minimal and included mild discomfort at the injection site and fatigue. No dose-limiting toxic effects were observed, so the highest dose of LV305, 1×10^{10} vector genomes, will be used for the ongoing expansion arm of the trial.

Further research

The expansion arm of the first-in-human trial will include six patients each with sarcoma, melanoma, non–small cell lung cancer, and ovarian cancer. The sarcoma cohort is full; however, Dr. Somaiah said, sarcoma patients whose tumors express NY-ESO-1 may be eligible for a new combination therapy trial that recently began enrollment at MD Anderson and other institutions.

In the new trial, LV305 is given sequentially with G305, a full-length NY-ESO-1 protein mixed with a synthetic TLR4 agonist, glucopyranosyl lipid A. In an early trial, G305 demonstrated NY-ESO-1–specific $CD4^+$ cell and antibody responses in patients with NY-ESO-1–positive tumors.

The sequential use of LV305 and G305 (called CMB305) is designed to produce NY-ESO-1–specific $CD8^+$ cell, $CD4^+$ cell, and antibody responses. The eligibility requirements of the CMB305 trial are similar to those of the LV305 monotherapy trial.

Future studies may combine CMB305 with a programmed cell death 1 (PD-1) inhibitor in patients with NY-ESO-1–positive tumors.

"Our early results show that LV305 is safe and generates an immune response," Dr. Somaiah said. "Future studies will determine the best combination and sequence of agents to generate an effective and durable immune response with a robust antitumor effect." ■

FOR MORE INFORMATION

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CLINICAL TRIALS: NY-ESO-1–Expressing Tumors

A phase I, open-label clinical trial evaluating the safety, tolerability, and immunogenicity of intradermally administered ID-LV305 in patients with locally advanced, relapsed, or metastatic cancer expressing NY-ESO-1 (2013-0823). Principal investigator (PI): Dr. Neeta Somaiah. The goals of this study are to test the safety of different doses of LV305 in patients with locally advanced or metastatic cancer and to see if LV305 causes patients' immune systems to react against the cancer.

A phase IB study evaluating the safety, tolerability and immunogenicity of CMB305 (sequentially administered LV305 and G305) in patients with locally advanced, relapsed, or metastatic cancer expressing NY-ESO-1 (2014-0952). PI: Dr. Somaiah. The goal of this study is to assess the safety of CMB305 in patients with locally advanced, relapsed, or metastatic cancer. ■

FOR MORE INFORMATION

Visit www.clinicaltrials.org.

Glypican-1 Shows Promise as a Biomarker for Pancreatic Cancer

An exosome-derived biomarker may be useful for the detection and diagnosis of early-stage pancreatic cancer, according to the findings of a recent study led by researchers from The University of Texas MD Anderson Cancer Center.

The biomarker, circulating cancer cell–derived exosomes (crExos) enriched in the protein glypican-1 (GPC1+), could serve as a noninvasive screening tool, the multinational team of researchers reported. "GPC1+ crExos were detected in small amounts of serum from about 250 patients with pancreatic cancer with absolute specificity and sensitivity, importantly distinguishing patients with early- and late-stage pancreatic cancer from those with chronic pancreatitis," said Raghu Kalluri, M.D., Ph.D., a professor in and chair of MD Anderson's Department of Cancer Biology and the study report's senior author.

The researchers first established that GPC1, a membrane-anchored protein that is overexpressed in breast and prostate cancer cells, is a specific marker of cancer exosomes—virus-sized extracellular vesicles that are secreted by cancer cells and contain DNA, RNA, and proteins.

The team then isolated crExos from blood samples from 190 patients with pancreatic ductal adenocarcinoma (the most common form of pancreatic cancer) and 100 healthy donors and found that the levels of GPC1+ crExos in the cancer patients were significantly higher than those in the healthy donors, indicating a strong correlation between GPC1+ crExos and pancreatic cancer.

"Our findings present an unprecedented opportunity for informative early detection of pancreatic cancer."

– Dr. Raghu Kalluri

Further analysis revealed that the levels of GPC1+ crExos were consistently higher in patients with histologically validated pancreatic cancer precursor lesions than in healthy donors and patients with benign pancreatic disease and could be used to distinguish these groups. The researchers validated these findings in an independent cohort of 56 patients with pancreatic cancer, six patients with histologically confirmed benign pancreatic disease, and 20 healthy donors.

If detected in its early stages, pancreatic cancer can be cured with a pancreatoduodenectomy (Whipple procedure); however, because pancreatic cancer is often diagnosed at its later stages, only about 15% of patients qualify for such surgery.

"Studies comparing stage of disease with outcome following surgery suggest that death rates for pancreatic cancer would be reduced if the disease were diagnosed at an earlier stage," Dr. Kalluri said. "Our findings present an unprecedented opportunity for informative early detection of pancreatic cancer."

The study's findings were reported in the June 24 issue of *Nature*. ■

Stereotactic Ablative Radiation Therapy for Stage I Non–Small Cell Lung Cancer May Offer Survival Benefit

Stereotactic ablative radiation therapy (SABR) for stage I non–small cell lung cancer (NSCLC) achieved a higher rate of overall survival than did invasive surgery, according to a combined analysis of two clinical trials.

The two randomized controlled trials, a multicenter study from the Netherlands and a multinational study conducted at The University of Texas MD Anderson Cancer Center and elsewhere, were the first to compare SABR and surgery head-to-head in NSCLC patients. In both trials, patients with operable stage I NSCLC were randomly assigned to undergo SABR or the standard of care, which is lobectomy with mediastinal lymph node dissection or sampling.

"SABR appears to be better tolerated [than surgery] and might lead to better survival outcomes for these patients."

– Dr. Joe Chang

Thirty-one patients underwent SABR, and 27 underwent surgery. The median follow-up time was 40.2 months. The estimated 3-year overall survival rates were 79% for the surgery group and 95% for the SABR group ($p = .037$). No significant difference was seen in recurrence-free survival.

Three patients who underwent SABR experienced grade 3 adverse events, whereas 12 patients who underwent surgery experienced grade 3 or 4 adverse events. One patient died of surgical complications.

"For the first time, we can say that the two therapies are at least equally effective and that SABR appears to be better tolerated and might lead to better survival outcomes for these patients," said Joe Y. Chang, M.D., Ph.D., a professor in the Department of Radiation Oncology and lead author of the study's report. However, he said that the study's findings should be interpreted with caution because of its small patient sample size and limited follow-up time.

Dr. Chang added that two larger trials comparing SABR and surgery for patients with NSCLC are scheduled to open later this year, one in the United States and one in the United Kingdom.

The study's report was published in May in *The Lancet Oncology*. ■

Prostate Cancer Screening

Who should get screened and when

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Prostate cancer is the second leading cause of cancer-related deaths among men in the United States each year. But many men are unsure whether or when they should be screened for the disease.

Doctors at The University of Texas MD Anderson Cancer Center suggest that men at risk of prostate cancer talk to their health care providers to discuss whether screening is appropriate and which tests should be used.

The goal of screening is to detect prostate cancer early—before it causes symptoms or spreads to other parts of the body. Symptoms of prostate cancer include lower back pain, problems with urination, and erectile dysfunction. However, these symptoms may not appear before the cancer is advanced.

Screening guidelines

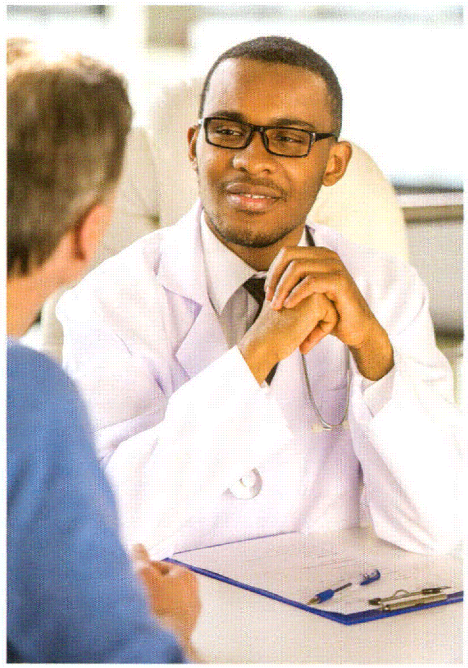
Who should be screened for prostate cancer and at what age depends on the individual's risk factors for prostate cancer. Each individual should discuss the general recommendations below with his own doctor when choosing whether to be screened.

Increased risk

Men are at increased risk of prostate cancer if they are African American or have a family history of prostate cancer. Men 40 years or older at increased risk for prostate cancer should meet with their primary care physicians to discuss whether screening is a good idea. MD Anderson recommends screening for prostate cancer every year starting at age 45 years for men at increased risk.

Average risk

Men without a family history of prostate cancer who are not African American are at average risk of the disease. For such men, MD Anderson recommends annual prostate cancer screening between the ages of 50 and 75 years. Men 76–84 years old should talk to their physicians about screening.



MD Anderson recommends that men 85 years or older not be screened.

Screening tests

Two tests are typically used to screen for prostate cancer. One is the prostate-specific antigen (PSA) test. This simple blood test measures the amount of PSA (a protein produced in the prostate) in the blood. High amounts of PSA indicate a high risk of prostate cancer. Furthermore, an increase in PSA over time can be a warning sign. MD Anderson recommends that an individual track changes in his PSA over time to determine whether further diagnostic tests are needed. It is important to note that a high PSA number does not always indicate prostate cancer; it can also mean an enlarged prostate or other prostate problems. On the other hand, a low PSA number does not rule out the possibility of prostate cancer.

The other test, usually used in conjunction with the PSA test, is the digital rectal exam. In a digital rectal exam, a physician checks through the lower rectum for abnormal prostate findings such as lumps. The PSA test and digital rectal exam together provide more accurate screening than either test used alone.

Follow-up tests

If the PSA test and digital rectal exam reveal something abnormal, tests such as transrectal ultrasonography, transrectal magnetic resonance imaging (MRI), and transrectal biopsy can determine whether or not the abnormality is prostate cancer.

During a transrectal ultrasound examination, a probe is inserted into the rectum, and sound waves produce a picture of the prostate.

Transrectal MRI produces a more detailed picture of the prostate and the area around it than does ultrasonography, but MRI is more expensive. The MRI procedure also involves inserting a probe into the rectum.

When MRI or ultrasonography shows an abnormal area on the prostate, a transrectal needle biopsy is done to remove a small sample of tissue from that area. The biopsy is often done with guidance from transrectal ultrasonography. The tissue is viewed under a microscope to see if the cells are cancerous.

If the tests indicate that a man has prostate cancer, he should talk with his doctor to determine whether treatment or watchful waiting is the best course of action. Not all patients with prostate cancer require immediate treatment, and those who do may have several options. ■

– K. Nair

FOR MORE INFORMATION

- *Ask your physician*
- *Read MD Anderson's prostate cancer screening guidelines at www.mdanderson.org/patient-and-cancer-information/cancer-information/cancer-topics/prevention-and-screening/screening/prostate.html*
- *Call* askMDAnderson *at 877-632-6789*

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USEFUL RESOURCES

Cancer Survivorship Algorithms

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As treatments for numerous cancer types continue to improve, an increasing number of cancer survivors are transitioning from their oncology teams back to their primary care physicians. The health care needs of these cancer survivors can vary greatly according to cancer type. To help physicians address these needs, The University of Texas MD Anderson Cancer Center offers a series of algorithms depicting best practices for the care of cancer survivors.

Currently, survivorship algorithms are available for lymphoma, germ cell testicular cancer, head and neck cancer, cutaneous melanoma, and cancers of the breast, anus, colon, rectum, bladder, kidney, penis, prostate, cervix, endometrium, ovary, and thyroid. Multiple algorithms are available for cancers with varying subtypes, such as lymphoma and head and neck cancer. The algorithm for each cancer type was developed by a multidisciplinary work group of MD Anderson physicians with expertise in that type of cancer.

The algorithms offer guidelines for surveillance to detect recurrences, monitoring for late effects of therapy, reducing the risk of recurrence, and psychological assessments for cancer survivors. With each algorithm comes a list of suggested readings for practitioners who wish to learn more about survivorship for that particular type of cancer. Separate algorithms are available for the management of bone health in survivors of breast, thyroid, and gynecologic cancers.

The survivorship algorithms, which can be downloaded as PDF files for easy printing, are available at www.mdanderson.org/education-and-research/resources-for-professionals/clinical-tools-and-resources/practice-algorithms/survivorship-algorithms.html. ■

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