Increasing the Pool of Academically Oriented African-American Medical and Surgical Oncologists

The commendable article by Newman et al.,1 “Increasing the Pool of Academically Oriented African-American Medical and Surgical Oncologists,” highlights an issue worthy of serious consideration. Not only must more effort be expended to raise the interest of African American medical students and physicians in academic oncology, but the disproportionately low representation of African Americans in medical school also must be recognized as a partly (if not wholly) systemic failure worthy of corrective attention. The oncology community should take an objective look at the factors that gave rise to and continue to perpetuate an unrepresentative oncology workforce excluding African Americans. This state of affairs arose historically on the basis of unabashed white privilege justified by illusions of inseparable racial superiority and inferiority. The beneficiaries of such apartheid-scale privilege are understandably loath to know the magnitude to which their sometimes stellar achievements stand diminished by such contextual foundations of cultural insensitivity and exclusion. Even while notable contemporary academic oncologists condemn the cancer disparities that affect the populations they treat, there is insufficient general recognition of the disparity among too many academic oncologists who were largely self-appointed to the near exclusive privilege of managing the social and institutional resources mandated to research solutions. Scarcely 1% of American medical oncologists are African American. Undoubtedly, this is the case in other oncology subspecialties as well. These figures are best approximations, because no thorough demographic study of the oncology workforce has been published. Our preeminent cancer specialty society, the American Society of Clinical Oncology,2 described its membership in 2000 as including over 11,000 medical oncologists, with only 94 who described themselves as African American. In academic oncology, surrogate indicators of ‘race’ appear to have supplanted overt racism, preserving deterrents to representative inclusion of African Americans in faculty positions with ‘color-blind’ precision. Because networks of these faculty members influence oncologist staffing in metropolitan community hospitals, the basis for market exclusion of African American cancer specialists is established in communities where African Americans live in large numbers. A superficial validation for this state of affairs may suggest that I am criticizing a rigorously sound meritocracy. The failing of such a conclusion lies in the prevalence of disparities in cancer mortality among the populations being treated by mostly commendable healers who have altruistic intentions with socially evolved boundaries to requisite cultural experiences. I have labored to believe that a better job would be done by all of us working together with curative intentions in...
addressing all of the disparities that constitute society’s cancer burden.

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We appreciate Dr. Taylor’s concerns, in response to our previous article,1 that the lack of ethnic diversity within the medical oncology community, and in particular the notable deficit in African American oncologists, has contributed to the ethnicity-related cancer outcome disparities that have been observed in the United States. It is appropriate for this issue to receive increased scrutiny at this time, when the legality of affirmative action is once again facing judicial challenge. As a medical oncology community, we must either be willing to accept our own limited ethnic heterogeneity and the resulting potential for increased difficulty in delivering optimal care to an ethnically diverse patient population, or we must be proactive in strengthening the diversity of our own ethnic composition. The demographics of the American population are shifting in the direction of larger minority-ethnicity communities. These trends cannot be influenced by the medical community (nor would we want them to be); however, we can exert some measure of control over the extent to which the members of the medical community reflect this diversity. Patient compliance and implementation of clinical trials are completely dependent on patient trust. If we accept the concept that our patients are likely to more readily trust a health care community that reflects their own diversity in culture, values, and ethnicity, then it is imperative that we aggressively address and correct any deficiencies in these areas. This can be accomplished with energetic mentorship and recruitment efforts.

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Mediastinal Irradiation in Patients with Esophageal Carcinoma after Heart Transplantation

In this correspondence, we report our experience using mediastinal irradiation in the treatment of a patient with esophageal carcinoma 8 years after heart transplantation. The number of heart transplantations performed appears to increase every year. The survival of these patients is reportedly improved with the use of cyclosporins for immunosuppression.1 Some of these patients are long-term survivors and the risk of treating them in oncology departments increases in time. As many as 30% of patients who undergo organ transplantation develop precancerous lesions and malignant tumors.2 If the cancer requires a radiation target-volume involving parts of a transplanted heart, a number of questions arise for the radiation oncologist.3 The tolerance of the heart for radiation has been studied previously,4,5 but to our knowledge no sufficient data have been collected to date.3

A male patient age 52 years who had a history of chronic tobacco use and alcohol intoxication presented to the study institution. The patient had undergone a heart transplantation in 1993 for end-stage ischemic cardiomyopathy with left ventricle severe dysfunction. After the transplantation, the patient was treated with cyclosporin, azathioprin, and corticoids. In January 2002, the patient presented with dysphagia, weight loss, and impaired performance status (PS)2. Fibroscopy demonstrated stenosis and a large tumor (measuring 40 mm × 30 mm × 25 mm) situated within 30 cm of the dental arch. Biopsy was performed and demonstrated squamous cell carcinoma. There were no peritumoral lymph nodes detected on the
Computed tomography (CT) scan, but some mediastinal lymph nodes were found. There were no distant metastases. After discussion between physicians in multiple disciplines, Santy-Lewis surgery was performed. Finally, the tumor was classified as pT3pN2R1 (according to the TNM Staging System). There were 20 positive lymph nodes found. After a second multidisciplinary discussion, postoperative radiotherapy was proposed. Conformal radiotherapy was performed using an 18-megavolt (MV) accelerator. A total dose of 45 grayes (Gy) in 25 fractions was delivered to the reference point. Because of the higher risk to the transplanted heart, the cardiac structures were delineated in all CT slides. The heart received a median dose of 8.6 Gy (range, 0.34–43.6 Gy). Approximately 85% of the cardiac volume received <25 Gy. A strict program of cardiac examinations (comprised of electrocardiogram, cardiac ultrasound, ejection fraction, standard blood tests, and cardiac enzymes) before, during, and after radiotherapy was established. The radiotherapy was well tolerated with Radiation Treatment Oncology Group (RTOG) Grade 2 dysphagia reported without any cardiac symptoms. A full cardiac examination was performed at 1 month, 3 months, and 6 months after irradiation and demonstrated no changes compared with pretreatment examinations (the left ventricular ejection fraction was 63%, and all cardiac enzymes were normal). Complete remission of the esophageal carcinoma was obtained. One month after last follow-up (7 months after radiotherapy), the patient died (by suicide).

Mediastinal irradiation after heart transplantation is feasible and should be considered after evaluation of the risk. Conformal radiotherapy or intensity-modulated radiotherapy appears to be the most appropriate treatment.

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Hospitalization of an Oncology Patient Suspected of Having Severe Acute Respiratory Syndrome

A Setup for an Infection Control Quagmire at a Comprehensive Cancer Center

Severe acute respiratory syndrome (SARS), caused by the SARS coronavirus, has emerged as a grave public health concern worldwide.1 Due to the potential for nosocomial transmission to patients2 and healthcare workers (HCWs)3 and the ability of SARS to lead to progressive and potentially fatal pneumonia without known effective therapy,4 especially in high-risk individuals,5 infection containment strategies are of the utmost importance. Recently, a patient suspected of having SARS required hospitalization at our tertiary cancer center (M.D. Anderson Cancer Center, Houston, TX).

A 48-year-old male with Hodgkin disease in clinical remission presented with 1 day of fever (39.0 °C), nonproductive cough, nasal congestion, headache, and photophobia. His symptoms commenced while he was returning from a trip to Bangkok, Thailand; Kuala Lumpur, Malaysia; and Singapore in June 2003. He appeared acutely ill and had a white blood cell count of 4000 cells/μL (lymphocyte count, 960 cells/μL), and a chest radiograph showed interstitial infiltrates in the right lower lobe. Respiratory (nasopharyngeal [NP]) samples were evaluated for influenza A and B, parainfluenza, respiratory syncytial virus, human cytomegalovirus, and SARS coronavirus. Moxifloxacin (400 mg) and trimethoprim-sulfamethoxazole (15 mg/kg) were administered empirically.
On the patient's arrival to the emergency center (EC), he promptly was secured in a negative air pressure (NAP) room. A security guard supervised strict compliance to 1) a patient contact log; 2) airborne precautions (N-95 mask); and 3) contact precautions (double gloves, double gowns, and disposable eye shields). The outer contact layers were disposed of inside the room; the inner layers and eye shields were discarded immediately outside the room. A single nurse was assigned, and the patient was admitted to the NAP room with a ventilation system that was separate from units that housed patients with hematologic malignancies and hematopoietic stem cell transplant recipients. Before further use, the EC room first was decontaminated thoroughly with 10% bleach solution and then decontaminated again 12 hours later.

The patient's spouse visited daily and had restricted institutional access (between the patient's room and the parking garage, with the spouse wearing an N-95 mask). All patient-contact HCWs were retested for N-95 mask seal adequacy. Pulmonary consultation was obtained; portable mechanical ventilation and cardiovascular monitoring units were secured. The primary oncologist provided non-patient-contact guidance. Influenza A antigen was detected in NP samples, and treatment with amantadine (100 mg) plus oseltamivir (75 mg) was initiated. Nonetheless, due to concerns regarding concomitant infection,5 infection containment measures remained uninterrupted. SARS coronavirus cultures and serology assays yielded negative results; the presence of influenza A serotype H3N2 was confirmed. The patient was discharged 72 hours after admission.

Due to uncertain infectious inoculum and potential airborne routes of person-to-person transmission,5,6 devising effective infection containment strategies is a daunting task, especially at institutions with prominent high-risk populations. The cautious infection containment approach presented includes 1) designated hospital units with secured ventilation systems; 2) restricted institutional access for patients, family members, and HCWs with prolonged patient exposure; 3) identification of primary and backup teams, including infection control personnel, infectious disease, and pulmonary and critical care physicians and nurses; and 4) continuation of infection control measures until the results of specific SARS coronavirus diagnostic tests become available. These measures may serve as an outline for preventing the potentially devastating nosocomial spread of SARS in centers that provide care for high-risk patients.

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