PARAAORTIC LYMPH NODE METASTASIS AS A SOLE SITE OF FAILURE FROM CERVIX CANCER: CURABLE OR INCURABLE DISEASE?

To The Editor: Grigsby *et al.* (1) have recently reported upon recurrent carcinoma of the cervix exclusively in paraaortic lymph nodes, arriving at the unhappy conclusion that, "even with treatment, this condition is uniformly fatal." I wish to present a case that proves an exception to this rule, and discuss briefly certain implications.

Case Report: The patient was a 50-year-old white female when first presented to me in September 1989. A left paraspinal mass at the level of T10 had been noted in a patient complaining of progressive left back or flank pain for 6 months and on September 1, 1989 needle biopsy was positive for squamous cell carcinoma. Bone scan was positive at T10 and computed tomography (CT) scan of the chest demonstrated the 4.5 × 3 cm left paraspinal mass invading the vertebra. The patient had been treated elsewhere by total abdominal hysterectomy bilateral salpingo-oophorectomy (TAH-BSO) followed by intracavitary brachytherapy for Stage IB squamous cell carcinoma of the cervix in June 1987. The patient at this time apparently had a solitary paraaortic lymph node metastasis; she was informed at a tumor conference at the original treating institution that she was incurable and could expect to be dead within 6 months. I noted the patient to have no other identifiable sites of disease, no other significant medical illnesses, lifetime nonsmoker, and a Karnofsky status of 90–100. I took the viewpoint that the patient's situation was serious but not necessarily incurable, and offered her the option of aggressive treatment. The patient received 45 Gy TD in 25 fractions through a posterior anterior (PA) field entirely including the spinal cord with total dose (TD) figured at 7 cm depth in a moderately obese patient. A concomitant/sequential boost was delivered (five fractions concomitant with a 4 h split, five fractions sequential) through an oblique field designed to cover tumor apparent on CT scan with narrower margin and splitting the spinal cord, 15 Gy TD in 10 fractions. The tumor would have received 60–65 Gy and the spinal cord a steep gradient of 50–60 Gy overall. The patient had been cautioned that there was a hazard of radiation damage to the spinal cord in the informed consent process, at the same time pointing out that this was also threatened by the tumor. The tumor was successfully eradicated by the treatment described, and the patient has been seen regularly in follow-up, remaining disease free at greater than 4 years following treatment. No symptoms of spinal cord damage have appeared. The patient did experience some mild fibrosis of the left paraspinal musculature that sometimes caused muscle spasm or pain, generally relieved by ibuprofen.

I consider the most likely basis for success in my series of 1 patient vs. uniform death in the series of 20 patients reported by Grigsby *et al.* (1) to be a matter of dose response. The patient noted in my case report received 60 Gy TD to her tumor; the 20 patients reported by Grigsby *et al.* (1) received a mean TD of 42.6 Gy. C. Perez *et al.* (3) had earlier reported data suggesting a dose–response relationship for local control of squamous cell carcinoma of the cervix in which doses less than 60 Gy to point A gave poor control in the pelvis. In the recent report by Grigsby *et al.* (1) there is also some suggestion of dose response in that patients treated to > 45 Gy TD had longer median survival than patients treated to < 45 Gy TD.

A major concern in treatment which necessarily burdens the spinal cord with radiation dose is the medical hazard of spinal cord injury and the medico-legal hazard which exists because of the textbook spinal cord tolerance of 45 Gy. Marcus and Million (2) have put their clinical data together with some from other sources to suggest that the real risk of spinal cord injury in the dose range of 50–60 Gy is about 1%. Schultheiss *et al.* (4) have studied radiation-induced spinal cord injury using the rhesus monkey model and 2.2 Gy fraction size; they estimate the dose for 50% showing damage to be 76 Gy and the dose for 1% risk of myelopathy to be 59 Gy. If the oncologist can trust his patient, a potentially curative treatment plan should be considered for solitary paraaortic lymph node mets; I submit that the dose required for the attempt is of the order of 60 Gy. It is reasonable to do some thinking with treatment planning to create a dose gradient between the spinal cord and the tumor.

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NOMOGRAM FOR PREDICTING THE RISK OF NODE INVOLVEMENT IN PROSTATE CANCER, GIVEN PRETREATMENT PROSTATE-SPECIFIC ANTIGEN AND GLEASON SCORE

To the Editor: A recent report by Partin *et al.* (1) based on 703 men who had undergone radical prostatectomy demonstrated the usefulness of pretreatment prostate specific antigen (PSA) and Gleason score (GS) in predicting final pathologic stage. Roach *et al.* (2) applied an equation empirically derived from data of Partin *et al.* to more than 200 men who underwent radical prostatectomies, and used the equation to predict the risk of nodal involvement. Their equation is as follows (N+ = risk of nodal involvement):

$$N+ = (2/3) \times PSA + (GS - 6) \times 10 \qquad \text{(Eq. 1)}$$

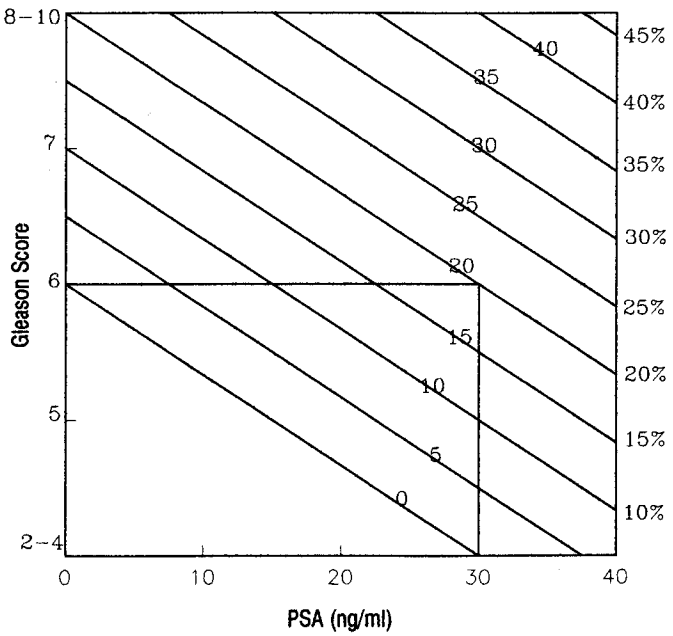

Fig. 1. Nomogram for predicting risk of node involvement in prostate cancer patients, given PSA and Gleason score, and an example of use of the nomogram. Prostate specific antigen and Gleason score are plotted on the x and y axes, respectively. The diagonal lines are isorisk lines, labeled with the percent risk of node involvement they represent. This percent risk is also repeated on the right vertical axis of the graph. For instance, the isorisk line labeled 20 is the 20% isorisk line. Example: a hypothetical patient has a PSA of 30 and a Gleason score of 6. The perpendicular lines constructed from these values intersect at the 20% isorisk line. Therefore, this hypothetical patient has a 20% risk of node involvement.

Roach *et al.* presented their data in tabular form. We have used their equation to create a nomogram (Fig. 1) which may expedite the determination of the risk of node involvement. We also provide an example of use of the nomogram.

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RESPONSE TO DRS. LEHRER AND SONG

To the Editor: Drs. Lehrer and Song are to be commended for their attempts to adapted published data to their specific needs (3). The authors should be aware however, that the published report by Partin *et al.* already contains nonograms (1). These nomograms are emprical and are likely to be more accurate than the nomogram generated by the lymph node equation. Furthermore, Partin *et al.* include in their report a nomogram that takes into account clinical stage, as well as PSA and Gleason score. I derived the empirical equations because they could be useful for several practical reasons:

1. They can be easily memorized. Note that the similar general form is the same in all three equations. Using these equations, we can avoid the need to carrying nomograms around for reference.
2. The equations could be used with a computer to retrospectively and prospectively compare and randomize patients undergoing radical prostatectomy and/or radiotherapy.
3. Most importantly, using the equaton(s), I was able to confirm the observations made by Partin *et al.* regarding the relationship between PSA and Gleason Score and pathologic stage.

Again, Partin and co-workers are to be commended for their major contribution to the literature. I don't think that Drs. Lehrer and Songs nomogram, derived from my Eq. 2, that was derived from Partin's nomogram, adds significantly to what has already been published, unless somehow it helps them confirm the general applicability of their nomogram to their own patients.

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