CORRESPONDENCE

REASON TO SCREEN FOR PROSTATE CANCER BASED ON SELECTIVE REFERENCING

To the Editor:

In their article, Ransohoff et al. describe why prostate cancer screening is so common, even though the evidence supporting screening is uncertain (1). We agree completely with the points made and with their conclusions. The authors overlook one important factor, however, and involuntarily demonstrate this omission in their article: the selective use of references.

They refer to the landmark study by Holmberg et al. (2), mentioning that "for persons with cancers discovered by means other than screening, surgical therapy may reduce prostate cancer mortality compared with watchful waiting" (1). In doing so, they omit the important remarks by Holmberg et al. regarding the probable lower effect of radical treatment in prostate cancers detected by screening owing to the lower baseline risk of death from prostate cancer (2). Moreover, they do not cite the most important conclusion—in our view—of the Holmberg study, which was "surgical therapy does not reduce overall mortality compared with watchful waiting" (2).

Although Ransohoff et al.'s citation of Holmberg et al. is correct word for word, an optimistic interpretation of their data is given, thus formulating a view on treatment of localized prostate cancer that is too optimistic. For clinicians, this might be another positive reason to screen their patients for prostate cancer. We believe that this is not sufficiently founded.

Readers may easily overlook the major nuances of a referenced article. Therefore, we think that it is important for authors and editors to guarantee the accuracy of objective information from cited articles.

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1. Ransohoff DF, McNaughton Collins M, Fowler FJ Jr. Why is prostate cancer screening so common when the evidence is so uncertain? A system without negative feedback. *Am J Med*. 2002;113:663–667.
2. Holmberg L, Bill-Axelson A, Helgesen F, et al. A randomized trial comparing radical prostatectomy with watchful waiting in early prostate cancer. *N Engl J Med*. 2002; 347:781–789.

The Reply:

We agree that the positive results of the Holmberg trial (1) do not apply to persons with screening-detected prostate cancer, for whom therapy may have a lower effect or even none at all. We also agree that the lack of an overall mortality effect is an important feature of the trial's results. We did not elaborate on the results of therapy in part to emphasize our main point, which is totally independent of whether one believes therapy has any benefit for screening-detected prostate cancer—i.e., multiple forces in the decision-making environment push toward aggressive decisions for both screening and therapy and toward a perception of benefit, independent of whether there is any actual benefit. We agree with the writers and others that there are no data to demonstrate the benefit of prostate cancer screening among asymptomatic persons (2,3).

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1. Holmberg L, Bill-Axelson A, Helgesen F, et al., and the Scandinavian Prostatic Cancer Group Study Number 4. A randomized trial comparing radical prostatectomy with watchful waiting in early prostate cancer. *N Engl J Med*. 2002;347:781–789.
2. Screening for prostate cancer: recommendation and rationale. *Ann Intern Med.* 2002; 137:915–916.
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SUBACUTE MOTOR WEAKNESS AND LEFT RENAL MASS

To the Editor:

A 65-year-old white woman presented with facial palsy and progressive weakness of both arms and legs. She had no previous history of fever, chills, recent infections, allergies, recent immunization, exposure to neurotoxic chemicals, and use of tobacco, illicit drugs, and alcohol. Her family history was unremarkable. Her temperature was 36.6°C, pulse was 105 beats per minute, and respiration rate was 25 breaths per minute. Blood pressure was 110/90 mm Hg. Hematologic laboratory values were normal. The physical examination showed no abnormalities. The patient was alert. Sensitivity to light, touch, and pinprick was normal, as was coordination. Neurologic examination revealed bilateral distal weakness on dorsal flexion of the feet, tingling paresthesias in the hands, and loss of tendon reflexes in both arms and legs. Muscle bulk, tone, and strength were normal. Neurophysiologic findings suggested a polyneuropathy of recent onset. Examination of the right median and tibial nerves showed reduced motor nerve conduction velocities in the upper and lower limbs. F waves and H reflex recorded after stimulation of tibial nerves were absent. Repetitive stimulation studies of the intrinsic hand muscles before and after exercise

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showed no decreased responses or postexercise facilitation. Needle electromyography showed no denervation potentials and a reduced pattern of motor unit discharge at maximum effort, which correlate with the degree of weakness. Analysis of the cerebrospinal fluid showed albuminocytologic dissociation with a marked increase in protein concentration (1.3 g/L) and mild pleocytosis (lymphocyte count, 10/mm^3). In the following days, weakness of the legs worsened and right facial palsy became evident, requiring the patient to be confined to bed. The slow conduction velocities, presence of conduction block, and absence of F wave and H reflex indicated a demyelinating peripheral neuropathy. Biopsy specimens of the sural nerve were uninformative.

The diagnosis of Guillain-Barré syndrome was made, using the criteria of Asbury and Cornblath (1). Treatment with plasma exchange (total exchange, 3.5 L) and intravenous immunoglobulin (0.4 g/kg/d for 5 days) was started. Corticosteroids were not administered. The patient's motor weakness improved. After 6 days, there was complete neurological recovery and she was able to walk. She was discharged after 2 weeks of hospitalization and instructed to take methylprednisolone (500 mg/d) and undergo rehabilitation.

One month later, she presented again with weakness of arms and legs. She was unable to stand on her toes or heels, and deep tendon reflexes were absent. Despite corticosteroid use, her neurological symptoms had worsened and right facial palsy had returned with pain in the left flank. Abdominal ultrasonography revealed a 3.5-cm mass of heterogeneous configuration in the left kidney. Total body computed tomography (CT) confirmed the presence of the mass but ruled out metastatic disease; a bone scan was negative. A left nephroureterectomy was performed. A moderately differentiated clear cell carcinoma limited to the kidney was found, classified as stage I (T1N0M0). After nephrectomy, the patient's condition improved. She remained in good health until about a year later when the neurological symptoms returned. She underwent a total body CT scan, which showed metastatic renal tumor dissemination to the liver, lung, and brain. However, she died before starting any first-line therapy.

Several neoplasias may manifest initially as a paraneoplastic neurological syndrome, although the pathogenesis is not known. Some authors have described a cross-reaction between autoantibodies against intracellular or membrane neoplastic antigens and neuronal nuclear or membrane antigens (2). These neuronal antibodies often cannot be identified. In patients without antibodies, neuropathies that occur about 3 years before cancer diagnosis are probably paraneoplastic; for neuropathies appearing many years before diagnosis, the association is most likely coincidental. Renal cell carcinomas are rarely associated with neurologic paraneoplastic syndromes (3,4), and Guillain-Barré symptoms rarely occur with solid tumors (5,6).

We did not examine cerebrospinal fluid for malignant cells because leptomeningeal carcinomatosis is common in malignant lymphoproliferative diseases and is rarely the presenting feature of a solid tumor. The types of solid tumor most commonly associated with this condition are breast, lung, and gastrointestinal carcinomas and malignant melanoma. There has been one report of cancer of probable renal origin with meningeal carcinomatosis (7). Meningeal carcinomatosis usually presents with multiple cranial neuropathies, patchy radiculopathies, and changes in mental status (8,9), which was not the case with this patient. Moreover, resolution of the neurological manifestations either after plasmapheresis or nephrectomy confirmed the autoimmune origin of the acute polyneuropathy.

Our report suggests an association between renal cell carcinoma and paraneoplastic Guillain-Barré syndrome, and that nephrectomy can lead to the dramatic resolution of neurological symptoms. As observed in this patient, neurological symptoms may precede cancer diagnosis. Hence, prompt recognition of Guillain-Barré syndrome may anticipate the diagnosis of an early renal malignancy or its progression.

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PERICARDIAL AND PLEURAL EFFUSION IN GIANT CELL ARTERITIS

To the Editor:

Giant cell arteritis usually presents as a typical clinical syndrome (temporal arteritis or polymyalgia rheumatica), but may also have unusual localization. We report a 69-year-old woman who presented with malaise and progressive dyspnea with chest pain on inspiration for 1 week. Eighteen months earlier, a biopsy-negative diagnosis of giant cell arteritis had been made based on the classic symptoms of temporal headache, neck and shoulder pain, jaw claudication, and hip stiffness. Treatment with prednisolone resulted in rapid resolution of symptoms. Twelve months later, she had an infection of both hip prostheses, complicated by sepsis due to *Escherichia coli,* and both prostheses were removed. Prednisolone was stopped, and maintenance treatment with oral ciprofloxacin was prescribed.

On presentation, the physical examination revealed a body temperature of 37.4°C, painful neck and shoulder region, and crackles over the left lung. Laboratory investigation showed normocytic anemia (hemoglobin, 9.0 g/dL) and a markedly increased erythrocyte sedimentation rate (>120 mm/h) and C-reactive protein level (27.7 mg/dL). Chest radiography showed bilateral convex heart enlargement (Figure), pleural effusion, and a pulmonary infiltrate. Pulmonary emboli were unlikely after a normal perfusion scan. Under the assumption of atypical pneumonia, oral clarithromycin was added, but was ineffective. Echocardiography showed 30 mm of circular pericardial effusion, and 1300 mL of exudate was aspirated (leukocyte count, $2.4 \times 10^3/\mu L$). Pleural puncture revealed similar findings. Microbiological cultures remained sterile, including negative results for tuberculosis by polymerase chain reaction.

The most likely diagnosis was pleuropericarditis due to relapsed giant cell arteritis. Prednisolone was restarted at 60 mg daily, which resulted in rapid clinical improvement and normalization of all laboratory parameters. Prednisolone was gradually tapered. Echocardiography and chest radiography 7 months later confirmed resolution of both pericardial and pleural effusion.

Although pericardial (1–3) and pleural involvement (4,5) have been reported in patients with giant cell arteritis, simultaneous occurrence is rare (2). This report emphasizes the possibility of unusual presentations of giant cell arteritis after premature corticosteroid withdrawal and under-

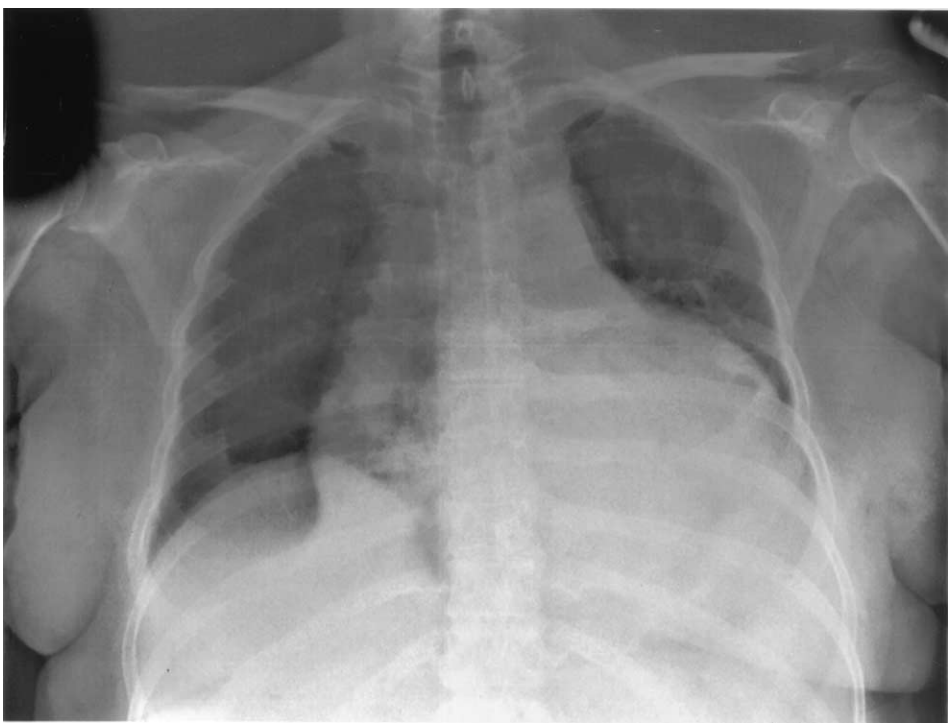

Figure. Chest radiograph showing bilateral convex heart enlargement.