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Basal cell carcinomas following roentgen therapy of ankylosing spondylitis

To the Editor:

Since 1902, ionizing radiation has been known as an important factor in the development of carcinoma in human skin (1). This is a report of multiple basal cell carcinomas in the lumbosacral area in a patient who had completed radiotherapy for ankylosing spondylitis 22 years previously.

In 1950 a 23-year-old man developed back pain that was diagnosed as ankylosing spondylitis. In 1951 his physicians administered radiation therapy totaling 400 scattered roentgens to the sacral, 1,000 to the lumbar, and 200 to the thoracic spinal region. In 1956 he received additional irradiation of 400 roentgens in air to each of five fields: cervical, upper thoracic, lower thoracic, lumbar, and sacroilac. In 1976 a lumbar spinal osteotomy was carried out for the relief of severe kyphosis. His cutaneous wound healed quickly and uneventfully.

In 1978, while the patient was hospitalized for newly discovered diabetes mellitus, a cluster of scaly pinkish papules scattered in the lumbosacral area on both sides and away from the osteotomy scar was noted. Biopsy of five of these lesions showed nests of basal cell carcinoma budding downward to the dermis. The lesions were treated with electrofulguration and curettage. Figure 1 shows the scars left by this procedure. In 1980, three new basal cell carcinomas were discovered within the area enclosed by the previous lesions.

Roentgen therapy for ankylosing spondylitis was introduced in 1930 (2) and was thenceforth applied in large numbers of cases until the late 1950s. The size of the recommended roentgen dosage was never clearly standardized. Smyth, Freyberg, and Lampe originally used 600 rads per total field dose, but later reduced this to 300 rads because temporary leukopenia developed in a few patients (3). Commonly, as in our patient, additional treatment was given as symptoms recurred or developed in new areas. Any portion or all of the spine and the sacroiliac joints were treated, as well as the hips and shoulders in a small number of patients. Irradiation therapy for ankylosing spondylitis fell into disrepute after an association with leukemia was published in 1955 that estimated the risk of leukemia at ten times that of nonirradiated patients or of the general population (4).

Five patients who received radiotherapy for an-

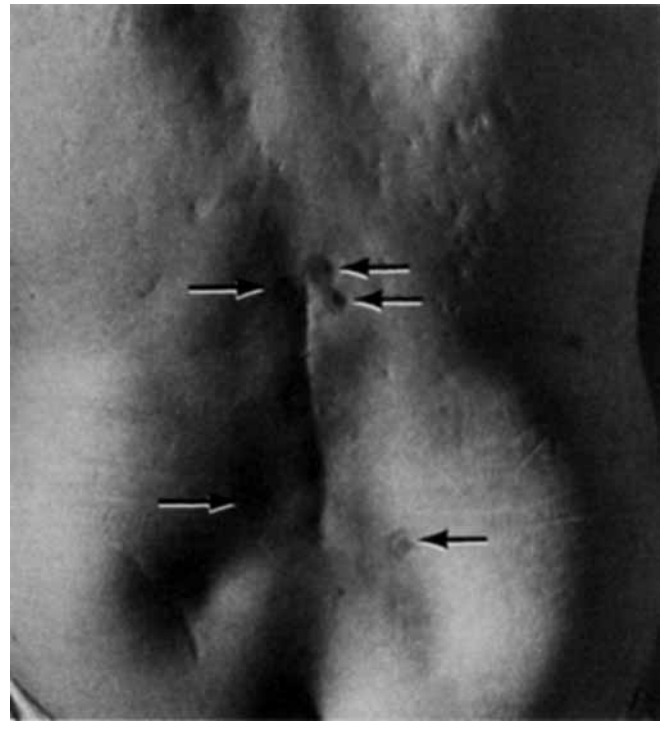

Figure 1. Lumbar osteotomy scar and sites of five basal cell carcinomas **(arrows).**

Table 1. Basal cell carcinomas following irradiation of spine for ankylosing spondylitis

Patients	Length of radiotherapy	No. of treatments	kV	Skin dose, rads	Area treated	Latent period*
Sarkany et al (5)	10 years	158	75	8750	Spine	23
1			140			
		121	75	5425	Front of trunk and	
			100		area lateral to central	
		66	75		spine; remainder	
			140		of trunk	
2	16 years	133	130	7980	Back of trunk	28
3	2 years	36	100	2000	Back of trunk	23
4	6 years	30	220	3335	Spine	20
5	2 months	8	220	1200	Spine	11
Present report	5 years	2	200	400	Cervical spine	27
				600	Thoracic spine	
				1400	Lumbar spine	
				800	Sacrum and sacroiliac	

* Years elapsing between start of radiotherapy and diagnosis of tumors.

kylosing spondylitis developed multiple skin tumors on the back 11–28 years later (5). Four were treated with 2,000 or more rads and widefield irradiation, plus local irradiation of the spine (Table 1). Development of carcinomas in the fifth patient who received 1200 rads and our patient who received a total of 1400 rads over the lumbar spine indicates that the population at risk includes patients treated with dosages employed commonly in North America.

Another report described patients with back pain (1 case), lumbar disc disease (1 case), and ankylosing spondylitis (1 case) who developed basal cell carcinomas as well as fibroepitheliomas of Pinkus (26, 26, and 19 years later, respectively) within exposed areas after unspecified amounts of therapeutic irradiation (6).

Basal cell carcinomas ascribed to radiation are usually encountered on the face, neck, and scalp, with latent periods averaging 20–25 years between irradiation therapy and diagnosis (7). Following treatment of ringworm of the scalp, this latent period may be as short as 7 years (8). However, a study of 26 such cases showed an interval averaging 45 years (9).

The latent period for irradiation-induced cutaneous neoplasm varies inversely with the dose, ranging from 7 weeks to 56 years (10). Squamous cell carcinomas are associated with large exposure and skin ulceration. On the other hand, basal cell carcinomas predominate following smaller doses, with or without history of radiation dermatitis, and often occur in skin that is otherwise clinically normal (10).

A survey for causes of mortality in a series of 14,544 patients with ankylosing spondylitis carried out 5–25 years after radiotherapy did not disclose deaths from skin tumors (11). Apparently, a survey for tumors in irradiated skin was not performed in this patient study as of 1972 (10). Extrapolating from the remarkably delayed onset of cutaneous neoplasia following irradiation of the scalp, it appears that patients treated for ankylosing spondylitis 25–50 years ago may still be susceptible to this complication. The skin at sites of irradiation should be examined carefully whenever these patients are encountered.

ARMIN E. GOOD, MD
LUIS A. DIAZ, MD
RICHARD A. BOWERMAN, MD
Veterans Administration Medical Center and the Departments of Medicine, Radiology and Dermatology
University of Michigan Medical School
Ann Arbor, Michigan 48105

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Late appearing nitritoid reactions

To The Editor:

Podell et al in their article "Pulmonary Toxicity with Gold Therapy" (Arthritis Rheum 23:347–350, 1980) note that nitritoid reactions to the thiomalate preparation are common and occur early in the course of treatment. I should like to report a somewhat different experience. All my patients are begun on the thiomalate preparation, and in only three has a nitritoid response appeared. In one case, it occurred at 1,000 mg. But in 2 instances the reaction was very late, as the following descriptions indicate.

CS, a 75-year-old woman, began gold injections 9 years ago. After 8 years and having received 6,225 mg of gold sodium thiomalate, she had a typical nitritoid reaction. She was switched to the thioglucose preparation and has had no further problem. An electrocardiogram (ECG) revealed no acute changes.

BB, a 76-year-old woman, began treatment 14 years ago. There was an excellent response for several years, followed by a gradual relapse. After 9 years, gold injections were discontinued after a total of 7,600 mg, and various other modalities were tried without success. After a year, gold sodium thiomalate was resumed which resulted in considerable improvement. After 2½ years and a total of 3,300 mg, a typical nitritoid reaction occurred. With the thioglucose preparation, there has been no problem. An ECG revealed no acute changes.

The above reports are intended to alert the physician employing gold salt injections to be aware of very late appearing nitritoid reactions with the gold sodium thiomalate.

SELVAN DAVISON, MD, FACP
Rheumatology Division
Department of Medicine
The Mount Sinai Medical Center
New York, NY 10029

Secondary amyloidosis and sicca syndrome

To The Editor:

Simon and Moustopoulos recently reported a case of sicca syndrome in a patient with primary amyloidosis and monoclonal gammopathy due to amyloid invasion of the lacrimal and salivary glands (Arthritis Rheum 22:932–934, 1979). We report here a patient with sicca syndrome due to secondary amyloidosis.

The patient is a 57-year-old white woman with severe, long-standing, seronegative, erosive rheumatoid disease. She recently noted the onset of severe xerostomia and swollen cheeks. Examination revealed bilateral non-tender swelling of the parotid glands, a large tongue, and a normal response to the Schirmer tear test. Lip biopsy demonstrated diffuse deposition of amyloid around atrophied salivary acini, normal salivary ducts, and no inflammatory infiltrate. Serum protein electrophoresis revealed no monoclonal spike, and urine was negative for Bence Jones protein.

This patient fulfills the necessary two of three criteria for sicca syndrome—parotid swelling and xerostomia. There were no signs of keratoconjunctivitis. Her amyloidosis is most likely secondary to severe rheumatoid disease of long duration. She thus represents a case of sicca syndrome due to secondary amyloidosis.

M. A. CATALANO, MD
J. H. VAUGHAN, MD
Scripps Clinic and Research Foundation
10666 N. Torrey Pines Road
La Jolla, CA 92037

Buerger's disease and multicentric fibromuscular hyperplasia mimicking Takayasu's arteritis

To the Editor:

Thromboangiitis obliterans (Buerger's disease) is an unusual vascular disorder of unknown etiology that has been recognized infrequently in females. We have recently treated a woman with severe Buerger's disease in whom roentgenographic studies demonstrated multiple obstructive lesions of the aorta and its branches. Surgical exploration of a carotid lesion was consistent with fibromuscular hyperplasia. The occurrence of coexisting Buerger's disease and multicentric fibromuscular hyperplasia is unique and initially suggested a diffuse form of vasculitis.

A 33-year-old white woman was admitted to Madigan Army Medical Center because of ischemic necrosis of her right foot and hypertension. An arteri-