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## COMMENTS AND OPINIONS

### Speckled Lentiginous Nevus: No Longer One Single Disorder

I read with interest the arguments presented by Barysch and Dummer<sup>1</sup> on how to distinguish speckled lentiginous nevi from segmental lentiginosis. I agree that such criteria are important because these disorders are sometimes confused. However, I would like to make the following annotations.

Speckled lentiginous nevi can no longer be considered a single disorder. The authors are confusing 2 distinct entities: the macular type (nevus spilus maculosus [NSM]), which is completely flat and shows a fairly even distribution of speckles; and the papular type (nevus spilus papulosus [NSP]), which is characterized by papular speckles showing a far more uneven distribution reminiscent of a star map.<sup>2</sup> In their text, Barysch and Dummer<sup>1</sup> report a typical case of NSM, but in the Table they describe the distinguishing features of NSP and mention a possible association with "spinal muscle atrophy and other neurological disorders in the context of speckled lentiginous nevus syndrome."<sup>1</sup>(p954) It is important to realize that this syndrome, which may also occur as a component of phacomatosis pigmentokeratocytica,<sup>3</sup> is always associated with NSP.<sup>2,4</sup> Hence, it has nothing to do with the NSM as reported by Barysch and Dummer. On the other hand, being unaware of the dichotomy, the authors do not mention that NSM can be associated with nevus roseus, resulting in phacomatosis spilorosea.<sup>5</sup>

In my view, the statement that speckled lentiginous nevi follow the lines of Blaschko<sup>1</sup> is incorrect. To my knowledge, no such case has been reported. All large nevi spili are arranged in a flaglike, blocklike, or checkerboard pattern.<sup>6</sup> The case presented by Barysch and Dummer<sup>1</sup> is no exception to this rule.

In conclusion, I fully agree with Barysch and Dummer<sup>1</sup> that, for a correct diagnosis of speckled lentiginous nevi and segmental lentiginosis, a thorough medical history, proper record of the distribution, and careful examination of the surrounding skin are essential.<sup>1,7</sup>

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## VIGNETTES

### Monoclonal Gammopathy of Undetermined Significance and Incidentally Identified Cutaneous Monotypic Plasma Cell Infiltrates in the Absence of Specific Skin Lesions

The present case highlights the nosologic complexity of monoclonal gammopathy (MG) associated with monotypic plasma cellular infiltrates in the absence of specific clinical skin lesions or systemic involvement.

**Report of a Case.** An 85-year-old woman presented for basal cell carcinoma (BCC) reexcision from her right leg. She had a history of MG since 1996, with 6% plasma cells in her bone marrow at that time. No skin lesions clinically suggestive of plasmacytoma or cutaneous involvement by multiple myeloma (MM) or evidence of end-organ failure (ie, anemia, hypercalcemia, or renal insufficiency) were seen at any time.

In addition to BCC, the excision specimen revealed a dermal and subcutaneous perivascular and interstitial predominantly mature plasma cell infiltrate (**Figure**). Rare plasma cells showed slightly enlarged nuclei with pleomorphism. No amyloid deposition was seen. Immunohistochemical studies revealed a kappa-lambda ratio greater than 10:1. A monoclonal IgH receptor gene rearrangement was detected in the skin by polymerase chain reaction. Serum protein electrophoresis results confirmed an IgG kappa M protein level of 0.79 g/dL. Complete blood cell count was within normal limits, and a skeletal survey did not identify lytic bone lesions.

**Comment.** Cutaneous monotypic plasma cell infiltrates associated with MG include MM, primary cutaneous plasmacytoma (PCP), cutaneous lymphoplasmacytic lymphoma/Waldenström macroglobulinemia (LPL/WM), and marginal zone lymphoma (MZL).<sup>1</sup> Monoclonal gammopathy in LPL/WM is typically of IgM phenotype, but both MZL and PCP can show MG of any phenotype (IgG, IgM, or IgA) in 27% and 25% of cases, respectively.<sup>2-4</sup> A rare tumor, PCP typically presents as single or multiple clinically evident skin nodules or plaques with no site predilection and a variable clinical course.<sup>4,5</sup> Risk of pro-

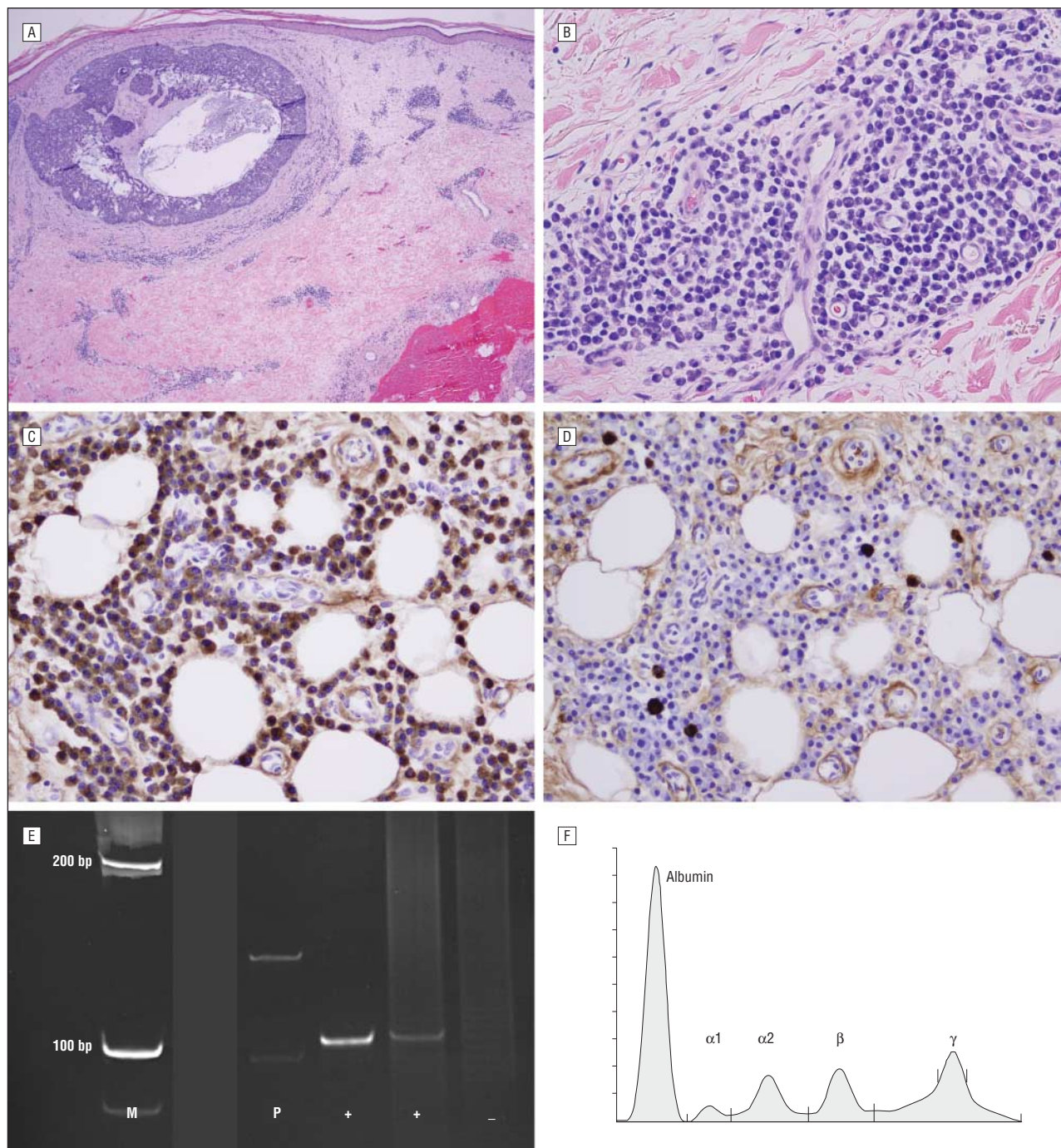


**Figure.** Histopathologic, immunohistochemical, and polymerase chain reaction (PCR) findings and electrophoresis densitometric tracing. A and B, Basal cell carcinoma reexcision specimens showing a dermal and subcutaneous perivascular and interstitial predominantly mature plasma cell infiltrate (hematoxylin-eosin, original magnifications  $\times 40$  and  $\times 200$ , respectively). By immunohistochemical analysis, a kappa (C) to lambda (D) ratio higher than 10:1 is indicative of light chain restriction (original magnification  $\times 200$ ). E, Monoclonal IgH receptor gene rearrangement is detected in the skin by PCR (bp indicates base pair; M, marker; P, patient; -, negative control; and +, positive control). F, Serum protein electrophoresis with densitometric tracing showing the presence of an M protein with a characteristic spike in the  $\gamma$ -globulin region.

gression to frank MM has been associated with the presence of multiple lesions, cytologic atypia, and IgA phenotype.<sup>4,5</sup> Primary cutaneous plasmacytoma can show a nodular or diffuse nonepidermotropic infiltrate of plasma cells with variable maturation and cellular atypia.<sup>4,5</sup> In some cases, cytologic atypia may be absent.<sup>5</sup> The existence of PCP has been recently disputed, some researchers suggesting that cases represent MZL with prominent plasmacytic differentiation.<sup>1</sup>

The detection in the peripheral blood of a low level of M protein (monoclonal immunoglobulin) that is below the defined diagnostic threshold for MM is known as MG of undetermined significance (MGUS), an asymptomatic disorder defined by (1) the presence of a serum M protein at levels lower than 3 g/dL; (2) bone marrow monoclonal plasma cell concentration of lower than 10%; (3) lack of end-organ disease; and (4) absence of a B-cell lymphoproliferative disorder associated with M protein produc-

tion.<sup>2</sup> The clinical course for most patients with MGUS is generally stable with about a 1% incidence of progression to MM per year. Risk factors for progression include the size of the M protein and a non-IgG phenotype.<sup>2</sup>

Our patient had not received any specific therapies for MG and remained well for 13 years without evidence of progression. The indolent course of her disease may be related to an absence of specific or multiple skin lesions, lack of significant cellular atypia, low M protein level, and its IgG phenotype.

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### Rare Cutaneous Manifestation of Prostate Cancer

**P**rostate cancer is the most frequent internal malignant neoplasm in men, but it represents only a minority of cutaneous metastases. In a meta-analysis of cutaneous metastasis, Krathen et al<sup>1</sup> report that the over-

all incidence of cutaneous metastases from visceral cancers appears to be around 5%. The highest incidence of cutaneous metastasis was seen in breast cancer, occurring with an incidence of 24%. In the same meta-analysis, prostate cancer was the least likely visceral malignant neoplasm to metastasize to the skin (0.7% incidence).<sup>1</sup> Herein, we describe a patient with multiple cutaneous nodules as the presenting sign of recurrent prostate cancer.

**Report of a Case.** A 67-year-old man with an 8-year history of prostate cancer presented 2 months after the simultaneous development of multiple asymptomatic nodules and he reported a 1-week history of lower back pain. His prostate cancer was Gleason score 9 with metastases to the spine at diagnosis. He received hormonal ablation and irradiation but was asymptomatic and had discontinued therapy for over 3 years.

On physical examination, multiple violaceous and telangiectatic firm nodules ranging in size from 8 mm to 2.3 cm were noted on the forehead, scalp, neck, flank, and right upper arm (**Figure 1**). Biopsy specimens from the forehead, flank, and neck demonstrated solid sheets of epithelial cells in the dermis with nuclear atypia, mitotic figures, necrosis, and foci of ductal differentiation. Forehead and flank biopsy specimens demonstrated focal immunoreactivity with prostate-specific antigen (PSA) (**Figure 2**) and prostatic acid phosphatase (PAP), whereas the neck biopsy specimen was negative for both. All 3 specimens demonstrated scattered cytokeratin 7 (CK 7) and 20 (CK 20) immunopositivity with focal positivity for carcinoembryonic antigen (CEA). No immunoreactivity was demonstrated for CD10, S-100, RCC, or vimentin.

The patient was referred to the oncology department, and a workup demonstrated multiple lung nodules, mediastinal adenopathy, innumerable skeletal metastases, and a PSA level of 17.4 ng/mL (normal range, 0.0-4.0 ng/mL [to convert to micrograms per liter, multiply by 1.0]). Despite treatment with bicalutamide and goserelin, the patient died 12 months following the onset of his skin lesions.

**Comment.** Prostate cancer typically metastasizes to lymph nodes, bones, and lungs, with less than 3% occurrence



**Figure 1.** The patient presented with the simultaneous appearance of multiple telangiectatic and firm nodules. A, Firm, telangiectatic nodule on the forehead. B, Pedunculated, telangiectatic nodule of the anterior neck.