out that invasion and metastasis in typical Bowen's disease is not common, and the absence of a fully documented case with metastatic disease cannot be used to eliminate the possibility that malignant transformation may rarely occur.

Another possible factor in the development of these lesions could be human papilloma virus. Recently, human papilloma virus has been found in some seborrheic keratoses with histologic changes consistent with a viral effect.[8] Human papilloma virus has been associated with various tumors and is felt to be a cofactor in the development of at least some malignancies.[9]

The most striking feature of the lesion we report is that it is a mucin-positive adenocarcinoma arising in the center of a lesion clinically and histologically seborrheic keratosis. In this case, there are three possible explanations for the occurrence of adenocarcinoma in seborrheic keratosis. These include metastasis from a distant primary tumor to the seborrheic keratosis, incidental origin of an adnexal carcinoma at the site of the seborrheic keratosis, and origin of an adenocarcinoma from the seborrheic keratosis. Since no other primary tumor was found, and since there was no evidence of any other cutaneous or malignant tumor after 10 years, the adenocarcinoma was probably not a metastasis. While incidental origin at the site of a seborrheic keratosis cannot be absolutely disproven, an epidermal origin of seborrheic keratosis is supported by direct dermal invasion of the tumor from the epidermis rather than from an adnexal structure, the central location of the adenocarcinoma, and the clinical history. As with some cases of extramammary Paget's disease, in which mucin-producing epithelial cells are present within the epidermis with no primary tumor and with no evidence of adnexal involvement, only theories exist to explain this phenomenon. These theories could include malignant transformation of a pluripotential germinative cell within the epidermis or transformation of a cell from the distal portion of an adnexal structure located within the epidermis. Cells from these structures would be at least partially committed toward adnexal differentiation.

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Widespread Cutaneous Necrosis in a Patient With Rheumatoid Arthritis Associated With Anticardiolipin Antibodies

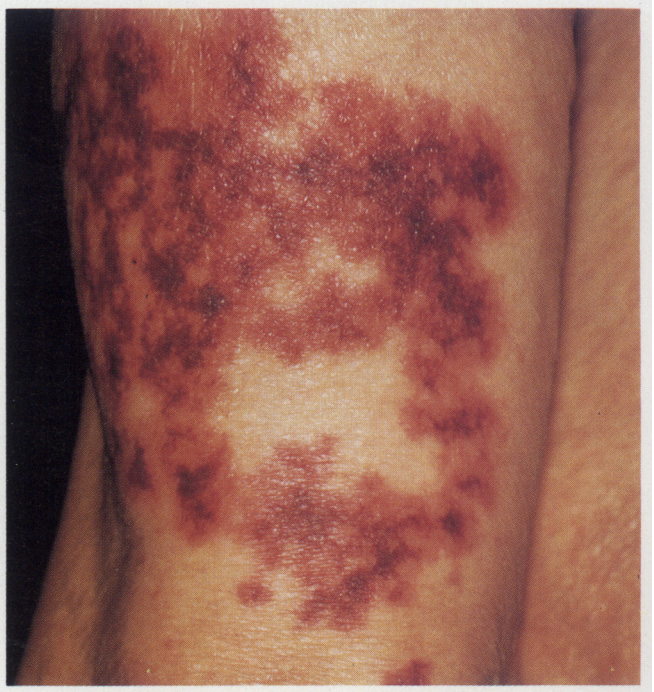
To the Editor.—Antiphospholipid antibodies, detected by the presence of lupus anticoagulant and/or abnormally high levels of anticardiolipin antibodies, have been shown to be associated with an increased risk of arterial or venous thrombosis. The association between antiphospholipid antibodies and thrombotic events was first noted in patients with systemic lupus erythematosus but were later also observed in a variety of other disorders and as an isolated finding. The existence of a separate entity, the anticardiolipin or antiphospholipid syndrome, was suggested in several studies.[1] Cutaneous symptoms linked to the antiphospholipid syndrome include thrombophlebitis, leg ulcers, livedo reticularis, livedo vasculitis, unfading acral microlivedo, peripheral gangrene, hemorrhage (ecchymosis and hematoma), and necrotizing purpura.[1,2] We observed widespread cutaneous necrosis[3] as a rare manifestation of the antiphospholipid syndrome in a patient with rheumatoid arthritis.

Report of a Case.—A 73-year-old woman with long-standing deforming arthritis, fulfilling the criteria of the American College of Rheumatism, Atlanta, Ga (formerly the Arthritis and Rheumatism Association), for "classic" rheumatoid arthritis, was admitted to our department in July 1988 because of painful widespread skin lesions of 4 days' duration. On clinical examination, several sharply demarcated, hemorrhagic patches with a bizarre configuration were observed on her arms (Fig 1), breasts, and legs.

A skin biopsy specimen of a hemorrhagic area on the left thigh (Fig 2) revealed thrombi within capillaries, venules, and small- and medium-sized vessels throughout the dermis and subcutaneous fat without any evidence of fibrin in the vessel walls. No nuclear "dust" was present. In addition, a sparse, perivascular mixed-cell infiltrate and extravasation of erythrocytes was observed.

Results or findings from laboratory studies were as follows: erythrocyte sedimentation rate, 108 mm/h; erythrocytes, 3.97×10^{12}/L; hemoglobin, 87 g/L; leukocytes, 6.9×10^{9}/L, with 0.69 neutrophils, 0.1 band forms, 0.1 eosinophils, and 0.29 lymphocytes; platelets, 336×10^{9}/L; serum electrolyte, serum urea nitrogen level, serum glucose level, and urinalysis, normal; liver function test results,

Fig 1.—Widespread cutaneous necrosis. Sharply demarcated, hemorrhagic patches with bizarre configuration on the left arm.



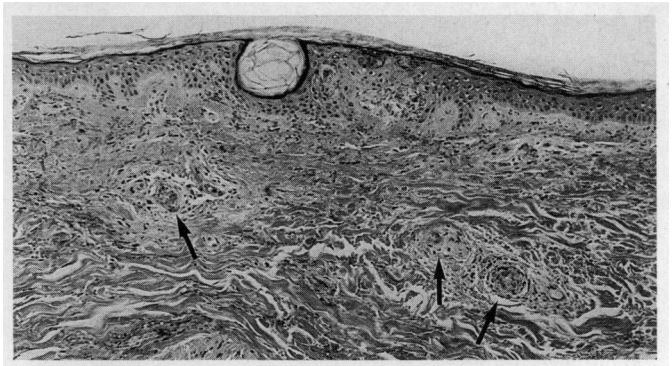


Fig 2.—Widespread cutaneous necrosis. Thrombi within dermal vessels (arrows). Extravasated erythrocytes in the papillary dermis. No signs of vasculitis.

Reports of Widespread Cutaneous Necrosis Associated With Antiphospholipid Antibodies *

Source, y	No. of Cases	Underlying Disease	ACA	LAC	VDRL	ds-DNA
Dodd et al,[3] 1985	1	No	ND	+	ND	−
Frances et al,[4] 1989	3†	SLE	−	+	+	+
O'Neill et al,[5] 1990	1	No	+	−	−	−
Present case	1	RA	+	−	−	−

*ACA indicates anticardiolipin antibodies; LAC, lupus anticoagulant; ds-DNA double-stranded DNA antibodies; RA, rheumatoid arthritis; ND, not done; plus sign, condition present; and minus sign, condition absent.
†All three reported cases revealed the same serologic profile.

normal, except for the alkaline phosphatase level (216 U/L) and the cholinesterase level (3395 U/L); direct Coomb's test, VDRL, and the test for cryoglobulins, negative; immunoelectrophoresis, increase of polyclonal immunoglobulins; latex rheumatoid factor, 3390 U/mL; and latex C-reactive protein, 32 mg/L (both positive); antinuclear antibodies, 1:1280 (speckled type, positive titer); antibodies against double-stranded DNA, Sjögren syndrome associated antigens A and B, SM–SCL-70, and nuclear ribonucleoprotein, not detectable; partial thromboplastin time, Russell's viper venom test, and prothrombin time, normal; lupus anticoagulant, none detected; tests for clotting factors, fibrinogen, 4.8 g/L; factor II, 0.82; factor V, 0.74; factor X, 0.74; factor III, 0.66; protein C, 0.75; enzyme-linked immunosorbent assay for anticardiolipin antibodies, IgG, 27.7 U/mL (normal, <12.0 U/mL); and IgM, 3.5 U/mL (normal, <6 U/mL) (Elias, Freiburg, Germany).

Initial therapy was prednisolone (50 mg/d). Within 7 weeks, skin lesions healed with moderate scarring. Six months later, anticardiolipin antibodies were still present but the level was clearly reduced (IgG, 15.7 U/mL).

Comment.—Widespread cutaneous necrosis, which is characterized by painful purpuric and necrotic areas with underlying dermal thrombosis, was first reported by Dodd et al[3] in 1985 in a patient with lupus anticoagulant. Subsequently, further cases with identical clinical and histologic features, but different serological profile of antiphospholipid antibodies, were described in systemic lupus erythematosus and in patients with no demonstrable underlying disease (Table).[4,5]

Cutaneous phenomena, including purpura resulting from disseminated intravascular coagulation, coumarin necrosis, coumarin-induced skin lesions in protein C deficiency, heparin necrosis, and purpura cryoglobulinemia, may present as widespread cutaneous necrosis, and all of these conditions should be considered in the clinical differential diagnosis. The significance of anticardiolipin antibodies in the development of skin lesions in our patient is suspected because of the lack of other possible causes and because the titer of these antibodies decreased after therapy. The histologic findings of thrombosis in dermal and subcutaneous vessels without any evidence of leukocytoclastic vasculitis is also characteristic for skin lesions of the antiphospholipid syndrome.[1,2]

Antiphospholipid antibodies in rheumatoid arthritis have been investigated in a number of studies, but estimates of its prevalence are conflicting and the clinical implications of these antibodies in rheumatoid arthritis remain controversial at present.[6,7] Our case, however, shows that anticardiolipin antibodies may be involved at least occasionally in the pathogenesis of skin lesions in rheumatoid arthritis.

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Friction Dermatitis of the Thumbs Caused by Pantyhose

To the Editor.—Dermatitis of the volar aspect of the thumb pads can be a difficult problem in clinical diagnosis. The thick skin here is rather resistant to many contactants, but friction may produce irritation.

Report of a Case.—A 57-year-old woman was seen because of persistent fissured dermatitis limited to the volar aspect of the thumb pads. When asked if some activities had become awkward or difficult because of the dermatitis, she answered that none had, but then added, "Except for putting on my pantyhose."

Millions of women carry out this daily maneuver that puts quite a lot of friction on the volar aspect of the thumb pads. Consider the following: the garment is gathered up, one leg at a time, thumbs on the inside. The toes are put in and the user, maintaining lateral pressure with the thumbs, pulls the garment up into place. It is during this latter action that the thumb pads run quickly up a long length of nylon. Repeated daily, this may produce enough of a frictional stimulus to cause skin irritation.

"It was the pantyhose that caused the problem," the patient wrote in a follow-up letter. "It has only come back (the dermatitis) when I use a certain type hose and/or don't protect the area from the abrasive pressure caused by pulling on the hose."

Comment.—Here is yet another example of the value of careful history taking when faced with a puzzling clinical problem.

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