Brief communication

Anti-DNA antibodies in discoid lupus erythematosus

Follow-up study

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SUMMARY Anti-DNA antibodies have been detected previously in patients with chronic discoid lupus erythematosus (DLE) despite the absence of overt systemic manifestations. 27 patients with DLE were followed up 3 years after the detection of anti-DNA antibodies in 7. None had developed other features of systemic lupus erythematosus. We conclude that the occasional finding of anti-DNA antibodies in patients with DLE does not predict those likely to develop systemic disease.

The detection of serum anti-DNA antibodies is an important adjunct to the investigation and management of patients with systemic lupus erythematosus (SLE) (Hughes, 1971). In the absence of systemic disease, patients with chronic discoid lupus erythematosus (DLE) usually do not have anti-DNA antibodies (Prystowsky *et al.*, 1976). However, some studies using sensitive techniques have reported the presence of anti-DNA antibodies in a proportion of patients with DLE (Mandel *et al.*, 1972; Sylvester *et al.*, 1973), but the clinical significance of this finding has not been established. A previous study from this unit included 42 patients with DLE of whom 15 also had systemic disease (Davis *et al.*, 1974). All patients with SLE had raised levels of anti-DNA antibody. Sera from 7 of 27 patients with DLE and no systemic features contained anti-DNA antibodies. As about 5% of patients with SLE present with discoid lesions, it was suggested that the finding of anti-DNA antibodies in patients with DLE might be a means of predicting those likely to develop the more severe systemic manifestations. Therefore, a follow-up study of those same DLE patients in whom anti-DNA antibodies had been detected was undertaken in order to investigate their subsequent clinical course.

Patients and methods

Twenty-seven patients with DLE alone were included in the original study, and their clinical

features have been described previously (Davis *et al.*, 1974). Information on the clinical course during the subsequent 3 years was available in 24; 3 patients could not be traced. 6 patients originally had anti-DNA antibodies, while 18 did not. The majority were seen and examined by one of us (B.B.), and serum was obtained. In 7 cases clinical information and serum samples were provided by other attending physicians.

As in the original study, anti-DNA antibodies were assayed by ammonium sulphate precipitation (Wold *et al.*, 1968). ^{14}C -DNA was supplied by the Radiochemical Centre, Amersham. Results were expressed as percentage ^{14}C -DNA bound by the serum. The amount of DNA binding by normal sera in our laboratory is 0-30%.

Results

The Table gives clinical information on the 6 patients shown previously to have anti-DNA antibodies. 2 had died 2 years after the original study, of causes unrelated to lupus erythematosus. In one, a 46-year-old woman with a 15-year history of DLE, death was due to a squamous cell carcinoma of the bronchus. There was no clinical evidence of SLE before death and serum antinuclear antibodies (ANA) were not present. A second woman, aged 67 years with a 22-year history of DLE, died of congestive cardiac failure secondary to ischaemic heart disease. ANA, though previously present, were not detectable 2 months before death. 2 patients continued to have active discoid lesions

Table Clinical progress of DLE patients previously having anti-DNA antibodies

Case no.	Anti-DNA*		Clinical progress
	Previous	Current	
1	41	—	Died carcinoma in a bronchus ANA negative before death
2	76	—	Died cardiac failure ANA negative before death
3	38	18	DLE alone; active lesions
4	45	11	DLE alone; inactive lesions
5	35	29	DLE alone; active lesions
6	50	25	DLE alone; inactive lesions

*Results expressed as percent DNA binding (normal 0–30%). DLE = discoid lupus erythematosus.

without overt evidence of systemic disease, and in 2 patients the cutaneous lesions were inactive. In all of these patients DNA-binding had returned to normal values.

Sera from the other 18 patients had not previously contained anti-DNA antibodies. During the period of follow-up none had developed other disease manifestations, and none of this group had developed serum anti-DNA antibodies.

Discussion

It has been suggested that DLE is part of a larger systemic disease spectrum (Tuffanelli *et al.*, 1969; Dubois, 1974; Prystowsky and Gilliam, 1975). While most patients with discoid lesions have disease that remains confined to the skin, about 14% of patients with SLE will manifest discoid lesions during the course of their diseases (Estes and Christian, 1971). Some patients with DLE develop antinuclear antibodies (Rothfield *et al.*, 1963; Shrank and Doniach, 1963; Beck and Rowell, 1966; Burnham and Bank, 1974), but this feature appears to bear no relationship to the course of the disease. The observation that some patients with DLE were found to have anti-DNA antibodies suggested that this might be a means of predicting those likely to develop extracutaneous manifestations. Our study was undertaken to investigate that possibility. The results show that none of the patients previously found by Davis *et al.* (1974) to have anti-DNA antibodies had developed any overt systemic complication after a 3-year interval. Furthermore, while the assay used was identical, anti-DNA antibodies were no longer detectable in their sera. This suggests that anti-DNA antibodies may be transiently present in the sera of some patients with DLE.

The finding of anti-DNA antibodies in conditions other than SLE may depend on a number of variables, including the sensitivity of the method used, and the purity of the double-stranded DNA used. Thus

the occasional appearance of circulating anti-DNA antibodies in DLE is of uncertain pathogenic significance. Certainly their presence does not appear to identify those patients who subsequently develop SLE.

We are grateful for the co-operation of Drs. I. B. Sneddon, R. E. Church, S. C. Gold, M. A. Smith, and Margaret F. Corbett. G. Hale provided expert technical assistance.

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