SLE syndrome, probably induced by labetalol

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Summary

A 45-year-old woman developed polyarthralgia and muscle tenderness after 6 months' therapy with labetalol 1800 mg daily. A raised ANF titre but normal DNA binding levels suggested a drug-induced SLE syndrome. Both symptoms and ANF titre improved when therapy was changed from labetalol to propranolol.

Case report

A woman patient was first treated for hypertension in 1970 when aged 45 years. Her hypertension proved difficult to control and she was treated with combinations of adrenergic neurone-blocking drugs (guanethidine or bethanidine), β -blockers (propranolol, oxprenolol or atenolol) and thiazide diuretics (bendrofluazide, mefruside or cyclopenthiiazide). From February 1978 she was treated with atenolol 200 mg daily and prazosin 4 mg daily, which were discontinued in May 1978 when she developed an urticarial rash. After one month without anti-hypertensive treatment she was admitted to hospital, and labetalol therapy (300 mg daily) started. The dose was steadily increased to 1800 mg daily by July 1978, with satisfactory control of blood pressure.

In January 1979 she developed increasingly severe stiffness of the shoulders and upper arms, together with stiffness and swelling of the hands. There was some pain in both knees and elbows, and in the right wrist, but no swelling of these joints. There was no rash, or other symptoms. In May 1979 she was first seen in the clinic with these complaints. The only findings were marked muscle tenderness and stiffness around the shoulders, with crepitus in the left shoulder and wrist and swelling of the left knee. Investigations showed a raised anti-nuclear factor

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(ANF) titre with normal DNA binding, and it was decided in June 1979 to discontinue labetalol therapy. Over a one-month period propranolol was gradually re-introduced and labetalol therapy withdrawn. Blood pressure is now (1980) well controlled by propranolol 1440 mg daily with phenoxybenzamine 20 mg daily and Navidrex-K (Ciba). Muscle stiffness and arthralgia required initial therapy with indomethacin but steadily improved so that by September 1979 there was residual minor stiffness in the left shoulder only, and by January 1980 she was asymptomatic.

Laboratory investigations: Hb, WBC and platelet count normal throughout. ESR maximum 26 mm/hr (July 1979). Serum immunoglobulins and complement C₃ and C₄ components normal (May 1979). DAT screening test negative. Urine - no proteinuria and no red cells. ANF titre and DNA binding (Farr test) - see Table below:

	ANF titre (reciprocal)		DNA binding (normal <25%)
	Homo- geneous	Nucleolar	
May 1979	100	400	-
June 1979	10	1600	17
July 1979	10	1600	18
September 1979	10	1600	19
October 1979	10	800	18
March 1980	50	100	-

Discussion

The polyarthralgia and myalgia, together with the markedly raised ANF titre in the presence of normal DNA binding values are compatible with a drug-induced lupus syndrome. Although the patient had been treated with several drugs, her symptoms and raised ANF titre developed after 6 months' treatment with labetalol alone. After withdrawal of labetalol her symptoms cleared and her ANF titre

has fallen significantly. The authors therefore regard the association between labetalol and the lupus syndrome in this case as probable. This appears to be only the second such case reported (Griffiths and Richardson, 1979), although there are other reports of increases in ANF titre during labetalol therapy in patients who remained asymptomatic (Wilson, 1980). Cases of lupus syndrome are reported to have developed during practolol (Raftery and Denman, 1973) and pindolol therapy (Bensaid, Aldigier and Gualde, 1979), and one possible case developed during propranolol therapy (Harrison, Sisca and Wood, 1976) although in the present patient, as in that of Griffiths and Richardson (1979), the syn-

drome improved after the patient was switched from labetalol to propranolol.

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