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RAPID COMMUNICATION

Cyclosporin for a severe case of aspirin-induced asthma

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Cyclosporin, an immunosupressive drug, predominantly inhibits T-lymphocyte dependent immune responses by interfering with synthesis of lymphokines. T-lymphocytes are an important source of inflammatory mediators, and some aspects of asthma may be accounted for by alterations in release of cytokines [1].

Cyclosporin was used to treat a 42 yr old woman with asthma. She has been under our treatment since 1975 when diagnosis of aspirin intolerance was established [2, 3] by oral provocation tests. Skin tests to common airborne allergens were negative, serum immunoglobulin E (IgE) was 101 IU·ml^{-1}. Her asthma was of severe type and over the years 1976–1989 she was on chronic corticotherapy at a dose of 20–30 mg·day^{-1}.

Table 1. – Mean peak expiratory flow (PEF) values (l·min^{-1}) calculated from 28 daily readings obtained during 4 wks before starting cyclosporin and 42 daily readings during the eighth month of cyclosporin therapy

Method of recording PEF	Before cyclosporin		During eighth month of cyclosporin therapy		
	Mean	SD	Mean	SD	p
Morning					
before bronchodilator	205	31.5	231	28.1	<0.001
after bronchodilator	235	31.3	254	23.8	<0.005
Evening					
before bronchodilator	249	23.9	267	17.3	<0.001
after bronchodilator	273	28.7	289	14.2	<0.005

Differences between means computed using two-tailed t-test. SD: standard deviation; p: probability.

The patient entered a 5 month baseline period during which she recorded peak expiratory flows (PEF) twice a day. Her medication was standardized at inhaled beclomethasone 1,600 µg·day^{-1} aminophyline 1,050 mg·day^{-1}, fenoterol 1,600 µg·day^{-1} and oral prednisone 25 mg·day^{-1}. An attempt to gradually reduce prednisone to 15 mg·day^{-1}, keeping doses of other drugs constant, was unsuccessful. Cyclosporin was then started, and 5 wks after its introduction a careful atempt to reduce prednisone by 1–2 mg·wk^{-1} was begun. Following 11 months' treatment with low-dose cyclosporin (whole-blood trough levels 70–150 mg·ml^{-1}) the patients's asthma improved and became more stable, spirometric values rose significantly (table 1), whilst the daily dose of prednisone was reduced from 25 mg to 8.5 mg. No side-effects were noticed.

In this patient, followed by us over the last 15 yrs, use of cyclosporin was associated with clear improvement of asthma. Our observations [4] indicate that cyclosporin might be of therapeutic interest in other types of steroid-dependent asthma. The preliminary nature of the present report has to be stressed. We have not observed any side-effects of cyclosporin treatment, possibly because the drug was given at a low-dose. Cyclosporin side-effects are, however, of concern and should be kept in mind in patients with bronchial asthma possibly subjected to cyclosporin treatment in the future.

References

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Emploi de la cyclosporine dans un cas grave d'asthme induit par l'Aspirine. E. Nizankowska, R. Dworski, A. Szczeklik.
RÉSUMÉ: La cyclosporine interfère sur la synthèse des lymphokines. Nous avons utilisé la cyclosporine pour traiter une femme de 42 ans, dont l'asthme grave était dû à une intolérance à l'Aspirine. La médication a comporté l'inhalation de beclomethasone à raison de 1,600 µg·jour^{-1} et la prise orale de prednisone à raison de 25 mg·jour^{-1}. Après la mise en oeuvre d'une médication comportant de petites doses de cyclosporine (creux dans le sang complet entre 70 et 150 mg·ml^{-1}), l'asthme s'est amélioré progressivement, et la dose de prednisone orale a été ramenée à 8.5 mg·jour^{-1}.
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