Communications

Successful Treatment of Chronic Urticaria With Mefenamic Acid

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Chronic urticaria, defined here as lesions that have recurred at least once in four weeks for a period of six months or longer,[1] remains an etiologic and therapeutic enigma. Described here is the case of a 32-year-old woman in whom daily severe chronic urticaria of three years' duration was totally ameliorated after treatment with mefenamic acid.

Case Report

A 32-year-old white woman presented with a chief complaint of a three-year history of severe urticaria occurring on a daily basis. She noted the onset of the chronic urticaria following a dilation and curettage procedure for dysfunctional uterine bleeding. Questioning failed to elicit a history of either patient or family evidence of allergy. She twice had a complete workup for chronic urticaria at a university hospital without discovery of an etiological agent.

Regimens including combinations of antihistamines, benzodiazepines, and corticosteroids were used without obvious improvement. Although the lesions occurred on a daily basis, usually becoming worse later in the day, the patient had documented a significant increase in severity at midcycle and during her menstrual period.

Physical examination of the patient was unremarkable except for typical urticarial lesions on the volar aspects of the forearms, face, and legs, with occasional lesions on the trunk. The patient reported the urticaria was at times so severe that she could not use her hands, and facial involvement was to such an extent that she would not appear in public. A pressure-induced component was demonstrated during a cardiopulmonary re-

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suscitation course when practice with the mannikin caused hand and lip involvement.

Because the initiating factor for this patient's chronic urticaria appeared to be a gynecologic procedure and severity seemed to be linked to her menstrual cycle, the possibility of a prostaglandin involvement was considered. Although several prostaglandin inhibitors are now proven effective, mefenamic acid was utilized because of previous clinical experience with the medication in treatment of dysmenorrhea.

The initial dosage was set arbitrarily at 250 mg twice daily. The patient was asked to keep a basal body temperature chart and to increase dosage at onset of ovulation to 250 mg four times daily for four days. During the remaining days of the cycle, the dosage was decreased to 250 mg twice daily until the onset of menstruation, at which time dosage was again increased to four times daily. Response was almost immediate. Within the first week the patient noted a substantial decrease in symptoms and was virtually lesion-free. Midcycle and menstrual flares were almost totally eliminated the first month.

Because there was some concern about long-term utilization of mefenamic acid, the daily dosage was decreased to 250 mg daily during the second month except during midcycle and menstruation, when a four-times-daily schedule was used. Again, symptoms were almost completely controlled. During the third month, mefenamic acid was used 250 mg four times daily during midcycle and menstruation and 250 mg daily as needed at other times without loss of control of symptoms. Currently, the patient is symptom-free on an as-needed dosage schedule.

Discussion

The etiology of chronic urticaria is not determined in 70 to 80 percent of cases in most reports in the literature. In fact, Jacobson et al[2] have shown that of 11 commonly recommended laboratory and radiologic procedures to evaluate chronic urticaria, none is likely to be of benefit unless specific findings were suggested in the history and by physical examination. Although some studies vary, there seems to be a statistically disproportionate number of middle-aged women with chronic urticaria.

One can readily ascertain that with the incidence of idiopathic chronic urticaria being 70 to 80 percent, the widely held view that allergy is the most common cause is invalid. Several studies have suggested that other vasoactive substances besides histamine may be responsible for idiopathic chronic urticaria.[3,4]

That prostaglandins do have dermal reactivity is not new; however, there seems to be a relative paucity of research in this direction. There are two primary groups of prostaglandins designated as PGE and PGF. The PGE group, specifically PGE_1, seems to be the most dermally reactive.[5-7]

Juhlin and Michaelsson[6] have noted that the vascular effect of PGE_1 is not changed by antihistamines, histamine depletion, atropine, and alpha-blockade with phenoxybenzamine hydrochloride and there is only a mild response to beta-blockade. These observations might account for the poor treatment response of chronic urticaria when using common regimens employing some of these elements.

Conversely, some evidence points to the possibility that PGE may inhibit histamine release. Bourne et al[8] showed that agents that increase synthesis of leukocyte cyclic adenosine monophosphate act to block histamine release at an early stage of the release process. Of course, this would be an important observation if histamine were the sole etiologic agent for urticaria. On the other hand, if prostaglandins are possibly etiologic agents of urticaria, blockade of histamine release will provide less than optimal results, a fact that has generally been noted.[4,9]

The prevailing theory regarding action of prostaglandin inhibitors is their blockade of conversion of arachidonic acid to prostaglandin. Prostaglandins are ubiquitous throughout the body but have been specifically related to the menstrual cycle and the onset of labor in pregnancy. One might postulate that the reason there are proportionately more women with chronic urticaria is that there is higher prostaglandin activity because of menstrual cycle variations.[10] This increase in activity would seem to explain the increase in severity at ovulation and menstrual period observed in the present case.

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