Drugs 1998 Feb 55 (2): 275-276
0012-6667/98/0002-0275/$02.00/0

Fexofenadine

A Viewpoint by Stanley P. Galant

Asthma and Allergy Research Center,
Orange, California, USA

Allergic rhinitis, the most common of all atopic diseases, affects 15 to 20% of the US population at a cost of several billion dollars per year.[1] There are 2 varieties of allergic rhinitis: perennial and seasonal. Perennial allergic rhinitis can occur at any time of the year, usually in young children, and is caused by indoor allergens such as the house dust mite, animal danders, mould and cockroaches. Seasonal allergic rhinitis occurs in spring and autumn, usually in older children and adults, and is caused by tree, grass and weed pollens. The latter is characterised by paroxysmal sneezing, itchy nose and eyes, profuse rhinorrhoea and nasal obstruction, which can be severe. Aggressive treatment is important not only for relief of sinus symptoms, but also to prevent complications such as eustachian tube malfunction, which can be associated with otitis media with effusion, sinusitis and bronchial asthma. The treatment strategy for any allergic disease should include medication and avoidance of precipitating allergens, with addition of immunotherapy if required.

Histamine is an important mediator of inflammation in seasonal allergic rhinitis and is responsible for most, if not all, of the symptoms mentioned above. Fexofenadine, a metabolite of terfenadine, is a new nonsedating H_1 antagonist, which, like terfenadine, inhibits mediator release from mast cells. It has demonstrated similar properties to other second generation antihistamines such as loratadine and cetirizine in that it selectively binds to peripheral H_1 receptors and not to muscarinic receptors, and thus does not have sedative and anticholinergic effects, which frequently occur with first generation antihistamines.

The clinical efficacy of fexofenadine is similar to that of terfenadine. However, since it does not undergo extensive hepatic metabolism, fexofenadine is unlikely to interact with drugs that are metabolised by the hepatic cytochrome P450 enzyme system. Of potentially greater importance is the finding that fexofenadine does not interact with cardiac potassium channels, and thus does not share the propensity of terfenadine to increase the cardiac QT interval, an adverse event that can be associated with serious arrhythmias.

In several multicentre double-blind controlled studies in patients with seasonal allergic rhinitis, fexofenadine 60mg twice daily significantly reduced total symptom scores, which included sneezing, rhinorrhoea, and itchy nose, eyes and palate. Nasal congestion was not affected, a finding consistent with those of studies investigating the effects of other antihistamines. The safety profile of fexofenadine was excellent in these studies.

Thus, fexofenadine appears to have similar efficacy and tolerability to loratadine and cetirizine, but is somewhat less sedating than the latter in patients with mild to moderate seasonal allergic rhinitis. Assuming that a once daily formulation including a chewable tablet and/or liquid form for children can be developed, fexofenadine should be competitive with currently available therapies. ▲

Reference

1. Naclero RM. Allergic rhinitis. N Engl J Med 1991; 325: 860-9