of MVP and BD was first reported in China.\textsuperscript{5,6} Shen et al.\textsuperscript{5} from Shanghai, China, postulated that MVP may have been the result of structural damage and functional derangement caused by the underlying vasculitis of BD. Whereas the association of MVP and BD may be either causally related or coincidental, it is important to keep in mind that, although BD is relatively rare in the United States, MVP is very common.

BD occurs most frequently in Japan and in the Mediterranean countries, and also in the population linking these two areas to each other. It often occurs most frequently between latitudes 30° and 45° north, in Asian and Eurasian populations. This area coincides with the old Silk Route. Thus, BD is often also called “Silk Route disease.”\textsuperscript{6}

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References

Montelukast and Churg-Strauss Syndrome

To the Editor:

In their recent review (March 2000), Wechsler and coauthors\textsuperscript{1} stated that the relationship between montelukast and Churg-Strauss syndrome (CSS) is not a direct drug effect, but rather it arises from unmasking a previously existing condition via corticosteroid withdrawal. However, since the true background incidence of CSS in asthmatics remains unknown, the possibility that leukotriene modifiers have a causative role in CSS, perhaps due to an allergic response, remains equally plausible.

First, there seems to be a high total number of cases of CSS associated with leukotriene-modifying agents. Wechsler and Drazen\textsuperscript{2} reported that, as of July 1999, there had been at least 52 suspected cases of CSS associated with zafirlukast, 42 of which were confirmed by Zeneca (Wilmington, DE); and at least 52 suspected cases of CSS associated with montelukast, 36 of which were confirmed by Merck (Whitehouse Station, NJ). A recent presentation indicated that > 100 cases of CSS have been reported to the US Food and Drug Administration, in association with the use of leukotriene modifiers.\textsuperscript{3}

Historically, there was no similar increase in the number of CSS cases when cromolyn was introduced for the treatment of perennial asthma in 1973. There was a report of a few cases that appeared to match the description of CSS.\textsuperscript{4} Unfortunately, no data were reported on the incidence of CSS for the years following the introduction of beclomethasone dipropionate and triamcinolone in this country. These drugs also were widely used as oral steroid-sparing agents. Perhaps their manufacturers could provide additional data on the occurrence of CSS with these agents.

Data from the United Kingdom, from the Medicines Control Agency Committee on Safety of Medicines, reported 63 cases of CSS through the Yellow Card Scheme since 1963.\textsuperscript{5} Of these, 59 cases were documented during the 2-year period from 1998 to 1999, and 90% of the cases were associated with drugs used to treat asthma, particularly leukotriene receptor antagonists. Indeed, in many cases there was documented evidence of reduction or withdrawal of oral corticosteroid therapy prior to the onset of the reaction. Although the reliability of reporting cases of CSS by general practitioners is questionable, it is important to note that other steroid-sparing agents introduced in the United Kingdom during that period, such as beclomethasone, budesonide, and salmeterol, were not accompanied by a similar rise in CSS, although occasional case reports were noted.

In addition, cases of CSS have been observed in steroid-naïve individuals.\textsuperscript{6} This possibility is also mentioned by the manufacturer in the package insert.

In light of the above, the concluding statement by Wechsler et al. that “montelukast does not appear to directly cause the syndrome (CSS) in these patients,” would seem to be somewhat premature. Clearly, further study is imperative.

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ECG Abnormalities Associated With Hypocalcemia

To the Editor:

The recently published case report in CHEST (July 2000)\textsuperscript{1} concerning the ECG manifestations of severe hypocalcemia producing injury current mimicking acute myocardial infarction merits additional clinical commentary. The authors also deserve to be complimented on the excellence of their presentation and discussion of the pathophysiology involved and their astute conclusion regarding the most likely cause of the dramatic ECG abnormalities as being due to coronary vasospasm. The report has the possibility of being a landmark article. The central issue discussed revolves around the production of an acute anterior