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Correspondence to: Robert Guthrie, MD, Professor of Emergency


To the Editor:

I appreciate the recognition by Drs. Weiss and Tillotson that the time between the acceptance of our manuscript and its publication has seen a rapid evolution in our understanding of the proper treatment of Streptococcus pneumoniae lower respiratory tract infections.

I also share their concern as to why the guidelines persist in recommending doxycycline in the face of increasing S pneumoniae resistance to it.

I agree with and support their comments on the proper use of fluoroquinolones in respiratory infections. I have long been concerned, but lacked the evidence to include in the manuscript that the widespread use of older quinolones with marginal MICs for S pneumoniae (specifically levofloxacin) could foster the development of quinolone resistance in this dangerous bacteria. The reports cited by Drs. Weiss and Tillotson support this concern.

Therefore, I think it is important not only that we use quinolones prudently, but that we also use the newer quinolones, specifically gatifloxacin, moxifloxacin, and gemifloxacin if it becomes available, when a quinolone is indicated in the treatment of acute exacerbations of chronic bronchitis: the Canadian Ciprofloxacin Health Economic Study Group. Chest 1998; 113:131–141

I appreciate them bringing this new, emerging, and critically important data to our attention. I completely support their concerns about the proper choice of quinolones to reduce the development of resistance to these critically valuable antimicrobial agents.

Robert Guthrie, MD
The Ohio State University
Columbus, OH

Adrenal Suppression Related to Inhaled Corticosteroids Revisited

To the Editor:

With growing concern, we read the report by Todd et al (October 2001 supplement) about clinically significant adrenal suppression related to the administration of inhaled corticosteroids. That article and others, such as the one by Zimmerman et al, have been linked to the use of the most potent of the inhaled corticosteroids, fluticasone, probably because of its well-known high lipophilicity, which confers a very high volume of distribution and a long plasma half-life. Another report described a 6-year-old girl with obvious features of hypercortisolism who experienced an acute adrenal insufficiency episode that had been induced by the administration of budesonide after her treatment had been switched from fluticasone.

Earlier, in 1993, we had reported on a patient with similar clinical features. This child had been treated with customary doses of beclometasone without any warning or evidence of hypercortisolism. All these reports were largely ignored and were considered to be extremely rare situations.

A few years later, Carrel et al described the condition of a 3.5-year-old child who had been treated with a low dose of prednisone (2.5 mg every other day) and inhaled beclometasone (255 mg/d) and who experienced an episode of severe hypoglycemia and cortisol deficiency that was virtually identical to that observed in our patient. At that time, this observation came to no surprise to us, since Tabachnik and Zadik also had reported severe adrenal suppression even at conventional steroid doses.

Recently, Lipworth has repeatedly elaborated on the issue of adrenal suppression by inhaled fluticasone and has raised concerns about the probable large volume of distribution of mometasone furoate, despite previous claims for its very low (ie, < 1%) systemic bioavailability.

In view of these observations, we encourage physicians to be aware of the possibility of systemic effects of inhaled corticosteroids in some asthmatic patients and of its clinical relevance, as these rare but very real events may occur not only with fluticasone treatment but also with all inhaled steroids that are currently on the market.

Fernan Caballero-Fonseca, MD
Mario Sánchez-Borges, MD
Caracas, Venezuela

Correspondence to: Fernan Caballero-Fonseca, MD, Immunology Department, Centro Médico-Docente La Trinidad, Caracas, Venezuela

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