CORRESPONDENCE

Explaining differential effects of tiotropium on mortality in COPD

The editorial by Jenkins and Beasley makes a speculative recommendation that tiotropium Respimat should not be prescribed in the treatment of chronic obstructive pulmonary disease (COPD), being primarily based on meta-analysis where mortality was not the primary end point. The meta-analysis by Singh et al reported that treating 124 patients per annum with tiotropium Respimat 5 ug resulted in one additional death, although the associated 95% CI of 32 to 5682 clearly indicates that the data are not particularly robust. In considering the risk-benefit ratio of tiotropium one has to consider the seed and the soil, in terms of the degree of systemic exposure and the pre-disposing cardiovascular status. There is a lack of biological plausibility for the apparent disconnect between the apparent increased mortality with tiotropium Respimat on the one hand, but reduced mortality with the Handihaler on the other. Such an opposite effect on putative cardiotoxicity seems hard to justify on the basis of a 22% difference in systemic exposure (as area under curve (AUC)) between the respective mean oxygen saturations of 92.5% and 91.3%—that is, these were clearly patients who would be at risk from potential cardiotoxicity with tiotropium.

Thus rather than extolling the merits of data torture to substantiate premature recommendations about the use of withdrawal of tiotropium Respimat, we would advocate waiting for more definitive data from the ongoing TIOSPIR study. In the meantime, on a more pragmatic basis, for those patients who prefer to use the Respimat device, physicians could perhaps use a 2.5 ug dose of tiotropium instead, which would exhibit lower systemic bioavailability than the Handihaler device, which has been shown to reduce mortality on COPD in a real life setting.

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