LETTERS

Pneumonia risk stratification in tropical Australia: does the SMART-COP score apply?

William B Grant

To the Editor: The recent article by Davis and colleagues reported that the SMART-COP score underestimates the severity of pneumonia in tropical northern Australia, but can be improved by using locally relevant additions. The authors’ revised scoring system, SMARTACOP, increased the score for an albumin level <35 g/L and added Aboriginal or Torres Strait Islander status as a variable. While these additions are useful, the reason for adding ethnicity was not fully clarified.

A factor overlooked was low serum 25-hydroxyvitamin D [25(OH)D] levels among dark-skinned Australians. Smoking, identified as a marginally insignificant risk factor, is also associated with lower serum 25(OH)D levels. Vitamin D enhances the innate immune system through induction by 1,25-dihydroxyvitamin D of cathelicidin and defensins, which combat several types of bacterial and viral infections including upper respiratory tract infections.
In the 1918–1919 influenza pandemic in the United States, many deaths were due to pneumonia that occurred as a complication of influenza infection. An ecological study found that indices for levels of vitamin D production from solar ultraviolet-B irradiance explained 50% of the variance in pandemic case-fatality rates among 12 communities. The mechanisms proposed for the beneficial effect of vitamin D were reduced proinflammatory cytokine production, which would reduce damage to the epithelial lining of the lungs, and induction of cathelicidin and defensins to fight the secondary bacterial pneumonia infection.

If sera are available for those included in the Australian SMART-COP study, they could be analysed for 25(OH)D levels to test this hypothesis.

Competing interests: I receive funding from the UV Foundation and Bio-Tech-Pharmacal. I received honoraria from the North Coast Cancer Foundation for a talk on vitamin D and breast cancer. I have received funding from the Sunlight Research Forum (The Netherlands) and the Vitamin D Society (Canada) for preparation of other manuscripts.

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IN REPLY: We thank Grant for his interest in our study on pneumonia severity assessment in tropical Australia. Our revised scoring system included increased weighting for hypoalbuminaemia, as well as adding a point for Indigenous status, because these two factors had the strongest association with the need for intensive respiratory or vasopressor support on univariate analysis. Unlike vitamin D status, these and the other factors included in the scoring system are readily available measures that can be used in the clinical setting to rapidly predict the need for intensive support. The scoring system was not intended to identify underlying aetiology or risk factors for severe pneumonia. For example, Indigenous status is likely to be a surrogate measure for undiagnosed comorbidities, lack of access to health care, and socioeconomic disadvantage.

We agree that vitamin D is important in immune function and that the levels of insufficiency that result in impaired resistance to infection are not well defined. Most data on vitamin D deficiency in dark-skinned populations in Australia come from temperate areas, and the reference offered by Grant to support the concern about vitamin D deficiency does not cite any data from Australian populations north of southern Queensland. Further studies are needed on the prevalence of vitamin D deficiency in Indigenous Australians in tropical areas, and the additional contribution of vitamin D deficiency independent of known risk factors of severity and outcome.

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