

immunoglobulin and oral aspirin was started, the patient recovered and was discharged on day 25. Follow up at one year is normal.

The aetiology of Kawasaki disease is unclear, but superantigens may play a part.^{2,3} By interacting directly with class two major histocompatibility complex molecules, superantigens can stimulate polyclonal T cell activation with subsequent massive cytokine release. Mycoplasma arthritis has been shown to produce superantigen,⁴ and it is therefore possible that other mycoplasma organisms may do likewise. Superantigen is also produced by staphylococci, streptococci, and retroviruses, all of which have been associated with cases of Kawasaki disease. However, to our knowledge no reports of Kawasaki disease related to mycoplasma infection have been recognised.

We postulate that the development of Kawasaki disease after mycoplasma infection in this patient may have been due to the production of superantigen by the mycoplasma organism.

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BCG and prevention of tuberculous meningitis

Thilothammal and colleagues' case-control study on the effectiveness of BCG vaccine in preventing tuberculous meningitis¹ was well conducted and did discuss the possibility of biases, but left some points that deserve clarification and discussion, as they may affect the validity of the results.

Firstly, it is unclear whether the authors accounted for matching in their analysis, as they did not mention it. The appropriate multivariate analysis technique applicable to matched case-control studies is conditional logistic regression, and the use of unconditional logistic regression for matched data bias the odds ratio towards the null.² In this study it would have therefore underestimated BCG effectiveness.

Secondly, children with febrile convulsions may have not been the best possible control group, as apparently the catchment areas were not the same for cases and controls (in the paper the authors argue that neighbourhood controls would have been best, but unfeasible as 'cases coming from different parts of the state was a limiting factor'). Selection bias may have occurred if the study bases (catchment areas) were different,³ and bias would have been towards over or underestimation of BCG effectiveness depending whether the BCG coverage was respectively lower or higher in the catchment area of cases as compared to the catchment area of controls.

Finally, the provided explanations for the lack of effectiveness of BCG in preventing tuberculous meningitis in the 8-12 years age group are all plausible. Further exploration of this finding will be of considerable interest, especially if waning of immunity is the true reason, as this information can be useful

when considerations about the need and the optimal timing for a second dose of BCG aiming at prevention of tuberculosis and/or leprosy are made.⁴

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Dr Thilothammal and coauthors comment:

Each case was not matched with controls as a set. But, block matching was done for the age, in order to have equal number of cases and controls in each age group. Matching was not taken into consideration in the logistic regression analysis.

Though the controls were selected from the hospital, the demographic parameters like type of house, maternal education, nutritional status, and crowding in the house were comparable between cases and controls indicating that both groups have probably arisen from similar population.

We agree with Dr Nishioka that further studies in children of an older age group will be both interesting and useful in guiding policies regarding BCG vaccination.

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