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However, much larger prospective studies will be required to determine if there are significant differences in the rates of postoperative complications with and without patching.

Conflict of interest

The author declares no conflict of interest.

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LW Lim¹, MC Chew¹ and CS Tan^{1,2}

¹National Healthcare Group Eye Institute, Singapore, Singapore ²Department of Ophthalmology, Tan Tock Seng Hospital, Singapore, Singapore

E-mail: Colintan_eye@yahoo.com.sg

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Sir, Response to Lim *et al*

We thank Lim *et al*¹ for their constructive comments and concur that a larger, prospectively designed study is certainly required. Our data were retrospective, and despite including a significant number of patients (1407) it was never our intention to statistically prove that omitting a shield confers a safety advantage over shielding. The data were collected and published to illustrate that not shielding in our practice over the last 12 months conferred no disadvantage. We hoped to stimulate discussion as to why routine shielding still occurs despite advances in surgical technique with some interesting comparative data.

We also would like to reiterate the differences between shields and patches. Patches protect the ocular surface against particulates and provide visual occlusion. Their role in the immediate postoperative period has both supportive (as above) and derisory² evidence. Shields are supplied to protect surgical wounds and are commonly prescribed for up to 3 weeks following cataract surgery at night. A small group of our patients were randomly selected and questioned about their experiences of shields. Comparing these shield-related responses to studies on patching is not justified.

Conflict of interest

The authors declare no conflict of interest.

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D Lindfield

Sutton Eye Unit, Sutton Hospital, Surrey, UK E-mail: drdanlindfield@gmail.com

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Sir,

Case report of cytomegalovirus retinitis in an HIV-positive patient with a CD4-count nadir of 254 cells per μ l

Cytomegalovirus retinitis (CMVR) is an AIDS-defining diagnosis, and typically occurs when CD4 counts fall below 50 cells per μ l.¹ We report an unusual case of CMVR in a patient whose CD4 counts never decreased below 250 cells per μ l.

Case report

A 30-year-old man was diagnosed with HIV infection 3 years ago, and CD4 counts remained between 600 and 700 cells per μ l since diagnosis. After 2 years, his counts decreased from 565 to 426 cells per μ l over 3 months. Antiretroviral therapy (ART) consisting of tenofovir/ emtricitabine and efavirenz was commenced. Three months later, he complained of right eye blurring with floaters. Vision was 6/9 and fundoscopy revealed active CMVR, corroborated on aqueous PCR for CMV (Figure 1). His CD4 count was 254 cells per μ l and HIV viral load was 42 593 copies per ml that increased to 165 800 copies per ml 2 weeks later. He had no other AIDS-defining illnesses.

Comment

CMVR is a late manifestation of AIDS when CD4 counts are <50 cells per μ l. Although reports have documented



Figure 1 Nine-view fundus photograph of the patient's right eye, showing both superotemporal and active CMV retinitis involving 40% of inferior retina.

cases in patients with CD4 counts >100 cells per μ l at time of diagnoses, they had low CD4 counts before diagnosis of CMVR, before or following ART.² Our case is unique that CD4 counts before the diagnosis of CMVR had never decreased below 254 cells per μ l.

Postulated reasons for the lack of correlation between CD4 counts and occurrence of CMVR include the functional dysfunction of CD4 T cells in AIDS. Although there is high correlation between counts and function, CD4 counts are a surrogate marker for immune dysfunction and do not reflect functional abnormalities in the immune system.³ Initial increment in CD4 counts after ART may be because of systemic redistribution of memory non-specific T lymphocytes, whereas the actual increase in CMV-specific T cells occurs later. CMVR may occur during this latent period between quantitative restoration of CD4 counts and actual functional restoration of immunity. Moreover, clonal deletions of CMV-specific T lymphocytes can occur, impairing immunity against CMV, while maintaining overall high CD4 counts.

In addition to absolute CD4 counts, CMVR may be correlated with other predictive factors; for example, rapid decline in CD4 counts by >100 cells per μ l after diagnosis of CMVR,^{1,3,4} high HIV viral loads >100 000 copies per ml, and presence of CMV viremia.⁵ Other risk predictors, such as trends in CD4 counts and viral load should also be considered. Therefore, the clinical diagnosis of CMVR should not be dismissed in the presence of a normal CD4 count.

Conflict of interest

The author declares no conflict of interest.

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BC Ang and SC Teoh

National Healthcare Group Eye Institute, Tan Tock Seng Hospital, Singapore, Singapore E-mail: Stephen_Teoh@ttsh.com.sg

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Sir,

Comment on 'Effects of Merogel coverage on wound healing and ostial patency in endonasal endoscopic dacryocytorhinostomy for primary chronic dacryocystitis'

We read the article on 'Effects of Merogel coverage on wound healing and ostial patency in endonasal endoscopic dacryocytorhinostomy for primary chronic dacryocystitis' by Wu *et al*¹ with great interest. The surgical procedure involved in this randomized controlled trial was clearly presented and reproducible, and the paper made excellent use of both per-protocol and intention-to-treat analyses in interpretation of the data. We had the following observations regarding the methodology and interpretation of the results.

The diagnosis of primary chronic dacryocystitis was made on the basis of a history of epiphora with purulent discharge and regurgitation on nasolacrimal irrigation. Unfortunately, either no attempt was made to locate the level of obstruction or it was not reported. Many factors influence the outcome of endoscopic dacryocystorhinostomy, and one of the most important prognostic factor is the level of obstruction in the lacrimal system.^{2,3} A recent study from South Korea showed that the ductsac junction obstruction was treated most successfully, followed by nasolacrimal obstruction, common canaliculus obstruction, and saccal obstruction.⁴

Various clinical tests are available to identify the level of obstruction of the lacrimal system. Simple tests such as probing and Jones test can identify punctual and canalicular obstruction, and can be performed in the office. Dacryocystography is considered the gold standard and can localize obstruction within the lacrimal sac or duct.⁵