Infections post-Nexplanon® insertion

We read with interest the letter by Dr Chaudhry\cite{1} entitled ‘Adverse reaction to Nexplanon®’ in the July 2013 issue of the Journal. Over the past 5 months our department has experienced certainly two, possibly three, incidents of
skin infection secondary to a contraceptive implant in patients with a comorbidity of atopic eczema.

The first case was a 15-year-old girl who developed an infection around 10 days post-subdermal implant fit. A skin swab revealed a growth of *Staphylococcus aureus* sensitive to both flucloxacillin and co-amoxiclav. The patient initially received oral flucloxacillin and then a secondary treatment with oral co-amoxiclav. However, despite the organism sensitivities the wound failed to heal, necessitating removal of the extruding implant (Figures 1 and 2). Interestingly, the patient had a history of atopic eczema, which was present in an adjacent area to the implant site.

The second patient was a 33-year-old woman who developed an infection at the entry site around 1 week post-implant fit. Moderate to severe atopic eczema was documented and felt to be poorly controlled. The implant site infection was treated with oral co-fluampicil and initially appeared to improve. However, after the antibiotic period, erythema was noted to track from the ante-cubital fossa up towards the implant insertion site. Again the implant verged on self-extrusion and was therefore removed.

Evidence for an implant-related infection was less clear with our third patient, who again had atopic eczema. The site surrounding the implant exhibited a prompt local erythematous reaction within 7 days of fitting. Although antibiotics appeared to settle the infection, the patient elected to have the implant removed despite symptom resolution. In contrast to the first two cases, there was no pus present at the implant entry site, and therefore with hindsight it is not entirely clear whether the erythematous reaction was due to infection or alternatively an allergic-type reaction.

Following a review of these three cases, we liaised with our local microbiology department to discuss strategies for preventing post-implant infection in women with atopic eczema. Patients with atopic eczema have a risk of staphylococcus colonisation. When eczema is poorly controlled a patient can potentially shed relatively large volumes of squamous epithelial cells carrying *S. aureus*.

To reduce the risk of implant-related infection in such patients we have now adopted three simple measures. First, we allow sufficient time after cleaning the skin with chlorhexidine solution to ensure that the skin is completely dry. Second, we ensure that there is a sterile field with drapes, to minimise the risk of staphylococcus-colonised squamous epithelial cells from falling back onto the already cleansed skin. Finally, an assessment of the patient’s skin and eczema status should be made as an implant would arguably be best placed when their eczema is under optimal control.

We have been fitting implants in this service for over 10 years and have never knowingly had a problem with implant infections. We would be very interested to hear if any other contraception services have also seen any patients with atopic eczema who have developed a post-implant infection.

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