To Buruli or not to Buruli

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Kiiza and Wood (January 2012, p. 48)¹ describe a case report of an 18-day-old infant with a purulent necrotizing cutaneous lesion of the anterior thorax which they subsequently clinically diagnose as Mycobacterium ulcerans (Buruli ulcer). Although the infant’s necrotizing lesion was surgically drained via a manual probe on three occasions, confirmatory laboratory testing for M. ulcerans was not done. To our knowledge, no laboratory-confirmed case of M. ulcerans in the neonatal period has been described in the medical literature. M. ulcerans transmission is hypothesized to occur via bites of aquatic insects, to which the child was not likely to have been exposed, and there has been no published evidence for human-to-human transmission.²

As M. ulcerans is a slow-growing mycobacteria, even in an infant with an under-developed immune system, the incubation time of 14 days is extremely short. For these reasons, we would like to call the diagnosis of M. ulcerans into question.

Bacterial abscess and necrotizing fasciitis should be included in the differential diagnosis of a rapidly progressive necrotizing cutaneous infection in a neonate. Bacterial abscesses are most commonly caused by Gram positive bacteria such as Staphylococcus aureus and Group A streptococcus (S. pyogenes) while neonatal necrotizing fasciitis is often a polymicrobial infection (Gram positive, Gram negative and anaerobic bacteria). Despite the polymicrobial nature of neonatal necrotizing fasciitis, the most commonly isolated bacterium is S. aureus.³

The mother of the affected infant had breast fissures on admission which subsequently developed into abscesses requiring incision and drainage. Both mother and infant were treated with rifampin monotherapy on the assumption that the infection was caused by M. ulcerans and, since both improved, the treatment was considered to have been successful. However, breast abscesses are most commonly caused by S. aureus.⁴ As the infant could have easily become colonized with S. aureus from the mother’s superinfection of breast fissures, there is reasonable likelihood that both infant and mother had cutaneous S. aureus infection rather than an infection caused by M. ulcerans. Improvement in both mother and child after being treated with rifampin monotherapy could be explained by rifampin’s significant in vitro activity against S. aureus rather than M. ulcerans.⁵ In addition, both mother and child could have improved as a result of both undergoing incision and drainage of their abscesses which is the standard of care in the treatment of bacterial abscesses.

Rifampin monotherapy is not recommended for the treatment of M. ulcerans. The World Health Organization⁶ recommends first line treatment of M. ulcerans with rifampin and intramuscular streptomycin. For pregnant and/or lactating women, streptomycin may be replaced by clarithromycin in order to avoid potential toxicity.

References


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