Reviewer's report

**Title:** Sporotrichoid cutaneous infection by Mycobacterium haemophilum and kansasii in an IgA-deficient man

**Version:** 3  **Date:** 15 July 2010

**Reviewer:** PK Ramdial

**Reviewer's report:**

Major compulsory revisions

1. The link between the described NTM infection and IgA deficiency requires strengthening. Please attempt to:
   - provide a pathomechanism for skin-based disease in the setting of IgA deficiency
   - provide information on the presence or absence of IgA-associated mucosal-based diseases
   - We are informed that “interestingly our patient’s history revealed an IgA-deficiency”. Perhaps it would be of value to know how / why the IgA studies were undertaken
   - unless a good pathogenetic link to IgA deficiency is formulated, the association, at most, appears fortuitous. One needs to make an informed statement to this effect.

2. Although there is synchronous evidence of Mycobacterial disease, there is no dual infection in a single lesion, and no clarity that the co-cultures were from lesions on the right side, to indicate sporotrichoid co-involvement. Please attend to this.

3. The sporotrichoid nature of the clinical lesions is not imaged to optimal effect in the submitted figures. There are many randomly placed flat and nodular lesions. Please demonstrate an unequivocal sporotrichoid pattern of spread in the limb/s.

4. My greatest difficulty is with the clinical presentation: the skin lesions are not “sporotrichoid” exclusively. There are disseminated cutaneous lesions. This does not therefore specifically mean that the disease was a primary focus in an acral extremity with spread along the lymphatics. The pattern of cutaneous involvement favours disseminated disease, as seen with blood spread.
   a. If the lesions did not contain acid fast bacilli, they are “older”; with lymphangitic spread, ipsilateral lymphadenopathy is a usual finding (in my experience: reactive/dermatopathic or infective). There was NO lymphadenopathy. We therefore require a mode of spread that will allow dissemination of infection other than by local lymphatics.
   b. We do not have a record of pulmonary or chest x-ray findings. This is a crucial omission given that M.kansasii usually targets the lung.
c. The clinical background should at least include occupational or hobby details that may strengthen or negate a primary cutaneous source of infection.

d. The authors state that there was no “clinical” evidence of mycobacterial systemic infection. This requires explanation. We need to be informed how systemic disease was excluded. Were blood and sputum cultures undertaken?

Minor Revisions:
1. Spelling errors”: mycobacterium,
2. Other corrections: culture instead of cultivation, circumscribed instead of sharp,
3. Clinical findings: Ziehl Neelsen not included as special stain used in histopathological assessment of skin biopsy.
4. The Mycobacterial profiling are part of the “Results” and should not be included under “Methods”. As all the results are included under “Clinical findings”, perhaps the best fit would be as a clinical finding.

Discretionary Revisions:
1. Were the lesions that were biopsied for histopathological assessment the same that were biopsied for culture purposes? I would undertake Mycobacterial PCR testing (nested, if necessary) or cloning/sequence studies to confirm a Mycobacterial origin and subtype in the histopathological paraffin sections.
2. Post treatment images demonstrating disease resolution will enhance the submission.

Declaration of competing interests:

I have no competing or conflicting interests that I am aware of.