Leprosy stigma & the relevance of emergent therapeutic options

In the history of modern medicine, in terms of physical stigma, leprosy is a predominant disease and the probable reason is that the disease, even in the lepromatous spectrum, does not account for mortality but only for the visible deformity which proves to be a major cause of life-long stigmatization. Stigma has been defined by Goffman as ‘an attribute that is deeply discrediting, and the stigmatized individual is one who is not accepted and not accorded the respect and regard of his peers; one who is disqualified from full social acceptances’.

The stigma in leprosy is at three levels - the patients (self-perceived stigma), the relatives and the community. A study from Nepal noted that varied myths still surround the disease, with only 62.6 per cent being aware of it being caused by a bacteria and a significant proportion (36%) associating it with various other irrelevant causes, including bad blood, curse, heredity and bad deeds. Only 43.8 per cent knew that leprosy is transmitted by prolonged close contact with leprosy patients, and surprisingly, 25.7 per cent reported religious rituals as its treatment.

From the patients’ perspective, the disease affects various aspects of life including marriage, employment and social interaction, especially with the presence of visible deformities (specific or non-specific). A study from Ghana reported that persons cured of leprosy preferred to stay in the leper colony. This was due to self-stigma, isolation and neglect, and would make effective treatment inconsequential in leprosy.

While the Global Partnership for Zero Leprosy has delineated three goals of zero transmission, zero disability and zero discrimination, the first goal is difficult to achieve and the last goal is based on the prevention of the second
goal. While effective education and more emphasis on leprosy training in the medical curriculum is the need of the hour, probably one of the most effective methods to reduce stigmatization is early diagnosis and appropriate treatment. A recent study found that patient delay (of more than three months) and healthcare provider delay (of more than one month) were two significant risk factors for disability among adult leprosy cases. It is thus useful to intervene early and effectively with appropriately tailored therapy with the twin goals of effectively reducing transmission and preventing disability. There are varied and important treatment scenarios which are useful to refresh and implement beyond merely dispensing the multidrug therapy (MDT) kits. Even though the emergent focus is on disabilities, simple concerns are rarely addressed such as clofazimine pigmentation which add to the stigma of leprosy. Although clofazimine forms the bulwark of therapy, largely as resistance to it is unproven to date, there is a need to possibly look at novel biomimetic preparations which have equal efficacy and cause no skin pigmentation. Furthermore, the fixed duration treatment is at variance with ground reality where patients with bacteriological index (BI) >4 at diagnosis usually get extended therapy. It is important to remember that relapses take about seven years to appear and there is a dearth of studies that examine this aspect consequent to uniform MDT regimen.

Treating and managing the side effects of drugs in MDT are largely ignored aspects of treatment, more so with the newer drugs. Resistance testing is sparse, and hence, such cases can transmit resistant strains to the community. Reports of reactions occurring in infections with resistant strains are an ominous, though largely ignored, aspect as such cases, with the added effects of concomitant steroids and oral immunosuppressants, can predispose to dissemination of the resistant strains. Nerve damage is the most important problem in leprosy, which is not surprising considering the marked tropism of Mycobacterium leprae for Schwann cells (SC). Trypanosoma cruzi and M. leprae are two unique organisms with a predilection for the SC. M. leprae has undergone a reduction in its genome, which enabled it to attain the lowest guanine-cytosine content (approximately 58%) among mycobacteria, and this process, referred to as genomic ‘reductive evolution’,
Table 4. Persistent nerve impairment is an emergent need both for diagnosis and prevention of disabilities. The primary focus of reactions is to prevent nerve damage and to address the issue of neuropathic pain. While steroids have been used, the regimens vary and the impact of steroids and other measures on preventing progression of nerve damage needs to be assessed. The lumping of both downgrading and upgrading reactions into type 1 reaction (reversal reaction) is unfortunate as downgrading reactions are evidently seen in clinical practice and these do not require long duration of steroids and are easier to control. Downgrading reaction is a distinct reaction that occurs without treatment. The importance is that the steroid dose and duration are either less or steroid may not be required in type 1 downgrading reaction. This is because suppressing a heightened immune response (type 1 upgrading) is more difficult than suppressing a downgrading reaction. Upgrading reactions are better documented than downgrading ones; the reason could be that patients under treatment are more likely to have their progress observed. Another therapeutic relevance is that lumping downgrading reaction with upgrading reactions as reversal reactions entails such cases to treatment with long duration of steroids, and in tuberculosis (TB)-endemic countries, like India, there is a real risk of reactivation of TB with prolonged steroids. A review of reported cases of leprosy and TB co-infections noted that 7 of 10 (70%) cases were on steroids before the diagnosis of TB, suggesting that steroids may be a risk factor for reactivation of TB in leprosy patients. This highlights the need to include interferon gamma release assay (IGRA) in the workup before treatment of leprosy reactions in TB-endemic countries.

A very relevant issue for stigma remains disability management and this is woefully inadequate. The risk factors for disabilities include male sex, multibacillary leprosy and leprosy reactions with steroids, and possibly early and adequate treatment would be useful to prevent disability.

For a large number of medical professionals, dermatologists are the only trained task force to address leprosy and it is imperative to re-emphasize the various treatment options and the role of early diagnosis and intervention that can effectively manage and prevent disabilities and reactions. Various methods of mitigating stigma have been suggested, mainly focusing on documenting the level of stigma in the communities and healthcare services and also assessing objectively its impact on patients. It is therefore recommended to address the negative attitude against patients at the community level through outreach efforts. However, in countries where health budgets are already constrained by other diseases of national importance and where widespread illiteracy is a real issue, such laudable ideas usually do not succeed consistently. Therefore, the present measures need to be honed and implemented fully before hurriedly enforcing new measures including single-dose regimen or vaccines. As it is important to understand the financial and logistical restraints for any national programme. Further, to convince dermatologists to implement ‘novel’ interventions is another challenge, largely ignored by the national and international bodies recommending guidelines in leprosy.

Thus, while stigma management and identification are important, it is our view that robust management of leprosy reinforcing the existent principles would be more effective in preventing disabilities than the laudable but complex issues that address stigma in recommendations. Stigma is ingrained into the conscience of the community and is a consequence of delay or lack of adequate treatment, and possibly to a large extent, ‘prevention by early diagnosis and adequate treatment’ could be a more practical approach to manage stigma in leprosy.

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