The Madras Leper Hospital and leprosy management in 19th century India

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Leprosy remained a major public-health issue in India until the later decades of 20th century. The National Leprosy Eradication Programme, launched in 1955, contributed to reasonably effective management[1]. Henry Carter (1831–1897) (Note 1) of Bombay Medical Service, after his travel in Norway studying the epidemiology of leprosy, issued an extensive memorandum to the government on leprosy management in 1884 (ref. 2). By the 1880s, approximately 120,000 patients of leprosy (PoLs) existed in India, which is suggested[3] to have dropped to 102,000 by the 1920s. In British India, the Madras Presidency had the lowest number of PoLs, yet the maximal government-supported treatment for the illness and asylums (≈ hospitals; Note 2).

The biology of *Mycobacterium leprae* (Actinomycetales: Mycobacteriaceae) was known only in 1874 (ref. 4). How leprosy management occurred in the Madras Presidency, before 1874, forms the remainder of this note. This note builds on a paper by the Madras surgeon van-Someren[5] (Figure 1, Note 3), with supplementary remarks and relevant annotations.

Madras Leper Hospital (1815–1826, 1826–1856)

A public letter of 26 September 1816 indicates that an exclusive leprosy hospital existed in Madras. This hospital, formally known as the Madras Leper Hospital (MLH), and informally the leper asylum, was in Washermanpet (13°06′N; 80°17′E, north of Madras city) first as a part of, and later as a distinct entity opposite to Monégar Choultry (Note 4), where both male and female PoLs were treated[6]. Before MLH, PoLs were admitted into the Madras Native Infirmary (MNI, Note 4) along with those not suffering from leprosy. By January 1813, the number of PoLs admitted into MNI rose to 40. Because of the prevalent idea that leprosy is contagious, the Superintendent and members of the Managing Committee of MNI wrote to the then Governor of Madras, Sir George Barlow on 20 March 1813 seeking a separate building.

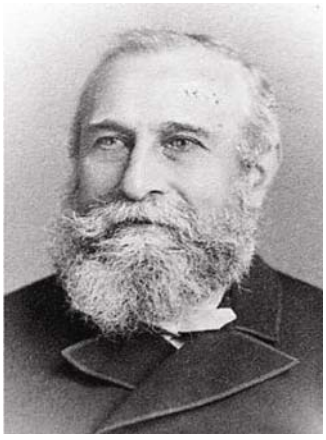

Figure 1. William Judson van-Someren (1824–1911) [http://records.ancestry.com/Maijke_Van_Someren_records.ashx?].

Because no response came forth, another letter from the Managing Committee of MNI followed on 16 July 1813. The Madras Government refused to accept that leprosy was contagious. David Hill, Chief Secretary, Madras Government, responded on 27 July 1813 saying that PoLs at MNI should be encouraged to return to their homes and none more admitted. Hill suggested that those already admitted and hopeless of total recovery should be accommodated separately by the least expensive arrangement achievable by MNI. Consequently, the number of PoLs at MNI dropped to 26, who were shifted to a temporary building. This temporary building grew into a separate establishment, becoming the first hospital in Madras (Note 5) for treating leprosy – the MLH. However, MLH did not survive as an exclusive facility for PoLs for long. On 30 November 1813, James Dalton (Note 6), Medical Officer, MNI, suggested that patients of venereal diseases at MNI be relocated 'into the place lately constructed as the hospital for lepers'. The change came into effect immediately (vide a public letter from the MNI Managing Committee to Hugh Elliott, the Governor of Madras, 1 August 1815).

The Elliott government responded quickly. Land was secured and a building for the hospital erected at an overall cost of 983 star pagodas (Note 7) in 1815. Although the facility was populated by PoLs, a majority of them preferred not to go to MLH. They preferred managing their lives by begging in the streets for a living. Towards the end of 1816, the MLH Managing Committee decided to bring them into MLH by force. Since police powers were weak, the managers of Monégar Choultry and allied charities sought the government's introduction of legislative provisions to confine them in MLH. Such legislation never came forth. Little reference to MLH between 1826 and 1840 exists. However, a grant of Rs 2000, seemingly due to efforts of James Lawder (Note 8), came on 3 July 1839. In his Surgeon's Report to the Government (1840), Lawder indicates that the expanded hospital came into existence on 1 July 1840:

> '… for the reception of patients (of both sexes) – whether Indo-Britons or Natives, suffering from leprosy, an agreeably to the orders of the Government, and its interior economy is wholly unconnected with the Native Infirmary.'

MLH thus became a government institution and the change promoted its stability, efficiency and usefulness. Two new buildings were added in 1856. MLH included 11 wards, three accommodating 31 females and eight accommodating 100 males, with an inter-bed space of 3′ (*c*. 1 m). Not only a tall wall separated the hospital[7] (Note 8), but similar walls separated each ward. Such walls were inappropriate for reasons of cross ventilation in a hospital making the facility similar to a prison, remarks van-Someren[5]. Between 1840 and 1856, the number of in-patients rose to > 100, reaching the maximum (170) in 1854, which, in high likelihood, was due to the rising popularity of MLH. The number of support staff rose to 14. Recognizing the value of providing healthy food to patients, MLH supplied food based on patients' principal food habits, treating the European and Eurasian (Anglo-Indian) patients as one category and Indians as another[5]. Patients were encouraged to

carry out gardening for which 'the premises furnished ample space and opportunities'.

Debate on physical transmission

van-Someren[5] describes the etiology of leprosy as diagnosed by him and as noted in pathology reports at MLH. The following are a few samples of his remarks:

> 'There are at present seventy-five lepers in hospital, in *forty-five* of whom the disease is of the *anaesthetic* form, and in *eighteen* it is *tubercular*. In *twelve* it is *complicated* (Note 9). Does the disease occur sporadically or endemically?… my enquiries as well as personal observations incline me to regard its occurrence as sporadic; ….'

Without including any 'extended' and 'numerically precise' data, he argues that leprosy in southern India is sporadic and is not an inherited illness[5], saying:

> '… that inheritance does not constitute a strong predisposition to the disease, even if do so at all.… The exceptions indeed are so few that it becomes a question when parents and children are really affected, whether the disease is not the result of both being subjected to the same external circumstances favorable to the development of the disease, instead of being attributable in the offspring to a taint inherited from their parents.'

van-Someren challenges Lawder's hospital notes made in 1840s. In his words:

> 'Surgeon Lawder, in the report from which I have already made some excerpts, thus strongly expresses himself regarding the Etiology of leprosy – "That this disease is not only hereditary, but also to a certain extent contagious, cannot be doubted." His assertion is not supported by any given statistical observations, and in the absence of such it must be regarded as a mere statement of opinion that may, or may not have a solid foundation in fact.'

In support of the above remark, van-Someren[5] quotes a table from notes made by Major William Porteous (Note 10), which lists 11 sanitary workers in employment from 2 to 14 years at MLH, none of whom had contracted leprosy.

Treatment efforts

James Dalton (JD) in 1813, while at MNI, which also housed PoLs, had trialled a treatment using a 'compound' (details not available to van-Someren) for treating leprosy. However, van-Someren infers that this compound, in high likelihood, included $HgCl_2$ as the principal ingredient.

JD wrote to the Surgeon at the Madras General Hospital on treating PoLs, while dispatching a box of 500 pills of the 'compound'. According to JD:

> 'One pill to be taken night and morning; pills are to be discontinued when patient's mouth turns sore and gums spongy. The patients to be advised to wash their mouth frequently and the throat gargled with a fluid mixed with alum, "Mel. Opiat." (honey-based opium compound?), and decoctum corticis Peruviani (decoction of "Peruvian bark": either of *Cinchona succirubra* or of *C. pubescens*) in specific quantities. After the soreness of the mouth has subsided, the pills are resumed; process repeated until symptoms of leprosy disappeared.'

JD talks of administering the 'right' food during the treatment and a mild laxative before treatment. For any externally manifested ulcers ('excoriation', according to JD), *unguentum album camphoratum* (Note 11) was to be applied twice a day. Ulcerated areas are to be cleaned with a lotion of Madeira wine (one part) and the urine of a healthy person (two parts), and dressed two times a day with copaiba balsam (Note 12). In case, copaiba-balsam dressing fails a 'mild' aqueous solution of bluestone ($CuSO_4$) to be used.

van-Someren[5] criticizes the use of the wine–urine solution as unsanitary and comments that the poor patients subjected to frequently repeated courses of mercurialization and ptyalism are hurried to their graves, and JD was vainly endeavouring to cure their disease. van-Someren lists the medications trialled at MLH and not one of them proved a 'real remedy': the Asiatic Pill (arsenic protoxide, powdered root of Calotropis Gigantea and 'some' black pepper), Fowler's solution, Donovan's solution (AsI_3), Hydrocotyle Nigra (Note 13), Chowl Moogree oil and Mercurial Pills.

At least the Asiatic Pill, Fowler's solution, and Donovan's solution included arsenic in one form or another; the Donovan's solution included mercurial compound (HgI_2) in addition to arsenic[8]. Exposure to As in the context of medical treatment has been occurring, for many centuries. In Europe and USA, As compounds such as the Fowler's solution and Asiatic Pill were used to treat various human illnesses[9] until 1965. In India, As has been used along with opium as an aphrodisiac and in herbal remedies[10]. van-Someren's remark – quoted *verbatim* here – is unclear:

> 'One or two of these were inert, and if they did the patient no good, did no harm. Others were positively noxious, acting as irritant poisons, and only served to reduce the strength and powers of resistance to diseases in some of those who were unfortunate enough to be subjected to their operation.'

He does not indicate what he means by 'one or two of these were inert'; are we to guess that the two plant products (*Hydrocotyle nigra* [= *Centella asiatica*] and the oil of *Hydnocarpus wightiana*), which were not blended with any mineral materials were the inert items?

On the inefficiency of different tested medications, Medical Officers J. Lawder, W. G. Davidson, Evans, Paul and Mudge, who were at MLH from 1841, indicate their experiences as follows:

> Lawder (1841): 'I have tried almost all the remedies recommended by the different medical authors, I am sorry to say without any hope of cure; … I have no doubt of its being incurable.'

> Davidson (no date): 'After the intestinal and cutaneous irritation caused with the use of the Asiatic Pill subsided, … the patient appeared to be a good deal better – skin much less unhealthy looking than before these medicines were given, but the improvement did not seem to be very considerable; … There appears to be a general fading of the system.'

> Evans (1847): 'I regret I cannot notify any encouraging improvement in the

leprous disease itself from the medicines employed. In one or two case, however, lately admitted, Hydriodate of Potass seems to have produced at least temporary benefit'.

Paul (1855): 'The therepeutic virtues of the Hydrocotyle and Chowl Moogree received a fair trial at the hands of Dr Porteous, and they were found to produce no amelioration whatever of the disease… . In a few,…, Donovan's Solution in small doses was given for long periods, but I cannot say with marked or material benefit.' He adds that personal hygiene was most beneficial.

Mudge (no date) confirms Surgeon Paul's remark (preceding statement) on temporary benefit from personal hygiene, and reaffirms: '…but beyond this remedial measures are of no avail'.

van-Someren concludes this section (and the 1861 paper) with the following summary remarks:

- Good food, pure air, cleanliness and some bodily exercise contribute more than anything else to improve the health of the patients of leprosy.
- Chalybeate (spring water inundated with iron), preparations of iodine, and cod-liver oil indicate promise as internally administered remedies.
- Sulphur–vapour baths, application of calamine, and water dressings should do good to cure external sores.

He condemns use of mercurial preparations. According to him they reduce RBC levels in the blood of patients. This van-Someren remark is noteworthy considering that today we know more about the impact of mercury and mercurial compounds on human health in general and human blood profiles in particular[11].

Conclusion

Our present understanding of the prognosis of the two forms of leprosy[12] reinforces that in tuberculoid leprosy – the milder form of the disease – the body's immune cells attempt to seal the infection from the rest of the body by isolating the infective bacterium. Because this response by the immune system occurs in the deeper layers of the skin, the hair follicles, sweat glands, and nerves are affected. In the other form, now known as lepromatous leprosy, the human-immune system struggles to cope with the intensity of infection. *Mycobacterium leprae* multiplies liberally in skin. Characteristically large nodules or lesions appear all over the body. Considering the WHO notes[12] on the prognosis of the disease, another van-Someren observation made in 1861 that the prognosis of leprosy was unfavourable and that the anaesthetic form was much more chronic in its progress than the tubercular, although both could last many years, appears prophetic. In terms of medications, today MDT (multi-drug therapy involving the use of a combination of Rifampicin, Clofazimine, and Dapsone for patients of multibacillary leprosy, and Rifampicin and Dapsone for patients of paucibacillary leprosy) is in vogue. Management strategies have indeed changed.

MLH remained opposite to Monégar Choultry until 1921. Freeman Freeman-Thomas (Baron Willingdon), Governor of Madras [1919–1924] closed MLH and shifted it to Chenglepat (12°42′N; 79°58′E) along the outskirts of Madras city in 1921. The Chenglepat facility, then run and managed by a missionary organization, became the Lady Willingdon Leper Settlement. This settlement was taken over by the Government of India (Directorate General of Health Services, Ministry of Health and Family Welfare) and re-named the Central Leprosy Teaching and Research Institute, Chenglepat in 1955 and continues to function.

Notes

1. Henry Vandyke Carter is remembered for his elegant sketches in *Gray's Anatomy* (J. W. Parker & Sons, London, 1958).
2. See Surgeon-General George Bidie's address: 'Geographical Distribution of Diseases in Southern India' at the annual meeting of the South Indian and Madras branches, British Medical Association (*Br. Med. J.*, 1889, **2**(1490), 115).
3. With an MD from Edinburgh, van-Someren joined Madras Medical Service as an assistant surgeon in Bangalore in 1846, became a full surgeon 1st District (Madras) in 1863. He rose to the rank of Deputy Surgeon-General in 1875, retired in 1880.
4. Monégar Choultry (MC) is a corruption of Maniakkārar Çattiram (Tamizh). MC commenced as a charity in Royapuram, off Popham's Broadway, in 1781, consequent to a famine. Because the raw materials for making porridge (kanji, Tamizh) were supplied free here, when MC grew into the Madras-Native Infirmary (MNI) in 1799, it became the kanji-tôtti hospital. MLH (leper asylum) developed in conjunction with MNI. The MNI grew into Royapuram Hospital in early 20th century, and later grew into the Stanley Medical College and Hospital.
5. Two other institutes for PoLs already existed in southern India: (1) The Cochin Lazarus Hospital, Pallipuram, Vypeen Island, Cochin, started by the Dutch in 1728; (2) In Bangalore.
6. James Dalton is remembered in Madras in the context of Madras Madhouse in Purasawalkam, which was started by Valentine Connolly in 1794. Dalton rebuilt this facility, which was known as Dalton's Mad Hospital (1807–1815).
7. Coinage in 16th–17th-century southern India.
8. James Lawder, Medical Officer at MNI and MLH, was keen on restraints on PoLs, to restrict the disease. Lawder preferred a high wall (3 m) around the asylum, whereas the government favoured a less tall wall. Lawder's Gate bus stop in Purasawalkam refers to him.
9. 'Anæsthetic' form – *Lepra anæsthetica*; 'tubercular' form – *Lepra tuberculosa*; italicization as in the original.
10. van-Someren (1861) cites this table without any bibliographic details.
11. Camphorated white ointment; according to the London Pharmacopeia, adding a drachm-and-a-half of camphor with some drops of almond oil to 'white' ointment; according to the Edinburgh Pharmacopeia, a drachm-and-half of camphor to a little oil (almond oil?) to a pound of white ointment.
12. Resinous oil of *Copaifera officinalis* (Fabaceae). Historically used against different skin disorders. Copaiba oil is shown to be antimicrobial in recent times.
13. Plant biological names have been reproduced as such; for extensive notes on chaulmoogra oil, see Parascandola, J., *Pharm. Hist.*, 2003, **45**, 47–57.

1. NLEP (no date). National Leprosy Eradication Programme: history of NLEP; http://nlep.nic.in/about.html, accessed on 18 July 2012.
2. Carter, H. V., Memoirs of the General Department, Bombay Government Gazette # 756, Part III, 14 December 1882, pp. 1002–1170; http://digital.nls.uk/indiapapers/browse/pageturner, accessed on 18 July 2012.
3. Medical History of India, Disease: leprosy, 2007; http://digital.nls.uk/indiapapers/leprosy.html, accessed on 18 July 2012.
4. Hansen, G. H. A., *Norsk. Mag. Lægev.*, 1874, **4**, 1–88.

5. van-Someren, W. J., *Madras Q. J. Med. Sci*., 1861, **3**, 271–294.
6. Mudaliar, A. L., In *The Madras Tercentenary Commemoration Volume* (ed. Madras Tercentenary Volume Editorial Committee), Madras Tercentenary Celebration Committee, Madras, 1939, pp. 51–61.
7. Buckingham, J., *Leprosy in Colonial South India: Medicine and Confinement*. Palgrave Macmillan, London, 2002, p. 276.
8. Piffard, H. G., *A Treatise on the Materia Medica and Therapeutics of the Skin*, William Wood & Company, New York, 1881, p. 351.
9. Rossy, K. M., Janusz, C. A. and Schwartz, R. A., *Indian J. Dermatol. Venereol. Lepr*., 2005, **71**, 230–235.
10. Datta, D. V., *Lancet*, 1977, **1**, 484.
11. Hyman, M., *Altern. Ther*., 2004, **10**, 70–75.
12. WHO, http://www.who.int/topics/leprosy/en/, accessed on 2 August 2012.

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