Lepr Rev (1985) **56,** 153–162

SPECIAL ARTICLE

Leprosy and procreation—a historical review of social and clinical aspects

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Accepted for publication 2 October 1984

The clinical and immunological aspects of interactions of leprosy and pregnancy in terms of relapse, reactivation, exacerbation of the disease and leprosy reactions have recently received considerable attention.[1] Furthermore, the development of techniques to demonstrate IgA and IgM anti-*Mycobacterium leprae* antibody activity in cord sera, presumptive evidence for the transmission of live *M. leprae* bacilli across the placenta[2] and the development of leprosy in very young children[3] have suggested that the incubation period of *M. leprae* may be very much shorter than hitherto acknowledged.

Further detailed but limited studies will be necessary to investigate further some of the findings.[4–6] Nevertheless it has been shown that due to maternal immunosuppression occurring during pregnancy, the mother's leprosy may become overt, relapse if cured, or deteriorate during pregnancy and puerperium, with increased likelihood of her developing erythema nodosum leprosum (ENL), especially during pregnancy and lactation, and reversal reaction during lactation, with the development in addition of progressive nerve damage with sensory and motor loss.

At the same time the child born to the mother with leprosy has a lower birth weight, a slower growth rate, an increased susceptibility to infection and a higher death rate under the age of 1 year than the child of a healthy mother. These are all most marked in the children of mothers with lepromatous leprosy.

The mother with leprosy, particularly lepromatous leprosy, living in a developing country without any form of social security, knows that she will be ultimately dependent upon her children to care for her, either in sickness or in her old age, and therefore has to produce and rear sufficient healthy children for this purpose. Because of increased infant mortality, in particular neonatal loss, she has to undergo more pregnancies than her healthy counterpart, to ensure live children and thus, although she realizes the risk of pregnancy, she has to undergo the risk of pregnancy time and again. Moreover, in time as she develops dapsone resistance, there is the ever present risk of her transmitting dapsone-resistant leprosy to her child—a further complicating factor in the battle to control leprosy.

In the light of new knowledge and understanding of leprosy, and advances in reproductive immunology, it is of great interest to review historical records and theories of causation and transmission of disease, and in particular the role of procreation.

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Earliest historical records

The earliest indubitable references to leprosy come from India and go back to the sixth century BC.[7] Earliest descriptions of the disease from India and China are surprisingly accurate, and clearly describe the disease which is known today as leprosy which is caused by *Mycobacterium leprae*. Both skin and nerve signs of the disease were recognized and chaulmoogra oil was mentioned as a treatment.[8] It was likely that leprosy was brought to Europe and the Mediterranean basin by the armies of Alexander the Great after his Indian campaign, 327–326 BC. Thus leprosy referred to in the Bible in the days of Jesus Christ may well have been 'modern' leprosy.

The Hebrew word translated as leprosy comes from the same root as 'stricken by God'. Thus leprosy came to be regarded as a curse or judgement by God, as seen in the story of Miriam, Moses' sister[9] and Gehazi, Elisha's servant.[10] Furthermore, while 'leprosy' referred to in the Old Testament is unlikely to have been what we understand as leprosy, but rather a collection of infectious diseases and conditions affecting the skin, clothing of wool, linen and leather, and the walls of dwellings,[11] Biblical references to leprosy[12] have done much to instil the idea of uncleanliness, incurability and communicability of the disease.

That leprosy was regarded as an infectious disease and was treated as such is clear from events of history. The spread of leprosy along the routes of invading, and retreating armies, and along trade routes by land and sea; the epidemic of leprosy coinciding with the return of the Crusaders; the establishment of leper hospitals outside the towns, the laws forbidding lepers entry to towns, are all evidence. Guy de Chauliac, practising in the fourteenth century not only provided an unequivocal description of leprosy but gave, in great detail, instructions regarding the examination of one suspected of having leprosy—as he observed:

'. . . in the examination and judgement of lepers there must be much circumspection, because the injury is very great, whether we thus submit to confinement those that ought not to be confined, or allow lepers to mix with people, seeing the disease is contagious and infectious.'[13]

Cullen (1772) in his 'Nosology' described the disease as contagious,[14] while Schillingii (1778) stated that leprosy could be transmitted by the pus from abscesses, also by respiration and from a leprous wet nurse to a suckling infant.[15]

Causation and transmission of leprosy

From earliest records 2 themes run side-by-side: leprosy was highly infectious and leprosy could result from incurring the anger of supernatural powers as has been referred to earlier and as is seen in the story of Troylus and Cresseid.[16] Cresseid attributed her misfortunes in love to the fickleness and pranks of the gods and, thus, incurred their wrath:

'Thy cristall ene minglit with blude I mak,
Thy voice sae clear, unpleasand hoar and hace,
Thy lustie lyre owerspred with spottis blak,
And lumpis haw appearand in thy face.
Where thou cumis, ilk man sall flee the place.
Thus sall thou go begging frae hous to hous
With cup and clapper like ane Lazarous.'
. . .
'Therefore in secreit wyse ye let me gang
Into yone hospitall at the tounis end.'
. . .
'Then in ane mantill and ane baver hat,
With cup and clapper wonder privily,

He opnit ane secreit yett, and out thereat
Convoyit her, that nae man suld espy,
Into ane village half ane mile thereby,
Deliverit her in at the spitail hous,
And daylie sent her part of his almous.'

(Henrysoun, c. 1420–1490)

In Judaio–Christian circles it was generally understood that leprosy was God's judgement for sin[17] and that this judgement could be extended to the third and fourth generation.[18] However, this thinking was not confined to the centres of Judaio–Christian teaching, as in Chinese tradition leprosy was regarded as punishment for sexual misdemeanour and was transmissible within the family to the third and fourth generations:[19–22] the children born of the fourth generation were considered healthy and could return to society.[23]

Sexual transmission of leprosy

The idea that leprosy was transmitted as a venereal disease was prevalent in England in the Middle Ages, and prompted some of the rules of the leper houses[24]. As Donne recorded:

'By thee the silly amorous sucks his death [sic: seely]
By drawing in a leprous harlot's breath.'

(Donne, 1573–1631)[25]

Richter[26] maintained that leprosy was transmitted by sexual contact. In China, women with leprosy believed they could be cured of their disease if they had sexual contact with a healthy male, thus transferring the disease to him. Thus, the practice of 'selling leprosy' was developed. The dread of this scourge exerted a great influence on promiscuous intercourse in China and on the general moral conduct of the people.[19, 21] In Mysore, India, it was also a common belief that leprosy was a form of venereal disease.[27]

After the advent of syphilis to Europe in the sixteenth century there was undoubtedly some confusion between the clinical features of syphilis and leprosy caused in part by the similarity of the clinical picture of ulcers, sores, lymphadenopathy and destruction of nasal bones: thus the term 'syphilitic leprosy' came into use. The observation that syphilis is transmitted by sexual contact may have given additional support to the theory of sexual transmission of leprosy.

Hereditary transmission of leprosy versus spread by contagion

The Greek and Arabian physicians had a universal belief in hereditary transmission of leprosy as they maintained 'all body fluids (including semen) were affected'.[28] It is likely that such a belief influenced laws regarding marriage and divorce in Europe dating from the seventh century, and the practice in Europe, of the castration of lepers and the burial alive of the leprous mother and child. The theory of hereditary transmission of leprosy was held by many physicians until the end of the nineteenth century.

The strongest case for hereditary transmission of leprosy seems to be that of the leprosy victims in the Shetland Islands, who in the eighteenth century were all from a few families.[29] However, if hereditary transmission was so important, one is faced with the question as to why leprosy in the Shetlands died out when the victims of the disease were effectively isolated on the island of Papastour.

Amongst the Punjabees generally, the belief in hereditary transmission of leprosy was so deeply grounded that they were in the habit of burying alive not only the leper himself, but also his relations and friends lest in multiplying their kind, the disease would be communicated to distant generations.[30]

In Iceland leprosy was considered to be both an inheritable and a very infectious disease.[31] The Lolos, natives of Szechuan province of China, the Siamese and Javanese all believed in hereditary transmission of leprosy.[32] The Annamites (amongst whom leprosy was very prevalent) declared 'leper parents always gave birth to leper children although, on the other hand, the malady does not declare itself before the tenth, eleventh or twentieth year.'[33]

The theory of hereditary transmission was not universally held—even Danielssen and Boeck who were the chief protagonists of this theory virtually denying the possibility of leprosy being contagious, stated that a few cases of leprosy could occur spontaneously.[34]

Schillingii in 1778 had observed that leprous parents could give birth to healthy babies but such babies could not remain free of leprosy unless they were separated from their parents at the time of birth and brought up in a healthy environment, with wholesome feeding.[35] This was supported nearly a hundred years later by Leloir who maintained that children separated from leprous parents early had a very good chance of escaping leprosy, especially if they were sent to a non-infected district, but if they remained with their parents there was very slight chance of escape.[36] Adams had maintained that as the reproductive capacity of both men and women was impaired, leprosy should be a self-limiting disease if hereditary transmission was so important.[37]

The dispute regarding hereditary transmission and contagion in the spread of leprosy raged throughout the nineteenth century.

The case for hereditary predisposition to the disease rather than hereditary transmission of leprosy was put forward by Hjaltelin[38] and supported by Kierulf who noted that the spontaneous development of the disease was always in endemic areas and never where it was unknown.[39] The observation that 11 parents of children with leprosy developed the disease *after* the birth of the children was made by Holmsen[40] who, along with Kierulf, believed in the existence of a specific virus for leprosy.

Drognat-Landré (1868) however was the first to seriously analyse the problem, prompted by seeing, in Holland, 10 Dutch patients with leprosy contracted in the colonies. After making observations on leprosy in the native and ex-patriot communities, and noting the appearance of leprosy in children of leprous parents, in a critical review of the history of the disease in Surinam, Drognat-Landré concluded that contagion was the sole means of propagation of leprosy.[41] His monograph, published at a time when leprologists in many countries were in hot pursuit of the 'virus of leprosy', was eclipsed by Hansen's discovery of the bacillus of leprosy and fell into obscurity for 70 years.[42]

Further support for hereditary predisposition to leprosy was given by Tache and Roose. Roose, who had observed the occurrence of leprosy in 4 children aged 4 to 6 years felt that these children, who possibly had inherited a predisposition to leprosy, had been infected by their leprous parents after birth by contagion.[43] However, Impey considered that there was no proof of hereditary transmission of leprosy and that hereditary predisposition occurred only in a small number of cases,[44] and Choksy in Bombay, observed that only 5% of leprosy was due to hereditary transmission, although 11% had a family trait.[45]

Hansen and Looft, who argued against the hereditary transmission theory on the grounds that the bacillus is a parasite and not a heredity factor with anatomical and physiological peculiarities, suggested that the term 'hereditary transmission' be replaced by 'hypothesis of latent infection'.[46]

It is of interest that the question 'Is the contagion of leprosy transferable by way of intra uterine infection?' was asked nearly 100 years ago[47] and that this question is only now being answered.

Social attitudes and laws in relation to leprosy, marriage, divorce and procreation

In the seventh century, Rothan, King of Lombards, made laws to prevent marriage of lepers.[48] A hundred years later, in 757 the parliament of Pepin, King of France, passed a law in which leprosy

was regarded as a cause of separation, thus the healthy partner of the marriage was allowed to remarry.[49] In 789 Charlemagne proclaimed laws forbidding the marriage of lepers.[50] A similar edict was made about 950 by the Welsh King Hoel Dha; however, at this time the term 'leprosy' covered various skin diseases.[49] In 1186, Pope Urban III allowed that subsequent leprosy was a sufficient reason why a betrothed couple should not be compelled to marry.[51]

Scots law, prior to the days of King Malcolm Canmore, in its practice of hygienic measures to control disease, ordered castration of epileptics, the insane or carriers of diseases transmissable from father to son; at the same time, to prevent the spread of leprosy, it banished any woman sufferer from the company of men, with the penalty of burial alive with her child, should she give birth whilst suffering from leprosy.[52] The practice of castration of lepers was apparently widely practised in the Middle Ages.[53]

Segregation of lepers from healthy persons in the Middle Ages was followed by separation of the sexes as evidenced by the rules of leper houses. Rules for St Julien's Hospital, thirteenth century, stated that those admitted were to be single: if they were married they were to part by consent and vow chastity.[24] Sometimes the lepers' wives lived with them, as was the case at the Edinburgh Greenside Hospital in 1591, where to enforce complete segregation of the lepers, one of the wives was allowed to go out to the market while the lepers took it in turns to sit and beg alms at the hospital door.[54]

In France in 1757, leprosy was a valid cause for divorce. In Britain at the same time there were regional laws which forbade cohabitation if either husband or wife were a leper: a leper in these circumstances being considered as dead.[50, 55] Icelandic law in 1776 forbade the marriage of lepers,[56] while Norwegian law in 1781 allowed divorce of lepers and remarriage of the healthy partner.[57] In 1790 in Norway, a second law was passed allowing husbands whose wives were placed in the leper hospital at Bergen, to remarry, the woman declared to be civilly dead.[50] In Crete, in 1874, the Bishop found it necessary to recommend to the priests not to sanction marriages with or among lepers.[50]

In China leprosy was regarded as legal grounds for annulment of promise of marriage contract[58] or divorce.[59] In the Kwantung province in China, in the nineteenth century, in arranging child betrothals, great care was taken in ascertaining the absence of leper trait in the other party: despite this the Chinese were willing to hire lepers to care for their children! In Cochin China, where leprosy was very prevalent, the Annamite leper did not remarry: if leprosy declared itself after marriage, the husband avoided his wife's bed for fear of giving her the disease.[60] In the Canton Province of China, marriage between lepers was only permitted with those having the same type or grade of the disease.[61]

In the nineteenth century, regulations regarding marriage within leper asylums varied considerably. On the one hand in South Africa, where leprosy was considered to be spread by contagion rather than hereditary transmission, conjugal intercourse was discouraged between lepers until they were past child-bearing age, and was not permitted at all between lepers and healthy persons.[44] On the other hand, in India, where marriages amongst lepers were not prolific[45] we find that marriage was permitted for mutual care rather than enjoyment of sexual relations.[62] It was observed that of 1600 inmates of Matunga leper asylum, Bombay, in 9 years only 7 children were born. A similar observation was made in Hawaii where in a colony of 2864 lepers, only 26 children were born,[62] and in the Maracaibo Island leper colony where marriage was permitted, only 2 children were born in 15 years.[45]

Reduced fertility amongst lepers, however, was not always the rule. In Indo-China, where the birth rate amongst lepers was high and hereditary transmission of leprosy was considered most important, a strong case was made for sterilization of leprosy patients of both sexes.[53] In Panama, at the Palo Seco asylum, marriages of lepers were allowed only after sterilization of the male on his written request.[63]

In Korea, the segregation of sexes practised in leper hospitals resulted not only in sexual perversion, but also in patients leaving the leper hospital. Such patients formed, in some cases, transient attachments with those of the opposite sex and joined leper camps—children born in such

circumstances not only had a precarious home life, but if they remained with their parents, half of them became infected with leprosy. A system of arranged marriages and arranged adoptions (in accordance with local customs) together with voluntary sterilization was found to be effective in providing for the needs of segregated lepers.[64]

Leprosy in relation to childbearing

In the pre-sulphone era leprosy was associated with subfertility, if not frank infertility.[62, 65, 66] This was attributed to frigidity,[67] and to 'decreasing sexual instinct' with progression of the disease.[68] Testicular atrophy had been observed[37, 65] and also azoospermia due to destruction of the testicle by scarring with connective tissue.[69]

Leprosy occurring prior to puberty resulted in primary amenorrhoea,[70] while leprosy occurring after puberty was observed to cause menstrual irregularity progressing to secondary amenorrhoea.[65, 70, 71] Secondary amenorrhoea was attributed to infection of the fallopian tubes, ovaries or uterus, based on observations of 2 autopsies out of 17 in which tubercles were seen on these organs.[72] However, as a number of those patients also had tuberculosis, one wonders whether the tubercles seen were lesions of tuberculosis rather than leprosy.

In 1897, Zambaco recorded a large number of abortions in women with leprosy. These he attributed to septicaemia with *M. leprae* resulting in placental infection leading to foetal infection and subsequently, abortion. He suggested that these abortions had not been diagnosed as due to foetal leprosy because as happens in many cases of abortion, both foetus and placenta had been discarded without being properly examined,[67] an observation repeated 30 years later by Montero.[73]

It was generally thought, and taught, that the few full-term pregnancies which *did* occur were observed to be normal, with most babies appearing healthy at birth and with no gross abnormality of the placenta.[66] It is therefore of particular interest that Zambaco observed that many of the children born to women with leprosy were remarkably small for the period of gestation.[74]

The adverse effect of pregnancy on leprosy appears to have been first observed by Zambaco.[75] He described 4 women who developed overt leprosy in association with pregnancy, 3 immediately postpartum and 1 during the third trimestre. He observed reaction in the skin occurring in 2 cases postpartum until 3 months of lactation, and in 1 patient he observed silent neuritis with the sudden development postpartum of 'main en griffe' in 2 successive pregnancies in 1 patient. His description is worth translating and runs thus:

'. . . the influence of pregnancy is disastrous for women with leprosy. The disease reveals itself for the first time and progresses rapidly. The birth of the baby in turn affects the development of the illness, in all comparable to the development of tuberculosis in the same circumstances.'[76]

Puberty was seen to affect the incidence of leprosy with a sudden increase in the number of girls in proportion to boys.[77–79] The appearance of overt leprosy in association with pregnancy or exacerbation of the existing disease was noted,[73, 80–83] and deterioration of leprosy patients was observed to be more frequent in those who were not receiving treatment (18 out of 23) than in those who were on treatment with sulphones (5 out of 23).[84]

Although exacerbation of leprosy was seen during pregnancy it was said to be much more common during the puerperium and the early months of lactation.[85] Reaction due to leprosy was observed postpartum,[80] while 1 out of 6 women with leprosy suffered from 'lepra fever' during pregnancy.[77]

Most of the observations on the association of leprosy and pregnancy and the effect of one on the other, are contained in individual case reports or relatively small retrospective surveys. No record of any clearly-defined prospective study could be traced in the available world literature prior to 1975.

The outcome of pregnancies in mothers with leprosy

While considerable research has been carried out on the children of parents with leprosy, this has been largely of an epidemiological nature to determine the susceptibility of these children to leprosy, and to investigate methods of preventing the infection, such as separation of children from parents at different ages after birth. Such studies were carried out in the Philippines, in India and in South America. More recently, massive immunization campaigns have been conducted to test the efficiency of BCG in preventing leprosy in children in leprosy endemic areas.[86, 87]

Relatively little has been recorded of the general state of health of the children of mothers with leprosy, although Zambaco recorded that many children born to mothers with lepromatous leprosy were small 'like an abortion at term', and that they died within a few months of birth of athrepsie.[67] In another context he wrote (here I translate):

'in former times children of leprous women, born like little old men, did not develop normally but died of athrepsie without showing in their body any sign of leprosy. This foetal cachexia which leads to death *in utero* or shortly after birth, without the lesions of leprosy is certainly due to leprosy and maybe described under the name of paraleprosy'.[88]

More recently, it was noted that whereas minor ailments were similar to those seen in the healthy community, among the children of lepers, skin diseases were very common and the infant mortality was very high, with 42% of the children dying of infections, debility, marasmus and athrepsie.[89] Similar observations have been made since then:

'. . . the children . . . are of a reduced vitality and frequently succumb shortly after birth',[90] and '. . . all children of leper parentage are very delicate and seem to have a predisposition to respiratory and gastric diseases which take a big toll of them'.[91]

The low birth weights, small placentae, and low placental coefficients in pregnancies of mothers with leprosy, most marked in those with lepromatous leprosy, were only recorded very recently.[92] As the majority of perinatal deaths occur in the small group of intra-uterine growth retarded infants born to non-leprous women,[93] it is tempting to assume a cause and effect regarding the low birth weight and increased mortality of children of mothers with lepromatous leprosy.

The future

While historical review and recent clinical research has indicated the nature of some of the problems of the interaction of leprosy in pregnancy, a number of investigations are still required to elucidate questions raised in recent studies.

A prospective trial of additional chemotherapeutic agents to prevent relapse in pregnancy while at the same time measuring the dapsone excretion in urine could be used to evaluate the question 'Is patient compliance more important than maternal immunosuppression during pregnancy in the causation of relapse/dapsone resistance?' A prospective clinico-pathological study of ENL in women in relation to the menstrual cycle and pregnancy/lactation could well elucidate the immunological trigger mechanisms in ENL. The high incidence of neuritis in BL patients, and the clinically puzzling mixture of ENL and reversal reaction in this group of patients also merit investigation as a prospective clinico-pathological study with frequent nerve and skin biopsy in addition to immunological tests.

Certain aspects of the effect of leprosy on pregnancy require further study in themselves. One is the hypothesis that impaired placentation in women with lepromatous leprosy is due to their impaired cell-mediated immunity. Another is the observation that patients receiving dapsone, rifampicin and clofazimine have impaired foeto-placental function in terms of oestrogen excretion. This is most marked in patients with lepromatous leprosy, and therefore could be related to the patient's lowered immune responsiveness rather than her drug therapy.

Further immunohistological studies of placentae from lepromatous women are also required using specific antisera to see whether and where *M. leprae*/*M. leprae* antigen lodge in relation to maternal and foetal circulations. Maximum information would be obtained from studying placentae from untreated lepromatous women reporting for the first time late in pregnancy.

A longitudinal study is necessary to assess the effects of the mother's leprosy on her child. Two separate issues require investigation, firstly the development of leprosy in the child, which children develop leprosy and why; and, in those who do not develop leprosy, whether hypersensitivity can be equated with protective immunity. Secondly, whether transfer of maternal cells across the placenta or through the breast milk after birth has any role in the causation of failure to thrive, and whether improving nutritional factors after birth results in improved infant growth. Also, whether immune complexes such as are found in patients with lepromatous leprosy cross the placenta from mother to child, or form in the foetus/neonate, causing intra-uterine growth retardation and failure to thrive after birth, as has been suggested in other infections.

Clearly, the study reported from Ethiopia is not 'an end' but 'a beginning'.

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