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The relationship between detection delay and impairment in leprosy control: a comparison of patient cohorts from Bangladesh and Ethiopia

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Summary

Introduction It is acknowledged that longer delays between first symptoms and diagnosis result in increased impairment in newly detected leprosy patients. However, it is unclear whether detection delay in relation to impairment can be used as a general or absolute performance indicator of leprosy control programmes. It is unknown whether similar delays always result in similar proportions of impairment. Therefore, the present study examined the quantitative relationship between delay and impairment in two different patient populations.

Methods Patients from two study cohorts (BANDS and AMFES) who reported voluntarily were included in the analysis. Data on detection delay, WHO impairment status, type of leprosy, age and sex were analysed using descriptive statistics and multivariate logistic regression analysis to identify significant risk factors for impairment and to quantify the relationship between detection delay and impairment status at intake.

Results Detection delay was an independent risk factor for impairment at presentation in multivariate analysis. The AMFES cohort reported more impairment at detection than BANDS. In multivariate analysis, this difference was significant among PB patients (51% in AMFES versus 15% in BANDS), but not in MB patients (56% in AMFES versus 45% in BANDS). In fact, for every delay category PB patients from AMFES had much higher proportions of impairment than PB BANDS patients. Impairment rates in MB patients from AMFES were higher in every delay category, but the differences between the two cohorts were much smaller compared to PB patients.

Conclusions Our analysis confirms earlier findings that with longer delays, the risk of impairment at presentation increases. With the same reported delay, however, the proportion impaired can vary considerably between different patient populations, in particular for PB leprosy. Delay can therefore not simply be used as a general or absolute performance indicator for programme evaluation. Achieving short delays remains important in general, but understanding and addressing the underlying mechanisms of delay specific to a patient population adds substantially to the effectiveness of leprosy control.

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Introduction

Leprosy is a disease especially known and feared because of the nerve function impairment and disabilities that can result. Time plays an important role in the development of leprosy and of nerve damage in particular. Therefore, early detection and treatment of patients is the main focus of leprosy control programmes. The quality of such programmes can be monitored and evaluated with specific indicators. These are tools for measuring the magnitude of the leprosy problem and progress towards achieving the objectives of the programme. Because the leprosy situation differs between countries, programme quality targets should be country-specific and based on recent trends.[1] One indicator of interest in leprosy control is detection delay in relation to the proportion of patients with impairment. Detection delay is defined as the time between noticing the first symptoms and the diagnosis of leprosy. Several studies (e.g. [2–5]) have investigated the relationship between detection delay and the impairment status of leprosy patients at the time of detection. All of these studies found that with longer delays the risk of impairment increases. However, the studies report different impairment rates when the reported delays are the same. For instance, data from Ethiopia showed that 36% of the patients with a delay of 0–1 years presented with impairment, while this proportion was only 12% in Bangladesh.[2,3]Nicholls *et al.* suggested that a threshold defining early presentation (e.g. less than 6 months) could be used as an indicator for good practice in leprosy control.[3] From the data, however, it is unclear whether the relationship between delay and impairment is the same in all situations. For example, is it true that with a delay of less then one year the impairment rate is always 15%? The question thus arises if this relationship is in general valid.

The present study was carried out to investigate differences in impairment status at the time of detection by comparing two patient populations from different countries. The question posed is whether detection delay in relation to impairment is a generally applicable performance indicator of the quality of leprosy control programmes in terms of reliability and consistency. The overall aim is to obtain a better understanding of the relationship between delay and impairment and the role of early detection in leprosy control.

Materials and methods

PATIENT POPULATIONS

For the analysis, two cohorts consisting of newly detected leprosy patients were compared. One cohort was from The Bangladesh Acute Nerve Damage Study (BANDS). This prospective cohort study was supervised by the Danish Bangladesh Leprosy Mission, which runs a well-developed vertical leprosy control programme. The BANDS cohort included 2664 patients newly registered for multi-drug therapy (MDT). Enrolment of patients was from April 1995 to March 1996.[6] Patient detection was either active or passive. Of the new patients, 43% reported voluntarily (passive detection).[3] Full details of the research design and methodology of BANDS can be found in an earlier paper.[6]

The other cohort was part of the ALERT MDT Field Evaluation Study (AMFES). The project was carried out in a selected area within the vertical leprosy control programme of ALERT in Central Ethiopia. The study design was a long-term prospective cohort study recruiting new, untreated patients in the period from April 1988 to June 1992.[7]The AMFES

cohort included 592 newly registered patients of whom most reported voluntarily (92%), reflecting the passive nature of case-finding in ALERT's leprosy control programme.[2] An earlier publication describes the design and methods of AMFES in more details.[7]

PATIENT DATA

For the present comparative analysis, only patients who reported voluntarily (passive case detection) were included. Patients with non-classifiable or missing data on one of the variables of interest were excluded from the analysis. These variables were sex, age, leprosy classification, bacterial index, detection delay and WHO impairment status.

The variable age was defined as age at registration in years and divided into five subgroups, following Meima *et al*.[2] These age groups were 0–14 years, 15–29 years, 30–44 years, 45–59 years and 60 years and over.

Ridley–Jopling classification and skin smear results were available at intake for both cohorts. To enable comparison of the cohorts, these data were used to redefine PB and MB. PB patients were those classified as indeterminate (I), TT, or BT and having a negative skin smear. All BB, BL and LL classified patients and all patients with a positive skin smear were defined as MB.[7]The definition of detection delay as described in the two projects differed. BANDS described detection delay as 'duration of symptoms at registration'. The estimate of the patient was cross-checked against significant data, such as family, local, religious or national events. Delay was recorded in months and years with delays up to 1 year in months and delays of more than 1 year primarily in years.[3] In AMFES detection, delay was calculated from the mid-year of the calendar year in which the patient had noticed the first symptoms and the registration date.[2] The present study used the delay categories as defined by Meima *et al.*[2]These were: up to 2 year, between 1 and 2 years, between 2 and 4 years, and more than 4 years. The BANDS programme recorded delay mostly in rounded years and included 1 year in the delay category 0–1 years, 2 years in the delay category 1–2 years, etc.[3]

Impairment status was assessed with the WHO disability grading system (grades 0, 1, 2), in this paper referred to by the more accurate term 'WHO impairment grades' or 'WHO impairment status'.[8]

DATA ANALYSIS

The data analysis was aimed at quantifying the relationship between detection delay and impairment status at intake in two different patient cohorts. For this aim, baseline characteristics of both cohorts were described. Further, logistic regression was conducted to examine whether the variables sex, age, leprosy classification and detection delay were risk factors for impairment at intake. To test the significance of each of these risk factors, odds ratios were calculated with 95% confidence intervals. A multivariate logistic regression model was used to test the significance of a risk factor independent of the other risk factors in the model. The multivariate analysis was carried out using a model with all risk factors included simultaneously. For comparison between the two cohorts, these regression analyses were done for both cohorts separately and for the combined cohort with respect to leprosy type (PB or MB). The statistical programme SPSS was used for the analysis.

Results

PATIENT CHARACTERISTICS

To enable comparison, only patients who reported voluntarily (passive case detection) were included. In the BANDS cohort, 1133 out of 2664 patients (43%) reported voluntarily, while the AMFES cohort had 538 out of 586 self-reported patients (91%). From these 1671 passively detected patients, 77 patients were excluded from the analysis. These were patients without data on bacterial index ($n = 17$) or delay ($n = 5$). Neural leprosy (NL) patients were not classifiable as either PB or MB patients and therefore also excluded from the analysis ($n = 55$). Thus a total of 1594 newly registered patients from both AMFES and BANDS cohorts were available for analysis. Table 1 shows characteristics of the patients at intake according to cohort.

Sex and age distribution were comparable between the two projects. Differences were observed in leprosy type, impairment status at intake and detection delay. The distribution of PB and MB patients was almost equal (48 versus 52%) in the AMFES cohort, while the BANDS cohort had 90% PB patients and only 10% MB patients. Impairment at intake was 3 times higher in the AMFES cohort compared with the BANDS cohort (54 versus 18%). With respect to delay, patients in the BANDS cohort presented earlier than patients from the AMFES cohort (57 versus 26% delay up to 1 year).

Table 1. Characteristics of new patients at intake by cohort

	AMFES ($n = 517$)		BANDS ($n = 1077$)	
Characteristic	no. cases	% of all cases	no. cases	% of all cases
Sex				
Male	325	63	672	62
Female	192	37	405	38
Age in years				
0–14	72	14	182	17
15–29	214	41	313	29
30–44	110	21	320	30
45–59	83	16	184	17
60+	38	7	78	7
Type				
PB	248	48	974	90
MB	269	52	103	10
Impairment status				
Grade 0	240	46	882	82
Grade 1	158	31	115	11
Grade 2	119	23	80	7
Delay in years				
0–1	136	26	613	57
1–2	161	31	218	20
2–4	144	28	121	11
4 +	76	15	125	12

ANALYSIS OF RISK FACTORS FOR PRESENTATION WITH IMPAIRMENT

To examine the differences in impairment at intake, regression analysis was performed for the combined cohort and for each cohort alone. Outcomes from univariate analysis were almost similar to the multivariate ones. Therefore, only the multivariate results are given in Table 2.

Table 2a shows the results for the combined cohort. From this table, it can be seen that a significantly higher proportion of impaired PB patients was found in Ethiopia than in Bangladesh (51 versus 15%), but this was not the case for MB patients (56 versus 45%). Differences in impairment rates between the two cohorts were mainly observed in PB patients. Sex was not an independent risk factor for presenting with impairment. Only among PB patients, females had a significantly lower risk of impairment than males, but the difference was not very strong ($P = 0{\cdot}03$). In both cohorts, higher age and longer delays were strongly associated with an increased risk of impairment at intake.

When examining the cohorts separately, some differences were observed. Sex was only significant in the PB BANDS cohort ($P = 0{\cdot}02$). Overall, age was an independent risk factor in both cohorts, but less significant for MB patients. Among PB patients, the influence of age was more marked in the AMFES compared with the BANDS cohort. Increasing delays were significantly associated with impairment, except among MB BANDS cohort patients. The effects of delay were much stronger in the PB AMFES cohort compared to the PB BANDS cohort. Not only did PB patients in the AMFES cohort have more impairment in each delay category, the increase in impairment rate with longer delays was much larger. The relationship between impairment at intake and delay are illustrated in Figure 1.

The two MB lines, one for each cohort, have different shapes. The BANDS curve starts with a higher proportion of impairment than that for AMFES, but remains at a constant level for delay up to 4 years. In contrast, the AMFES cohort shows a gradually increasing line, reflecting higher impairment rates with longer delays. When delays become longer than 4 years, the BANDS cohort shows a rapid increase in the proportion impaired. At this point, the impairment rates are very comparable with the AMFES cohort having 81% impairment and the BANDS cohort reporting 70% impairment.

The PB curves of both cohorts also show different patterns. The AMFES curve starts with a higher proportion of impairment and suddenly becomes steeper at a delay of more than two years. For the BANDS cohort, the line starts with a relatively low impairment level and remains quite flat with only a slight increase when delays become longer. When delays are 4 years or more, the proportion with impairment is more than three times higher in the AMFES compared with the BANDS cohort (76 versus 23%).

The differences in impairment rate are more pronounced in the PB group, as can be observed from the larger distance between the two PB curves compared to the MB curves.

ANALYSIS OF POSSIBLE CONFOUNDERS

The relationship between delay and impairment was studied in more detail by checking for confounding variables. Sex and age had almost similar distributions in both cohorts. Although the distribution of PB and MB patients was different in AMFES and BANDS cohorts, the proportions of patients according to the Ridley–Jopling classification were nearly the same in the two cohorts.

The case-finding methods differed in both cohorts. In the AMFES cohort, almost all patients reported voluntarily (91%), while in the BANDS cohort only 43% of all patients were detected passively. From the BANDS data, it could be seen that in both the PB and MB

Table 2. Multivariate logistic regression odds ratios and 95% confidence intervals of risk factors for impairment at intake based on PB AMFES-BANDS cohort and MB AMFES-BANDS cohort

Risk factor	No. impaired (% of cases within subgroup)	Multivariate odds ratio (95% confidence interval)	No. impaired (% of cases within subgroup)	Multivariate odds ratio (95% confidence interval)
(a) AMFES + BANDS	PB ($n = 1222$)		MB ($n = 372$)	
Sex				
Male	182/738 (25)	Baseline[1]	140/259 (54)	Baseline[1]
Female	94/484 (19)	0·70 (0·51–0·96)[2]	56/113 (50)	0·87 (0·53–1·42)
Age in years				
0–14	22/214 (10)	0·60 (0·34–1·05)	8/40 (20)	0·29 (0·12–0·70)[2]
15–29	66/383 (17)	Baseline[1]	68/144 (47)	Baseline[1]
30–44	77/324 (24)	2·12 (1·40–3·22)[2]	66/106 (62)	1·86 (1·08–3·22)[2]
45–59	74/207 (36)	3·44 (2·23–5·33)[2]	37/60 (62)	1·76 (0·92–3·38)
60+	37/94 (39)	3·52 (2·04–6·07)[2]	17/22 (77)	3·62 (1·20–10·90)[2]
Delay in years				
0–1	92/636 (15)	Baseline[1]	39/113 (35)	Baseline[1]
1–2	68/273 (25)	1·41 (0·95–2·07)	55/106 (52)	1·98 (1·10–3·56)[2]
2–4	65/165 (39)	2·35 (1·53–3·60)[2]	60/100 (60)	2·70 (1·47–4·97)[2]
4 +	51/148 (35)	2·38 (1·53–3·69)[2]	42/53 (79)	6·23 (2·76–14·05)[2]
Cohort				
BANDS	149/974 (15)	Baseline[1]	46/103 (45)	Baseline[1]
AMFES	127/248 (51)	6·73 (4·76–9·51)[2]	150/269 (56)	1·25 (0·73–2·14)
(b) AMFES	PB ($n = 248$)		MB ($n = 269$)	
Sex				
Male	79/146 (54)	Baseline[1]	104/179 (58)	Baseline[1]
Female	48/102 (47)	0·95 (0·52–1·71)	46/90 (51)	0·82 (0·46–1·44)
Age in years				
0–14	13/46 (28)	0·58 (0·25–1·30)	6/26 (23)	0·30 (0·11–0·85)[2]
15–29	37/96 (39)	Baseline[1]	60/118 (51)	Baseline[1]
30–44	28/42 (67)	2·40 (1·06–5·46)[2]	44/68 (65)	1·46 (0·76–2·81)
45–59	31/41 (76)	5·25 (2·23–12·39)[2]	28/42 (67)	1·57 (0·72–3·42)
60+	18/23 (78)	5·73 (1·84–17·85)[2]	12/15 (80)	4·14 (1·02–16·80)[2]
Delay in years				
0–1	30/78 (39)	Baseline[1]	16/58 (28)	Baseline[1]
1–2	31/79 (39)	1·22 (0·60–2·48)	45/82 (55)	3·05 (1·43–6·50)[2]
2–4	41/58 (71)	3·90 (1·76–8·65)[2]	54/86 (63)	4·24 (1·98–9·05)[2]
4+	25/33 (76)	6·13 (2·26–16·60)[2]	35/43 (81)	10·24 (3·80–27·65)[2]
(c) BANDS	PB ($n = 974$)		MB ($n = 103$)	
Sex				
Male	103/592 (17)	Baseline[1]	36/80 (45)	Baseline[1]
Female	46/382 (12)	0·63 (0·43–0·92)[2]	10/23 (44)	1·19 (0·42–3·35)
Age in years				
0–14	9/168 (5)	0·55 (0·25–1·20)	2/14 (14)	0·37 (0·06–2·11)
15–29	29/287 (10)	Baseline[1]	8/26 (31)	Baseline[1]
30–44	49/282 (17)	1·90 (1·16–3·13)[2]	22/38 (58)	3·18 (1·06–9·53)[2]
45–59	43/166 (26)	2·98 (1·76–5·05)[2]	9/18 (50)	2·26 (0·63–8·18)
60+	19/71 (27)	2·84 (1·47–5·50)[2]	5/7 (71)	4·39 (0·62–31·34)
Delay in years				
0–1	62/558 (11)	Baseline[1]	23/55 (42)	Baseline[1]
1–2	37/194 (19)	1·69 (1·07–2·67)[2]	10/24 (42)	1·05 (0·37–3·05)
2–4	24/107 (22)	1·92 (1·12–3·29)[2]	6/14 (43)	1·28 (0·36–4·61)
4+	26/115 (23)	1·83 (1·08–3·10)[2]	7/10 (70)	2·75 (0·56–13·59)

[1] Baseline or reference subcategory.
[2] Significant.

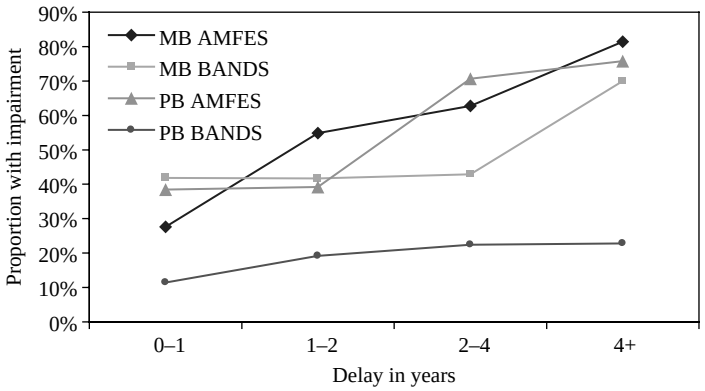

Figure 1. Relationship between impairment at intake and delay by leprosy type and cohort.

groups, passively detected patients had more impairment and MB patients were more impaired than PB patients. However, the differences with actively found patients, also when taking delay into account, were not large.

The Ridley–Jopling classification does not take into account the number of skin lesions, so we examined this separately. Data on skin lesions were only available for the BANDS cohort. The results are shown in Table 3. Of the 969 PB patients, 86% had five or less skin lesions and 14% more than five skin lesions. Impairment rates increased with higher numbers of skin lesions. While patients with less than five skin lesions had low impairment rates, for patients with more than five skin lesions the proportion with impairment was nearly as high as for PB AMFES patients (41 versus 51%). The impairment rates of the last two groups were also comparable with respect to the delay categories.

Discussion

Detection delay is often seen as an important risk factor for the development of impairment. Therefore, many leprosy control programmes give high priority to early detection of leprosy patients. A tool that can predict the proportion with impairment for different delays in all

Table 3. Frequency distribution of impairment at intake in 969 passively detected PB patients from BANDS by delay and skin lesions[1]

	1 lesion (52%)	2–5 lesions (35%)	>5 lesions (14%)
Delay in years	No. impaired (% of cases within subgroup)	No. impaired (% of cases within subgroup)	No. impaired (% of cases within subgroup)
0–1	18/294 (6)	21/197 (11)	23/66 (35)
1–2	10/86 (12)	12/72 (17)	14/35 (40)
2–4	6/54 (11)	7/33 (21)	9/18 (50)
4+	10/66 (15)	7/35 (20)	8/13 (62)
Total	44/500 (9)	47/337 (14)	54/132 (41)

[1] From the 974 passively detected PB patients there were five patients with missing data on skin lesions. Four of them had impairment. These five patients were excluded from the analysis.

situations would thus be very helpful. The objective of this study was to examine the quantitative relationship between detection delay and impairment in two different patient populations. The question to be answered was whether detection delay in relation to impairment is a generally applicable performance indicator of leprosy control programmes. The main finding was the higher impairment rates in AMFES compared with the BANDS cohort, while the duration of the delay was the same. This suggests that the relationship between delay and impairment is not consistent across populations. For example, of all PB patients presenting with a delay of more than 4 years, 76% were impaired in the AMFES cohort and only 23% in BANDS. Similar to other studies, we found that with longer delays the risk of impairment increases. The differences were most striking among PB patients.

There were some difficulties in making a good comparison between the two populations. Firstly, it was necessary to redefine the leprosy classification, because different definitions were used in the studies. If this was not done, 8% of the patients classified as MB in the BANDS cohort would have been labelled PB in AMFES. Although this is a small proportion, differences in definition may have a confounding effect on the size and direction of the relationship between delay and impairment in the different defined groups. Secondly, since 1998 the World Health Organization (WHO) recommends that PB and MB classification is based on skin lesion count only; patients with one to five lesions are PB and patients with six or more lesions are MB.[9] Neither of the studies in this analysis used these current WHO criteria. Skin lesion count data were only available for the BANDS cohort. The BANDS data show that 132 out of the 969 passively detected PB patients had more than five lesions (14%). The impairment rates of these PB patients were very comparable to the ones found in AMFES PB patients.

Several factors may have a confounding effect when comparing detection delay in different populations. Various studies describe factors related to delay in presentation and start of treatment. The most important reasons found for delay are inadequate knowledge and awareness of the disease and its early symptoms, more belief in traditional medicine as first action, misdiagnoses and stigma among staff and poor accessibility to health services.[10–15]

In the present study, some confounding factors may have played a role in comparing the data. First, the assessment of detection delay depended mainly on the recall of the patients themselves. With longer duration of the disease, inaccuracy of recall will be more likely. Differences in recall form an important source of bias. Also, the reported delay might have depended on knowledge and awareness of symptoms. It might be that nerve damage rather than skin patches are regarded as first sign of leprosy due to inadequate knowledge. This may have led to underreporting of the actual delay, especially in Ethiopia, where leprosy control is less well-developed. In addition, the two studies used different methods to define the duration of detection delay. Therefore, the data were transformed for comparison which also can cause bias.

Another difficulty in the comparison were differences between the leprosy control programmes. Two important aspects here are case finding methods and the coverage of leprosy services. With regard to case finding, in the the AMFES cohort the vast majority of patients reported voluntarily (passive case detection), while in the BANDS cohort, 43% reported voluntarily. More than half of the patients were found actively in the BANDS cohort(57%). It is possible that patients were found in the BANDS cohort who would not have been detected if there had been no active case finding. The active nature of the BANDS control programme might also have led to more awareness among the general population of signs and symptoms of leprosy and thus to earlier presentation of individuals with the disease,

in particular of PB patients having a limited number of skin lesions and no nerve damage. In addition, the coverage of leprosy services in terms of availability and accessibility is much better in the BANDS districts than in the AMFES area, making it easier for patients in the BANDS cohort to visit a clinic.[6,7,15] The difference in degree of delay between the AMFES and BANDS cohorts may thus in part be caused by the difference in leprosy control, with the AMFES programme functioning at a more basic level than the BANDS programme. Finally, the differences in impairment and delay may also reflect biological variety, although no evidence is available to confirm this.

We did not include patients with neural leprosy (NL) in the analysis, because these patients were not classifiable as either PB or MB patients. In AMFES there were three NL patients, all with impairment. The BANDS cohort had 52 NL patients, 35 of whom had impairment (67%). Because of the high impairment rates in this group of leprosy patients, examining this group in more detail would be indicated. Due to the explorative nature of this study, we only compared two patient populations. To validate and explain further the results found in this study, analysis of more patient cohorts would be needed.

From this study, it is clear that the relationship between delay and impairment must be seen in the light of the context (e.g. patient population, quality of the leprosy programme, social and cultural attitudes and beliefs). Also, the need for uniform definitions and classification becomes visible when doing comparative analysis.

We conclude that our data support the hypothesis that delay is a useful, but relative indicator. Shorter delays are in general indicative of lower impairment rates. However, with similar reported delays these rates can vary greatly between different patient populations, especially among PB patients. Delay can therefore not simply be used as a general or absolute performance indicator for programme evaluation. Understanding why certain patients or populations delay more than other patients or populations should be just as important as achieving short delays in prevention of disability (POD) programmes.

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