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Effect of diabetes on tuberculosis presentation and outcomes in Kiribati

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Abstract

OBJECTIVES—To determine the association between diabetes and the clinical features and treatment outcomes of TB in Kiribati.

METHODS—We enrolled consecutive patients with TB who presented from August 2010 to February 2012 and compared clinical features and TB treatment outcomes for patients with and without diabetes, as measured by haemoglobin A1c assay. Poor outcome was defined as death, default or treatment failure, and good outcome as treatment success or cure.

RESULTS—Two hundred and seventy-five eligible persons with TB disease were enrolled; 101 (37%) had diabetes. TB patients with diabetes were more likely to have acid-fast bacilli (AFB) seen on sputum smear microscopy (RR: 1.3; 95% CI: 1.03–1.62). The risk of poor outcome did not differ between patients with or without diabetes (RR: 1.1; 95% CI: 0.5–2.7).

CONCLUSION—TB patients with diabetes are more likely than those without to have sputum with AFB on microscopy. This could increase transmission in the community. Early detection of TB by screening patients with diabetes, and the converse, could be important public health interventions where diabetes and TB are prevalent.

Keywords

Tuberculosis; diabetes mellitus; Pacific; case-control study; treatment outcomes

No authors report any conflict of interest.

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Introduction

Globally, tuberculosis (TB) is one of the leading infectious disease causes of death, responsible for more than 1.5 million deaths in 2013 [1]. Although HIV is the strongest amplifier of TB disease worldwide, increasing attention has been given to the risk associated with diabetes [2]. The prevalence of diabetes is increasing at an alarming rate and, although the per person TB risk associated with diabetes is substantially less than that for HIV, it could soon become one of the most important drivers of the global TB epidemic [3, 4].

South-East Asian and Pacific populations have experienced a remarkable increase in the prevalence of diabetes over the past two decades [5]. This represents a threat to TB control and treatment efforts in the nations with an established TB epidemic because diabetes increases TB incidence and transmission by increasing the risk of progression from latent TB infection (LTBI) to active disease [6].

In the Pacific island country of Kiribati, diabetes prevalence exceeds 25% among adults older than 24 years of age, and the prevalence of TB, 748 per 100 000 population, is among the highest in the Western Pacific Region [1, 7]. In 2011, we conducted a case–control study on Kiribati to examine the association between TB and diabetes. We found that the odds of having diabetes are 2.8 times higher for patients with TB than for persons without TB, which suggests that up to a quarter of the TB incidence is associated with diabetes (in press).

The evidence linking diabetes to TB case characteristics and outcome is conflicting, but a systematic review suggests that diabetes may be associated with worse outcomes [8]. Better knowledge of the impact of diabetes on TB outcomes will help the Kiribati Ministry of Health and Medical Services and the National TB Program (NTP) refine screening and treatment protocols. Patients with TB who were enrolled as cases in the case–control study mentioned previously were followed prospectively to learn about associations between diabetes and clinical characteristics and outcomes.

Methods

Setting

We conducted this study in Tarawa, the capital island of Kiribati and home to >50 000 persons, approximately half of the nation's population [9]. We prospectively followed patients with TB who were enrolled as part of a larger case–control study to understand the relation between diabetes and TB treatment outcomes. Cases were enrolled from Tungaru Central Hospital, which is the national referral centre for TB treatment in Kiribati, and the location of the National TB Control Center and the national TB laboratory.

Patients

Eligible patients were 18 years or older, resident of Tarawa, newly diagnosed with TB, and able to provide written informed consent. Pregnant women were excluded. Consecutive eligible patients were enrolled between August 2010 and February 2012.

Measurements and definitions

The Kiribati NTP uses standard and internationally accepted definitions for sputum smear microscopy results and TB treatment outcome [10, 11], and these data were abstracted from TB registers and medical records for our analysis. Patients were diagnosed either clinically (on the basis of signs, symptoms and radiological findings) or by laboratory findings. Not all patients had laboratory confirmation of disease. We categorised patients into mutually exclusive diagnostic categories based on how they were diagnosed. Patients who had any acid-fast bacilli detected on any of their sputum smears were defined as 'pulmonary TB, sputum smear positive'. Patients with pulmonary TB who had at least two sputum smears that were negative by microscopy were defined as 'pulmonary TB, sputum smear negative'. Patients with pulmonary TB who had no sputum smear performed were defined as 'pulmonary TB, smear not performed'. Patients diagnosed with TB who had no pulmonary parenchymal involvement on chest radiograph were defined as extra-pulmonary TB; they were further classified by the extra-pulmonary site of disease.

In addition to routinely collected data, we measured height, waist circumference and haemoglobin (Hgb) A1c for every patient. We used a portable point-of-care device to measure Hgb A1c (the Bayer A1c Now[®] monitor by Metrika, Bayer Healthcare LLC, Sunnyvale, CA) on any patient who was not already diagnosed with diabetes and taking medication for it (Available from: http://www.bayercontour.com/Blood-Glucose-Monitoring/ClassicMeters/A1CNow?WT.mc_id=&WT.srch=1 [accessed February 6, 2014]). For the purposes of this study, a person was considered to have diabetes if they reported a diagnosis of diabetes and were taking medication for it, or in the absence of such history, a Hgb A1c 6.5% (47.5 mmol/mol)[12]. All persons found to have an HgbA1c 6.5% (47.5 mmol/mol)[12]. All persons found to have an HgbA1c 6.5% (47.5 mmol/mol)[12].

Outcomes were analysed as poor versus good. Poor outcomes included patients who died, defaulted or experienced treatment failure. Good outcomes included patients who were cured or completed treatment.

Data collection

Patients with TB were enrolled, and their Hgb A1c tested, when practical after TB diagnosis and registration, but we did not specify a timeframe for A1c testing. Patients were interviewed in the TB ward, the TB clinic or at home. Routine programmatic data, which included demographic information, weight, registration category, type of TB, sputum smear microscopy results and the TB treatment outcome, were collected for each patient. We recorded the results of chest radiographs, when performed, as reported by the treating physician and verified by a medical officer from the United States (US) Centers for Disease Control and Prevention (CDC) blinded to whether or not the patient had diabetes. When there were differences in the interpretation of the radiograph, the readers consulted and determined a mutually agreeable interpretation.

Analysis

Data from the paper forms were double-entered into Microsoft Access, verified and corrected. Analyses were carried out in SAS (version 9.3). We compared the frequencies of different treatment outcomes, smear result, smear grade and smear conversion among those with and without diabetes using a chi-square test. For continuous variables, we calculated medians and compared distributions using the Kruskal–Wallis test. We calculated the relative risk, with 95% confidence intervals, for poor outcome by smear status, comparing TB patients with and without diabetes. We repeated analyses excluding patients who were enrolled and had their Hgb A1c tested > 3 months after TB registration. We defined as significant those comparisons with a *P*-value < 0.05 or 95% confidence intervals that did not cross one.

Ethical considerations

Ethical permission was obtained from the Institutional Review Board at the CDC and the Australian National University, Australia. The Government of Kiribati does not have a human research ethics committee; however, the Ministry of Health and Medical Services provided approval for the study. Participants provided written consent after receiving written and oral information about the study (Available from: http://www.hhs.gov/ohrp/archive/irb/ irb_preface.htm [accessed February 3, 2014]).

Results

Of 275 patients with TB who were eligible and enrolled, 174 were enrolled into the study and had Hgb A1c testing within 90 days of TB registration. Among the 275, 101 (37%) were diabetic: 54 previously diagnosed with diabetes and taking medication for it, and 47 without a prior diagnosis of diabetes who were found to have a Hgb A1c 6.5% (47.5 mmol/mol) or higher. 174 patients had no history of diabetes and had an Hgb A1c less than 6.5%. Hgb A1c measurement ranged from 3.7% to 16.0% (16.9 to 151.4 mmol/mol). Age, weight, body mass index (BMI) and waist circumference were significantly higher in the patients when analyses were restricted to the 174 patients enrolled within 90 days of TB registration, although the *P*-values changed with the smaller sample size (Table 1).

Bacillary burden differed between patients with and without diabetes (Table 2). Patients with diabetes were at greater risk for positive smear, relative to two negative smears, than patients without diabetes (82% vs. 72%; relative risk [RR]: 1.2; 95% confidence interval [CI]: 1.04–1.40). The risk for positive smear, relative to a composite variable which included negative smear, only one smear negative, and smear not performed, was also higher for patients with diabetes (RR 1.3, 95% CI 1.03–1.62). Among the 174 patients enrolled within 90 days of TB registration, 120 met our definition of sputum smear positive or sputum smear negative TB. Of those, 45 of 49 patients with diabetes (92%) had positive smears, compared to 51 of 71 patients without diabetes (72%) (RR: 1.3; 95% CI: 1.08–1.51).

Thirty-four of the 60 (57%) patients with diabetes who were sputum smear positive had sputum smear grades of 2 or 3; 40 of the 80 (50%) patients without diabetes who were sputum smear positive had sputum smear grade of 2 or 3 (RR: 1.1; 95% CI: 0.8–1.5).

Of the 140 patients with sputum smear-positive disease, 133 (95%) had sputum smear microscopy performed at 2 months. Among the 57 patients with diabetes, 46 (81%) converted their sputum smears at two months; by comparison, 64 (84%) of the 76 patients without diabetes converted sputum smears at 2 months (RR: 0.95; 95% CI: 0.82–1.12).

Chest radiography was performed on 111 patients with TB, 48 (48%) of 101 patients with diabetes and 63 (36%) of 174 patients without diabetes (Table 3). Disagreements between interpreters occurred in three cases and were easily resolved. Patients with diabetes were more likely to have cavitary disease, bilateral abnormalities, consolidation and hilar lymphadenopathy; none of these differences, however, were statistically significant.

There were no statistically significant differences in TB treatment outcomes between patients with and without diabetes (Table 4). This remained true when analysis was restricted to the 174 patients enrolled within 90 days; in that group, 3 of 68 patients with diabetes (4%) had a poor outcome, as compared to 8 of 106 (8%) (RR: 0.6; 95% CI: 0.16–2.12).

Discussion

This is the first study comparing TB patients with and without diabetes in Kiribati, an island nation with a high prevalence of both diseases. Our findings suggest that TB patients with diabetes are more likely to present with sputum smear-positive disease when compared to patients without diabetes, and are consistent with other research [13, 14]. Clinical outcomes, however, did not differ.

Diabetes not only increases the likelihood of progressing from LTBI to active TB disease, but our data suggest an association between diabetes and positive sputum smear microscopy. Thus, diabetes may increase the likelihood of TB transmission not only by increasing the prevalence of active TB but also because positive sputum smear is associated with increased infectiousness [15, 16]. An important priority for TB control efforts is to interrupt transmission by diagnosing the most infectious cases (i.e. those with higher bacillary burdens, as demonstrated by positive sputum smear microscopy or other diagnostic testing) and initiating treatment as early as possible [17, 18]. Earlier diagnosis requires active case-finding in populations at risk [19–22]. This could be accomplished by identifying diabetes in the community, and regular, active screening for TB among patients known to have diabetes [2, 23]. Data from our associated case–control study suggest that a quarter of all TB in Kiribati may be attributable to diabetes (in press); therefore, diabetes may be the single biggest contributing factor to the TB epidemic in Kiribati, and a critical focus for TB control efforts.

In some settings, diabetes appears to affect treatment outcomes [8, 13, 24], but this is not a universal finding [25–27]. Our data suggest that in Kiribati diabetes does not influence outcomes. This might be because, in general, TB outcomes in Kiribati are very good, with

the proportion of successfully treated patients already exceeding the 2015 target of 85% established in the Global Plan to Stop TB [28]. Tarawa is a relatively small and contained community, and it is likely that patients are referred early in the course of disease, compared to patients in other settings; it is also fairly easy to track patients and support adherence. In such an environment, with a well-functioning programme of directly observed therapy, diabetes may not have a demonstrable negative impact on outcome. It is also possible that the patients diagnosed with diabetes in this study were diagnosed early, before diabetes substantially compromised immune functioning. We excluded patients from the outer islands because of logistical difficulties in follow-up, and in those patients, diabetes may be more likely to impact TB treatment outcome.

Our data demonstrate associations between age, weight, waist circumference and diabetes. These findings are not surprising, given that these variables are understood to be causally related to diabetes; therefore, we do not report multivariate analyses that adjusted for them. It follows that an important method to reduce the burden and transmission of TB is primary prevention of diabetes, with protocols aimed at promoting weight reduction and maintaining physical fitness.

There are some important limitations of these findings, and the limitations do not allow definitive statements about the association between diabetes and TB presentation or outcome to be made. Data presented here were part of a case-control study designed to detect an association between TB disease and diabetes, comparing those with TB to control patients from the community; it was not specifically designed to detect differences in the presentation or outcomes between TB patients with and without diabetes. It may be that diabetes does impact treatment outcome in Kiribati, but the effect may not have been detected due to the relatively small numbers of patients in the study. Because we used a single point-of-care assay, our definition of diabetes might be considered somewhat less accurate than a quality-assured pathology laboratory test [29]. However, it was reassuring to note that the results from the case-control study revealed the expected prevalence of diabetes in the randomly selected control group and the expected magnitude of association between TB and diabetes, both of which suggest that the assay we used was accurate [6, 30, 31]. We did not have access to TB culture to verify diagnoses of TB; at the time of our study, the TB laboratory relied on sputum smear microscopy to confirm a diagnosis of pulmonary TB and other cases were diagnosed clinically. Finally, testing for Hgb A1c did not uniformly occur at the time of TB diagnosis and registration, and in some cases, patients were enrolled into the study months after TB registration and treatment began. Because active TB disease and TB treatment can change glucose metabolism, it may be that delays in testing affected the diagnostic categories. Inflammation associated with TB disease could have raised glycosylated Hgb independent of participants' diabetic status. Conversely, patients with diabetes who were substantially underweight at the time of TB diagnosis could have had a spuriously low glycosylated Hgb (compared to their baseline). Given these possibilities, a delayed glycosylated Hgb could be considered more accurate than testing carried out at the time of TB diagnosis. Our methodology is strengthened by our use of the Hgb A1c assay for diabetes diagnosis, and our conclusions are supported by 2 additional findings: 1) analyses restricted to persons enrolled within 90 days of TB registration yielded similar findings, and 2) the proportion of patients who were diabetic was similar among

those tested within 8 weeks before and within 8 weeks after TB registration (38% vs 35%). Despite the limitations, we believe these data are compelling and provide evidence to support the collaborative activities between the management of TB and diabetes that are

Conclusions

being promoted in Kiribati.

In Kiribati, diabetes is associated with higher bacillary burdens at diagnosis, and this may contribute to increased transmission of TB. We did not find evidence that diabetes is associated with worse TB treatment outcomes. These data provide a rationale to broaden both diabetes and TB case-finding in Kiribati, and add to a growing evidence base for expanding these activities globally. These data do not indicate a need to alter or enhance TB treatment regimens for diabetic TB patients in Kiribati, but they must be interpreted within the context of the limitations. Further studies designed to detect differences in TB presentation or outcome between patients with and without diabetes are warranted.

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(a) Characteristics of TB patients with and without diabetes (n = 275). (b) Characteristics of TB patients with and without diabetes who were tested within 90 days of TB registration (n = 174)

	Patients with diabetes $n = 101$	Patients without diabetes <i>n</i> = 174	P-value
(a)			
Age – median (interquartile range (IQR))	49 (39–56)	26 (20-40)	< 0.01
18–29 – no. (%)	12 (12)	106 (61)	
30-44 - no. (%)	24 (24)	33 (19)	
45–59 – no. (%)	48 (48)	23 (13)	
60 – no. (%)	17 (17)	12 (7)	
Female – no. (%)	44 (44)	87 (50)	0.30
HIV	0	0	n/a
Previous TB – no. (%)	6 (6)	10 (6)	0.95
Hgb A1c – median (IQR)	9.3% (7.0–11.8%); 87.1 mmol/mol (53.0–105.5 mmol/mol)	5.5% (5.0–5.9%); 36.6 mmol/mol (31.1–41.0 mmol/mol)	< 0.01
Initial weight in kgs [*] – median (IQR)	66 (58–77)	60 (54–69)	< 0.01
Waist circumference $\dot{\tau}$ – median (IQR)	89 (78–97)	82 (78–90)	<0.01
Height in cms ^{\ddagger} – median (IQR)	168 (162–173)	167 (160–172)	0.31
Body mass index $(BMI)^{\delta}$ – median (IQR)	23.9 (19.8–27.2)	21.7 (19.2–25.0)	< 0.01
	Patients with diabetes $n = 68$	Patients without diabetes $n = 106$	<i>P</i> -value
(b)			
Age - median (interquartile range (IQR))	49 (39–58)	26.5 (21-43)	< 0.01
18 – 29 – no. (%)	8 (12)	60 (57)	
30 – 44 – no. (%)	14 (21)	23 (22)	
45 – 59 – no. (%)	32 (47)	16 (15)	
60 – no. (%)	14 (21)	7 (7)	
Female – no. (%)	28 (41)	55 (52)	0.17
Previous TB – no. (%)	5 (7)	8 (8)	0.96
Hgb A1c – median (IQR)	9.5% (7.0–11.5%)	5.6% (5.1-6.0%)	< 0.01
Initial weight in kgs [¶] – median (IQR)	67 (58.5–77)	62 (54–72)	0.04
Waist circumference ** – median (IQR)	89 (77–95)	81 (78–90)	0.07
Height in cms ^{$\dot{\tau}\dot{\tau}$} – median (IQR)	169 (163–173)	168 (160–173)	0.24
Body mass index (BMI) $\stackrel{\ddagger}{\downarrow}$ – median (IQR)	23.9 (19.5–26.9)	22.0 (19.0–27.3)	0.19

* Initial weights were available for 92 patients with diabetes and 165 patients without diabetes.

 † Waist circumference was available for 99 patients with diabetes and 173 patients without diabetes.

 \ddagger Height was available for 100 patients with diabetes and 169 patients without diabetes.

 $^{\$}$ BMI was available for 91 patients with diabetes and 161 patients without diabetes.

IInitial weights were available for 64 patients with diabetes and 102 patients without diabetes.

** Waist circumference was available 66 for patients with diabetes and 105 patients without diabetes.

 $^{\dagger \dagger}$ Height was available for 67 patients with diabetes and 103 patients without diabetes.

 $^{\ddagger \ddagger}$ BMI was available for 63 patients with diabetes and 99 patients without diabetes.

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Association between diabetic disease and type of tuberculosis

	Pulmonary TB, smear positive <i>n</i> (%)	Pulmonary TB, smear negative <i>n</i> (%)	Pulmonary TB, Only 1 smear <i>n</i> (%)	Pulmonary TB, smear not performed <i>n</i> (%)	Extra-Pulmonary TB n (%)	Relative risk (95% confidence interval) [*] n (%)
Patients with diabetes	60 (59)	6) 6	7 (7)	5 (5)	20 (20)	1.21 (1.04–1.40)
Patients without diabetes	80 (46)	31 (18)	14 (8)	12 (7)	37 (21)	Ref

* Relative risk for patients with diabetes presenting as smear positive restricted to those who were either smear positive or smear negative 60/69 (87%), as compared to patients with non-diabetes 80/111 (72%).

Table 3

Chest radiographic findings, comparing patients with diabetic and non-diabetic tuberculosis

	Diabetics $n = 48^*$, (%)	Non-diabetics $n = 63^*$, (%)	<i>P</i> -value
Cavity	24/48 (50)	27/63 (43)	0.45
Bilateral disease	35/48 (73)	39/62 (63)	0.27
Consolidation	28/47 (60)	27/62 (44)	0.10
Hilar lymphadenopathy	38/48 (79)	50/61 (82)	0.71
Pleural effusion	18/48 (38)	24/63 (38)	0.95
Miliary disease	4/48 (8)	12/63 (19)	0.11

* Chest radiographs were available for 48 (48%) of the 101 patients with diabetes, and for 63 (36%) of the 174 patients without diabetes; some aspects of some radiographs were unreported.

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Association between diabetes and tuberculosis treatment outcomes *

	Poor outcome			Success		
	Treatment Failure n (%)	Default n (%)	Died n (%)	Cure n (%)	Treatment complete n (%)	Relative risk (95% confidence interval)
Patients with diabetes $(n = 101)$	1 (1)	3 (3)	4 (4)	49 (49)	44 (44)	
	8 (8)			93 (92)		1.1 (0.5–2.7)
Patients without diabetes $(n = 174)$	2 (1)	8 (5)	2 (1)	68 (39)	94 (54)	
	12 (7)			162 (93)		ref

es to those without diabetes.

The highlighted values indicate how the relative risk was calculated.