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Type 2 diabetes and multi-drug resistant tuberculosis

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Abstract

The association between tuberculosis (TB) and diabetes is re-emerging with the epidemic of type 2 diabetes (T2DM). We analyzed retrospective data from 2,878 TB patients across the Texas/Mexico border. Overall 161/2878 (5.6%) patients had MDR TB (resistance to rifampin and isoniazid): Texas 49/1442 (3.4%) and Mexico 112/1436 (7.8%). In Texas MDR TB was significantly associated with T2DM (OR 2.1 95% CI 1.1–4.2) when adjusted for age, gender, drug and alcohol abuse, HIV infection and history of previous episode of TB, and in Mexico (OR 1.80 95% CI 1.1–2.9) when adjusted for age and gender. Patients with T2DM were consistently more likely to be compliant with DOTS therapy (OR 2.4 95% CI 1.1–5.4) than patients without T2DM. In Texas, all but 3 of the T2DM patients with MDR TB were resistant at their first culture at the time of diagnosis. It is possible that impaired immunity in T2DM increases susceptibility to infection with resistant strains.

Introduction

Until recently understanding of the association between diabetes and tuberculosis (TB) was chiefly based on reports from the 1950s in which type 1, insulin-dependent, diabetes predominated.[1,2] With the current global epidemic of obesity there is now increasing awareness in several countries of the association between type 2 diabetes (T2DM) and TB, [3] particularly among Mexicans and Mexican Americans [4,5,6]. Since T2DM constitutes in

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With the **Nuevo Santander Tuberculosis Trackers**, (http://www.nstt.info/)

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most studies at least 95% of all diabetes patients, the population at risk greatly exceeds that of the type 1 diabetes patients.[7] Thus the association between T2DM and TB is clearly significant in some populations and presents a particular challenge where there is high prevalence of both diseases.[4,8,5] In the largest published study so far, we have recently reported a strong association between self-reported T2DM and TB among several thousand Mexican-American TB patients both sides of the US/Mexico border.[6] In that report T2DM was by far the most frequent risk factor for TB, particularly in patients over 40 years of age, women, and those without the usual social risk factors for TB (incarceration, substance or alcohol abuse, HIV infection).

The “rediscovery” of the association between diabetes and TB raises important questions for TB control. Treatment is currently the major method of TB control world wide.[9] Increasing numbers of multi-drug resistant (MDR TB) strains, resistant to at least isoniazid (INH) and rifampin (RIF) are a threat to success. Though tuberculosis in the United States has been steadily decreasing, nearly 10% of all new cases are now resistant to at least one primary drug and 1.6% are resistant to both INH and RIF.

Patients with T2DM are known to have altered immunity, specifically a chronic inflammatory state in which many cytokines and chemokines are upregulated. These patients are particularly susceptible to bacterial infections, notably *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Pseudomonas aeruginosa* and *Mycobacterium tuberculosis*.[10] Since immunodeficiency in HIV/AIDS is an important driving force behind MDR TB, and now extensively drug resistant TB (XDR TB), both of which threaten recent gains in the control of TB in many countries, [11] we questioned whether the epidemic of T2DM might also have an effect on spread of drug resistance.

An association between T2DM and MDR TB infections has been reported in a small series of hospitalized patients.[12] In discussion of the significance of that report, the need for more conclusive investigations in different populations was highlighted. Our large database provided an opportunity to examine more thoroughly the association between MDR TB and T2DM in a community-based patient population.

Methods

Our working consortium, the Nuevo Santander Tuberculosis Trackers, shared and analyzed data on 2,878 adult (18 years or older) TB patients in south Texas and adjacent northeast Mexico (map at http://www.nstt.info). The Texas database included all TB patients identified between 1996–2002 in the 19 South Texas counties comprising Public Health Region 11. The Mexican data included all TB patients in the border Sanitary Jurisdictions 3 (Matamoros), 4 (Reynosa) and 5 (Nuevo Laredo) from the State of Tamaulipas, from 1998 through 2003. We excluded from final analysis all patients for whom diabetes or culture and sensitivity data were missing. This resulted in two final datasets comprising 1,442 patients from Texas and 1,436 patients from Mexico for whom we had complete data on sputum culture with drug sensitivities and information on self-reported T2DM. (Self-reported diabetes is a “yes” response to the question as to whether the patient has been told they have diabetes.) For simplicity we will refer to the study sites as “Texas” and “Mexico”. Both programs apply DOTS (directly observed therapy, short course) regimes. DOTS is enforced by law in Texas and cultures and drug sensitivity are performed for all patients at diagnosis. In Mexico, sputum smears are used for diagnosis for all patients. Specimens for culture from Mexican patients are only available for selected patients, frequently but not exclusively patients failing to respond to regular therapy, through the Center for Disease Control-funded binational initiative ‘Grupo Sin Fronteras’ (http://www.tdh.state.tx.us/news/acc0323.htm). There is no selection bias in patients with culture data for or against inclusion of patients with diabetes. The timing and

selection of patients for this program is not systematic and is not recorded. Furthermore there are differences in data collection in the two countries; in particular more than one potential risk factor can only be recorded in the Mexico database. We therefore analyzed the two datasets separately.

For the purposes of analysis, TB with T2DM was any TB patient with self-reported T2DM while TB without T2DM was any TB patient not reporting T2DM. Though this definition is subject to misclassification, particularly of undiagnosed diabetes, it is in line with the Centers for Disease Control and Prevention BFRSS surveys and the ENSA surveys from Mexico.[13,14] Blood glucose is not measured in any of the clinics. Patients recorded with alcohol or drug abuse were those reporting use, as there is no quantitative information provided. Patients re-entered into either data base for a second course of TB treatment were defined as 'previous history of TB'. Since we wished to determine the contribution of failure of adherence to therapy in leading to development of MDR TB, we documented whether each patient had satisfactorily completed DOTS treatment.

All analyses were run on datasets without personal identifiers using the Statistical Analysis Software System (SAS Institute, Cary, NC). Ethical approval was obtained from all the participating institutions including the University of Texas Health Science Center at Houston, TDSHS and the Institutional Review Board of the Secretaria de Salud in Tamaulipas. Logistic regression models were used to assess potential associations between MDR and T2DM as well as treatment compliance among TB patients adjusting for age, gender, drug and alcohol use and for previous episode of TB in Texas, or age and gender in Mexico. For each model we tested for potential interactions between each pair of independent variables but there were no significant interactions. Next we proceeded to drop out of the model variables that were no longer significant, until we were left with a model containing only variables with significant effect on our chosen outcome. Based on our final logistic regression models we report adjusted odds ratios along with their corresponding 95% confidence intervals. All statistical hypotheses testing procedures were conducted at 5% level of significance.

Results

Table 1 shows the characteristics of the patients included in the final database for whom we had culture data All but one of the Texas patients had culture data reported, so the patients in this analysis represent essentially the entire Texas TB population. In Mexico, however only 1443/3411 (42.3%) of the patients had culture data reported. When we compared these characteristics with those in the original databases we found no significant differences in any of these variables (data not shown). As previously reported using the full TB dataset, univariate analysis showed that T2DM was significantly associated with gender and being of Mexican American origin in Texas.[6] However, in Texas about 94% of the population is Mexican American, and in Mexico, approaching 100% are Mexican.[15] Younger age group, history of drug or alcohol abuse, HIV infection, history of incarceration and homelessness were significantly associated with no history of T2DM. The Mexican data could only be adjusted for age and gender since only one risk factor is routinely recorded in their data collection system.

Overall 161/2878 (5.6 %) patients had MDR TB, 51 (31.6%) of whom reported T2DM. In Texas 49 of 1442 (3.4%) had MDR TB and in Mexico 112 of 1436 (7.8%). In Texas 18/49 (36.7%) patients with MDR TB reported T2DM, and in Mexico 33/112 (29.5%). Though not statistically significant, TB patients in Texas with DM were l.5 times more likely to have MDR TB than patients without DM in the univariate analysis (table 2). In the multivariate model controlling for age, gender, alcohol and drug abuse, HIV infection and history of previous TB infection, patients with diabetes in Texas did have a statistically significant higher odds of

MDR TB than patients without diabetes (OR 2.14 95% CI 1.10–4.17). Similar analyses using the Mexico data set (data not shown) showed that T2DM was significantly associated with MDR TB in a univariate analysis (OR 1.76 95%CI 1.15–2.70) as well as a model adjusted for age and gender (OR 1.80 95% CI 1.13–2.87).

Diabetes patients were more likely to complete DOTS satisfactorily than patients with risk factors such as drug and alcohol abuse and HIV infection. In a model adjusting for age, gender and history of previous episode of TB we found that patients with T2DM were significantly more likely to complete their DOTS therapy (OR 2.4 95% CI 1.06–5.4) when compared with patients without T2DM. Among the non-compliant were the drug and alcohol abusers (OR 0.64 95% CI 0.36–1.15 and OR 0.65 95% CI 0.34–1.24 respectively).

As has previously been reported, we found that patients with previous history of TB were significantly more likely to have MDR TB. However, patients reporting T2DM (27.8% in Texas and 20.0% Mexico) were no more likely to have a history of previous TB than those without T2DM even after adjusting for age, gender, and alcohol and drug abuse. Among patients with a previous history of TB, those with T2DM were more likely to have MDR TB than patients without T2DM in Mexico (OR 3.0 95% CI 1.1–8.2) but not in Texas. Similarly we looked for, but did not find significant associations between T2DM and deaths, with 11.8% mortality among T2DM patients and 11.0% among patients without T2DM (Texas data).

We reviewed the records of all the Texas TB patients with or without DM in this study who had drug resistant strains reported. All but three had resistant strains from their first culture. In Texas the first culture is taken at the time of diagnosis, so that we may reasonably conclude that these patients had primary resistance. In Mexico diagnosis depends on smear with sputum only being cultured later if the patient is entered into the TDSHS binational program. We are therefore not able to distinguish primary and secondary resistance in the Mexican patients.

Discussion

Our data show that T2DM is significantly associated with MDR TB in Mexican and Mexican American TB patients. Over 31% of patients with MDR TB reported T2DM, compared with 27.8% of all TB patients.[6] This compares with a prevalence of self-reported diabetes in the same general population of 15.5%.(Fisher-Hoch, unpublished data) The Texas database comprises the entire TB population. The Mexican database comprises patients selected for culture; however presence or absence of diabetes does not influence that selection. We conclude from our data in both Texas and Mexico that T2DM patients with TB are more prone to drug resistance.

Patients with TB and T2DM comprise a large population with active infection among Mexicans and Mexican Americans. Now we show that this population also contributes to an increased pool of MDR TB infected patients. Indeed the overall rates in this study qualifies our border area as a “hot zone”, defined as an area that has ≥5% prevalence (or incidence) of MDR TB. [16] Paradoxically, ‘hot zones’ can be ones with good TB control programs, possibly because they successfully treat and thus reduce transmission of wild-type pan-sensitive strains whereas MDR TB is less successfully treated and thus spreads preferentially.[16]

We found that younger TB patients were also significantly more likely than older patients to have MDR TB. In our previous analyses [6] we have shown that most of the patients with established social risk factors (alcohol and drug abuse, incarceration and HIV infection) are in this younger age group. Though the T2DM patients are mostly older and often female, we feel that our model is sufficiently robust in controlling for age. To doubly ensure we were correct we re-ran our model using a wide range of different age groups and cut-offs, and found the association between MDR TB and T2DM remained consistently significant.

Failure to complete prescribed therapy is important in the development of MDR TB, and occurs frequently in drug and alcohol abusers.[9] We find T2DM patients fully compliant with DOTS. A possibility is, as has been recently shown in patients with T2DM, that diabetes patients do not achieve or maintain adequate blood levels of rifampin, one of the foundation drugs of the DOTS regime.[17,18] However, failure to comply with therapy or to metabolize drugs effectively would result in secondary resistance; that is, resistance developing later in the course of treatment in a patient who initially had a sensitive strain. Secondary resistance is often associated with recurrent infections which we did not find significantly increased in T2DM patients. In our study MDR TB was isolated from the initial cultures of nearly all of the Texas patients, that is, primary resistance. Only 3 MDR TB patients with T2DM had records of previous TB, insufficient to account for this effect. We have to posit that T2DM patients may be more susceptible to disease following infection with resistant strains.

There could be several explanations for the increased frequency of disease due to resistant strains in T2DM. Bacterial genetics may play a role. Some studies suggest that MDR TB strains are less fit due to multiple single locus mutations, [19,20,21] though others have challenged this conclusion.[22] An epidemiological study using genotyping on isolates from a large number of patients in San Francisco found that MDR TB strains are only half as likely as drug sensitive strains to lead to secondary cases.[20] This study measured rates of disease, however, not infectivity. Infectivity measured by skin test positivity in contacts has previously been shown to be the same with drug sensitive as with drug resistant strains.[23] Burgos et al. conclude from their study that the observed reduction in secondary cases in persons exposed to MDR TB strains must be because fewer develop active disease. This can be explained by heterogenous mutations, some resulting in loss of virulence.[20,20,23]

T2DM may itself involve immune impairment, with potential mechanisms likely linked to poor control of hyperglycemia, which may allow less fit strains to flourish.[24] Known impairments linked to poor glucose control include phagocytosis, chemotaxis, generation of reactive oxygen species (ROS) and T-cell function.[17] Recently Martens et al. used a mouse model of chronic diabetes infected with TB.[24] These mice had very high blood glucose levels (average non-fasting glucose 463mg/dl, and fasting glucose >200mg/dl). When infected with MTB chronically diabetic mice manifested high bacterial burdens and increased inflammation in their lungs compared with controls and with acutely diabetic mice. This study demonstrated impaired initiation of the adaptive immune response to the infection in chronically diabetic mice, marked by reduction in early interferon-γ production, and fewer ESAT-6-specific T cells in lungs compared with euglycemic mice and mice with acute diabetes.[24] These studies in a mouse model support the notion that impaired responses in diabetes to MTB infection is associated with poor glucose control. We have recently demonstrated similar differences in immune responses to MTB in patients with diabetes, further supporting the concept that immune impairment may play a role.[25] These impaired responses in poorly controlled T2DM patients may allow less fit resistant strains to flourish more readily.

A combination of bacterial and host factors may be important FOR WHAT. Molecular studies show that mutations in the MTB *kat*G gene are important. This gene contributes to protection of the bacterium against oxidative stress, but it also encodes a catalase-peroxidase which transforms isoniazid into its active form.[21],28] Strains with these mutations may be better able to thrive in patients with T2DM in whom production of ROS may be impaired.[10,21, 10] The situation is likely to be complex and there may be multiple other mechanisms in play, including failure of phagocytosis and inadequate T-cell responses.

We did look to see whether there was clustering of MDR TB strains which might be considered to be less fit, but we had insufficient genotyping data. We have limited genotyping data on other strains, however, which indicated that about half the T2DM patients had isolates which

clustered and half did not (data not shown), consistent with previous published observations from Mexico.[5]

There are limitations to our study. Firstly the data are retrospective and collected for surveillance rather than research purposes. There may be errors in sampling, misclassification of cases, and we observe variations in diagnosis and treatment practices. Differences in reporting procedures, together with missing data make it difficult to compare between countries for risk factors with low prevalence. We believe that the proportion of patients self-reporting T2DM is an underestimate since about 6% or more T2DM patients are not previously diagnosed.[26,14] This means that our estimation is the minimum, and that we might be including some cases of MDR TB in the non diabetes group who actually belong in the diabetes group. We cannot account for differences in duration of exposure to MTB nor infectious dose. Despite these limitations our conclusions are of considerable public health importance, providing the basis for smaller, prospective studies designed to answer specific questions with greater accuracy.

The epidemic of T2DM is developing against a background where approximately one third of the world's population is latently infected with MTB. Convergence of these two pandemics is most marked in transitional cultures, among which is the US/Mexico border. Countries such as India and China are experiencing increases in T2DM in rapidly developing, very large populations with high rates of endemic tuberculosis. T2DM may soon join the many forces driving the spread of TB, including MDR TB in these populations.[27] The molecular and immunological basis for this susceptibility needs to be explored to establish its basis and the implications for prevention and treatment.

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Table 1

Prevalence of T2DM in TB patients by sociodemographic

Variable	Texas				Mexico			
	*Total	Diabetes		†OR (95% CI)	*Total	Diabetes		†OR (95% CI)
		n	%			n	%	
All patients	1442	401	27.8		1436	287	20.0	
Female	453	148	32.7	1.41 (1.11–1.80)	401	96	23.9	1.09 (0.71–1.66)
Male	989	253	25.9	1	1035	191	18.5	1
Age group (yrs)								
20–39	520	54	10.4	0.22 (0.15–0.32)	730	49	6.7	0.27 (0.15–0.49)
40–69	669	260	38.9	1.22 (0.96–1.64)	620	220	35.5	2.08 (1.21–3.58)
70+	253	87	34.4	1	86	18	20.9	1
Hispanic	1305	382	29.3	2.57 (1.56–4.23)				
Mexican origin	691	212	30.7	1.29 (1.02–1.63)	Data not available			
Incarcerated	127	7	5.5	0.11 (0.52–0.24)				
Homeless	40	3	7.5	0.21 (0.07–0.69)				
HIV	69	7	10.1	0.24 (0.11–0.54)				
Alcohol abuse	292	51	17.5	0.48 (0.35–0.67)				
Drug abuse	152	18	11.8	0.32 (0.19–0.53)				
Previous episode of TB	75	23	30.7	1.15 (0.69–1.01)				

*Total number of patients with complete data on diabetes status, culture and sensitivity and risk factor of interest.

§Model adjusted for age, gender, alcohol and drug abuse, HIV infection and history of previous episode of TB.

†MTB sputum isolate resistant to Rifampin and Isoniazid

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Table 2

Prevalence of MDR-TB by potential risk factors in Texas

Exposure variable	*Total	†Multidrug Resistant		Univariate analysis	§Multivariate analysis
		n	(%)	Odds Ratio (95% CI)	Odds Ratio (95% CI)
All patients	1442	49	3.4		
Diabetes	401	18	4.5	1.53 (0.85–2.77)	2.14 (1.10–4.17)
No diabetes	1041	31	3.0	1	1
Age group in years					
20–39	520	26	5.0	2.17 (0.89–5.33)	3.13 (1.18–8.28)
40–69	669	17	2.5	1.07 (0.42–2.75)	1.15 (0.44–3.03)
70+	253	6	2.4	1	1
Female	989	16	3.5	0.94 (0.51–1.73)	
Male	453	33	3.3	1	
HIV positive	69	1	1.5	0.31 (0.04–2.36)	
HIV negative	559	25	4.5	1	
No HIV information	814	23	2.8	0.62 (0.35–1.11)	
Alcohol abuse history	292	7	2.4	0.65 (.029–1.46)	
No alcohol abuse history	1150	42	3.7	1	
Drug abuse history	152	5	3.3	0.96 (0.38–2.47)	
No drug abuse history	1290	44	3.4	1	
Incarcerated	127	5	3.9	1.15 (0.44–3.00)	
Not incarcerated	928	37	3.5	1	
Homeless		1	2.1	0.73 (0.10–5.46)	
Not homeless		39	2.9	1	
Recurrent tuberculosis	75	9	12.0	4.62 (2.15–9.94)	5.00 (2.26–11.03)
No previous tuberculosis	1361	39	2.9	1	1

*Total number of patients with complete data on diabetes status, culture and sensitivity and risk factor of interest.

§Model adjusted for age, gender, alcohol and drug abuse, HIV infection.