Spontaneous pneumothorax and tuberculosis: long-term follow-up

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ABSTRACT: **Though spontaneous pneumothorax (SP) is a well-known complication of pulmonary tuberculosis (TB), there are very few reports addressing this topic. For this reason, we retrospectively analysed the experience of SP in patients diagnosed with TB in our hospital between 1989 and 2010.**

Out of 872 patients treated for SP during this period, 47 (5.4%) had TB antecedents, 21 with active TB (0.95% of the 2,089 TB cases diagnosed during this period) and 26 with residual inactive TB. 46 cases were treated with pleural drainage (PD): 40 (85%) with only one PD, two with two, and four with three. The mean ± SD length of PD treatment was 12.9 ± 11.3 days. In 11 (23%) cases, a relapse of SP occurred, with no statistical relationship between the different studied variables. In 13 (28%) cases, it became necessary to carry out a resection (atypical segmentectomy in all cases) for persistent air leaks with PD. Survival statistics were unfavourable only in elderly patients and those infected with HIV.

We conclude that the treatment of SP secondary to TB with PD is usually a sound response, with a good general prognosis and a low percentage of cases that require another PD and surgical treatment.

KEYWORDS: Pleural drainage, pneumothorax, pulmonary tuberculosis, secondary spontaneous pneumothorax, surgical treatment

Spontaneous pneumothorax (SP) is defined as the sudden presence of air in the pleural cavity without apparent external cause. The majority of cases can be classified as primary SP, ocurring in the young without associated pulmonary or general disease. The production mechanism of primary SP is the rupture of some subpleural pulmonary alterations known as blebs [1]. Secondary SP (SSP) is associated with clinical or radiological evidence of significant lung disease, most frequently a consequence of chronic obstructive pulmonary disease (COPD). Pulmonary tuberculosis (TB) has been repeatedly described as a frequent cause of SSP [2–6], with a very variable presentation frequency according to the few authors that have studied this matter. Over the past few decades, a clearly growing incidence of SSP caused by TB has been described [4]. The frequency with which SSP can complicate the course of TB has also been little studied, with figures ranging from 0.6 to 1.4% [5]. These figures, although appearing to be very low, can be supremely important if we keep in mind the 9.5 million new cases that the World Health Organization reports are produced in the world every year [7]. 80% of these cases occur in developing countries. In spite of the fact that TB is a frequent cause of SSP, very few series, with very few patients, have been reported. Therefore, the frequency of presentation, the clinical and therapeutic management, and the prognosis of these patients have been poorly studied. There is very little contrasting experience with this type of SSP. With the aim of reviewing our experience and shedding some light on these matters, we present our studies of SSP and TB over the past 21 yrs.

MATERIALS AND METHODS

The Dr Negrín General Hospital (Las Palmas, Spain) serves a population of ~500,000 people, corresponding to the northern area of the island of Gran Canaria. There is one thoracic surgery unit in this area, to which all cases of SP are referred. The average incidence of TB on the island of Gran Canaria was 32.2 cases per 100,000 inhabitants in 1988 and 23 cases per 100,000 inhabitants in 2009 [8].

The present study is observational, retrospective and longitudinal, analysing data corresponding to clinical reports of all patients with SSP and TB treated in our hospital between January 1, 1989 and December 31, 2009. The following data were

collected from the medical records: age, sex, domicile, co-morbidity and past medical history (smoking, alcoholism, drug addiction, social indigence, functional criteria of COPD, previous respiratory infections, previous TB and other pneumological antecedents), clinical data (dyspnoea, pleuritic pain, fever, cough, expectoration, diagnosis of active TB, bronchopleural fistula and whether the patient was in treatment with anti-TB drugs), diagnostic method for pneumothorax (radiography and computed tomography (CT) scan), its location and radiological characteristics (presence of pulmonary infiltrates, pulmonary atelectases, caverns, bronchiectasis, destroyed lung, granulomas, pachypleuritis, calcium plaques, pleural effusion and mediastinal adenopathies), data related to pleural drainage (PD) (duration in days and recurrence of SSP needing PD again), necessity for surgery and type of surgery performed, hospital stay, number of admittances, complications and perioperative mortality (*exitus* during the same admittance), and long-term survival. Long-term survival was considered to be length of the period between the performance of the pleural drainage and death, or the date of the last follow-up observation before the analysis if the subject was still alive. For the follow-up, the hospital's database was used. The variables considered as end-points were recurrence of SSP and long-term survival after having suffered a pneumothorax.

Diagnosis of SSP was always confirmed through radiological studies, either conventional thoracic radiography or CT. All radiological examinations were evaluated by an independent radiologist.

In all but one case, PD was performed. The procedure was carried out under local anaesthesia, inserting a 28-French drainage tube in the anterior axillary line, fifth intercostal space. Surgical intervention (thoracotomy or video thoracoscopy) was indicated in the presence of air leaks lasting for >10 days and when there was a recurrence of SSP. For this indication, the general performance status of the patient was taken into account as well as pulmonary parenchymal involvement, and the presence of pleural sequelae. In all cases possible, a test of respiratory function was performed. If there was any suspicion of endobronchial pathology, bronchoscopy was indicated.

Statistical analysis

Analyses were performed using SPSS version 14.0 (SPSS Inc., Chicago, IL, USA). The placement of PD was considered as the initial date of the study. A descriptive analysis of the sample was performed, estimating the frequency and the corresponding percentages in the case of categorical variables and the mean$\pm$SD. Afterwards, possible relationships among descriptor variables were studied along with those considered end-points. In the case of categorical variables, the Chi-squared test was used and, in cases where this did not meet the conditions of the application, Fisher's exact test was performed. As for numerical variables, in order to compare measurements, unpaired t-tests were applied.

Survival curves were calculated according to the Kaplan–Meier method. Differences in survival were analysed with the log-rank test and the hazard ratio (HR) was estimated. The relationship between the numeric variable age and survival was estimated using Cox regression. In all cases, a p-value <0.05 was considered significant.

RESULTS

During the study period, a total of 872 patients with SP were treated in our hospital, 47 (5.4%) of whom had SSP secondary to TB. Also during this period, 2,089 cases of active TB were treated. Of the 47 cases with SSP secondary to TB, 21 presented with active TB at the moment of producing SSP (positive culture for *Mycobacterium tuberculosis*) and 26 with inactive residual TB. This supposes that 0.95% of active TB cases had this complication. In the other 26 cases, there were clinical data and radiographic evidence to support inactive TB, all with extensive residual lesions considered to be the cause of SSP. Of the 47 patients studied, 43 (91.5%) were males and four (8.5%) were females (p<0.001). The mean$\pm$SD age was 46.6$\pm$15.6 (range 23–92) yrs.

Personal antecedents are shown in table 1. Of the total sample, 14 (29.8%) patients had some type of pulmonary antecedent, with 11 standing out as meeting the criteria for COPD and two having more than one antecedent. As stated, 21 (44.7%) patients had a diagnosis of active TB and were undergoing a complete anti-TB drug therapeutic regimen at the time the SSP occurred.

The clinical manifestations presented by the patients are shown in table 2, with dyspnoea (63.8%) standing out, followed by expectoration (55.3%), cough (48.9%) and pleuritic pain (51.1%). Almost half of the patients with SSP secondary to TB presented no thoracic pain.

Radiological diagnosis was performed by simple thoracic radiography in all patients, and confirmed by CT in the other 15 (32%). In 26 (55.3%) cases, SSP was found on the right side, and in 19 (40.4%) cases it was found on the left. In two (4.3%) cases, SSP was bilateral. Apart from the SSP, all patients had radiological alterations, bilateral in 40 (85.1%) patients, limited to the right side in five (10.6%) and to the left in two (4.2%). The observed radiological alterations accompanying SSP are shown in table 2. It is notable that 38 patients (80.8%) had some form of destroyed lung and that 33 (70.2%) presented infiltrates.

With regard to PD as a treatment for SSP, 40 (85%) patients needed one PD, two (4%) needed two, and four (9%) needed three. A decision to wait was made in only one patient, and the evolution of their SSP was favourable during their stay. The mean$\pm$SD stay for PD was 12.9$\pm$11.3 (range 4–58) days. There were no complications from this technique.

In 11 (23%) patients, SSP recurred. Table 2 shows a univariate analysis that was performed in order to study the possible causes of the recurrence, without finding any statistical relationship among clinical variables, analysed radiography and this recurrence. Therefore, not even factors such as suffering active TB, fibrotic TB (radiological infiltrates) or having radiological patterns of destroyed lung were more associated with this recurrence. The treatment for these recurrences was to perform another PD in eight (17%) cases, one of them contralateral, and three patients needed surgery.

An atypical pulmonary segmentectomy was performed in 13 (28%) patients for persistent air leakage after PD (n=10) and

TABLE 1	Univariate analysis[#] between predictive variables and recurrence of tuberculosis (TB) pneumothorax			
Variable	**Cases n**	**Recurrence of pneumothorax**		**p-value**
		Yes	**No**	
All patients	47	11 (23.4)	36 (76.6)	
Sex				
Male	43	10 (90.9)	33 (91.7)	0.670
Female	4	1 (9.1)	3 (8.3)	
Age yrs	47	42.8±14.9	47.7±15.8	0.717
***i.v.* drug abuse**	12	2 (18.2)	10 (27.8)	0.703
Alcoholism	19	5 (45.5)	14 (38.9)	0.737
Smoker	36	7 (63.6)	29 (80.6)	0.256
Social indigence	7	3 (27.3)	4 (11.1)	0.330
Pneumonia	5	1 (9.1)	4 (11.1)	0.668
COPD	11	1 (9.1)	10 (27.8)	0.416
HBV	1	0 (0.0)	1 (2.8)	0.766
HCV	4	1 (9.1)	3 (8.3)	0.670
HIV	3	1 (9.1)	2 (5.6)	0.560
Mediastinal adenopathies	5	2 (18.2)	3 (8.3)	0.332
Pulmonary atelectasis	13	4 (36.4)	9 (25.0)	0.467
Bronchiectasis	17	3 (27.3)	14 (38.9)	0.373
Pleural effusion	6	0 (0)	6 (16.7)	0.181
Caverns	21	6 (54.5)	15 (41.7)	0.505
Active TB	21	5 (45.5)	16 (44.4)	0.610
Incomplete TB treatment	26	6 (54.5)	20 (57.1)	0.575
Pulmonary destruction	25	6 (54.5)	19 (52.8)	0.857
Pulmonary infiltrates	33	9 (81.8)	24 (66.7)	0.464
Pleural thickening	26	5 (45.5)	21 (58.3)	0.505
Bronchopleural fistula	10	3 (27.3)	7 (19.4)	0.430
Granulomas	14	5 (45.5)	9 (25.0)	0.177
Pachypleuritis	24	4 (36.4)	20 (57.1)	0.229
Calcium plaques	10	4 (36.4)	6 (16.7)	0.164
Surgery	13	4 (36.4)	9 (25.0)	0.353

Data are presented as n (%) or mean±SD, unless otherwise stated. COPD: chronic obstructive pulmonary disease; HBV: hepatitis B virus; HCV: hepatitis C virus. [#]: using the Chi-squared test.

TABLE 2	Descriptive analysis: clinical characteristics and radiological findings of the sample studied
Variable	**Cases**
Clinical characteristics	
Active TB	21 (44.7)
Enolism	19 (40.4)
Drug addiction	12 (25.5)
COPD	11 (23.4)
Social indigence	7 (14.9)
Pneumonia	5 (10.6)
HCV	4 (8.5)
HIV	3 (6.4)
HBV	1 (2.1)
Dyspnoea	30 (63.8)
Expectoration	26 (55.3)
Cough	23 (48.9)
Chest pain	24 (51.1)
Fever	20 (42.6)
Bronchopleural fistula	10 (21.7)
Radiological findings	
Infiltrates	33 (70.2)
Pleural thickening	26 (55.3)
Pulmonary destruction	25 (53.2)
Caverns	21 (44.7)
Bronchiectasis	17 (36)
Granulomas	14 (29.8)
Pulmonary atelectasis	13 (27.7)
Pleural calcifications	10 (21.3)
Pleural effusion	6 (12.8)
Mediastinal adenopathies	5 (10.6)

Data are presented as n (%). n=47. TB: tuberculosis; COPD: chronic obstructive pulmonary disease; HCV: hepatitis C virus; HBV: hepatitis B virus.

relapses (n=3). Operations were carried out by axillary thoracotomy in five cases, lateral thoracotomy in seven and videothoracoscopy in one. There were five (38.4%) post-operative complications: persistent air leaks for >5 days in three cases, pneumonia in one case and surgical wound infection in one. There was no post-operative mortality. The mean±SD hospital length of stay was 17.09±17.04 (range 4–73) days. On three occasions, Heimlich valves were placed for persistent air leaks (two post-operatively). Intrahospital mortality was 4% (two patients, in both cases due to severe pneumonia, not SSP).

At the close of the study, 14 (30%) patients had died and 31 (66%) were still alive. Two patients were lost to follow-up. Cause of death was respiratory insufficiency in eight cases, pneumonia in three cases and liver insufficiency in two cases. The cause of death could not be determined for one patient. Table 3 shows a univariate analysis of the multiple factors analysed in relation to long-term survival using the log-rank test. Of the different variables analysed, the only predictors of poor prognosis were the patient's age (HR 1.063, 95% CI 1.026–1.101; p<0.001) (the older the patient, the worse the prognosis) and HIV infection (HR 6.336, 95% CI 1.301–30.867; p=0.009).

The curve of long-term survival for the studied series is shown in figure 1. The average survival was ~11 yrs (135 months, 95% CI 64.7–205.1 months). The probability of survival at 1, 3, 5 and 10 yrs was 95.6, 84.8, 81.5 and 56.8%, respectively.

DISCUSSION

The presence of SP during the evolution of active TB is a well-known situation. However, it has not been sufficiently studied to date. Previous studies have shown a 0.6 [2] to 1.4% [4] possibility of this occurring, which is very similar to the 0.95% found in the present study. It would seem, therefore, that we could estimate ~1% of patients with active TB might present

TABLE 3	Factors related to long-term survival in tuberculosis (TB) pneumothorax[#]				
	Cases[¶] n	Events	Censored	HR (95% CI)	p-value
Total	46	14 (30)	32 (70)		
Age[+] yrs		58.5±17.1	41.3±41.1	1.06 (1.03–1.10)	0.001
Sex					
Male	42	14 (33.3)	28 (66.7)	0.04 (0.00–21.12)	0.102
Female	4	0 (0.0)	4 (100)		
Drug addiction					
Yes	12	1 (8.3)	11 (91.7)	4.95 (0.65–37.96)	0.088
No	34	13 (38)	21 (61.8)		
Alcoholism					
Yes	19	8 (42.1)	11 (57.9)	0.586 (0.20–1.70)	0.320
No	27	6 (22.2)	21 (77.8)		
Smoker					
Yes	36	13 (36.1)	23 (63.9)	0.23 (0.03–1.73)	0.117
No	10	1 (10.0)	9 (90.0)		
Social indigence					
Yes	7	3 (43.0)	4 (57.0)	0.27 (0.07–1.11)	0.052
No	39	11 (28.2)	28 (71.8)		
Pneumonia					
Yes	5	2 (30.0)	3 (60.0)	0.48 (0.11–2.19)	0.335
No	41	12 (29.3)	29 (70.7)		
COPD					
Yes	11	6 (54.5)	5 (45.5)	0.37 (0.12–1.11)	0.064
No	35	8 (22.9)	27 (77.1)		
HBV					
Yes	1	0 (0.0)	1 (100)	20.76 (NC)	0.881
No	45	14 (31.1)	31 (68.9)		
HVC					
Yes	4	1 (25.0)	3 (75.0)	0.526 (0.07–4.24)	0.540
No	42	13 (31.0)	29 (69.0)		
HIV					
Yes	3	2 (66.7)	1 (33.3)	6.34 (1.30–30.87)	0.009
No	43	12 (27.9)	31 (72.1)		
Mediastinal adenopathies					
Yes	5	0 (0)	5 (100.0)	23.16 (0.00–2186)	0.409
No	41	14 (34.1)	27 (65.9)		
Pulmonary atelectasis					
Yes	13	2 (15.4)	11 (84.6)	1.77 (0.39–7.95)	0.453
No	33	12 (36.4)	12 (63.6)		
Pumonary bronchiectasis					
Yes	17	6 (35.3)	11 (64.7)	0.59 (0.20–1.78)	0.347
No	29	8 (27.6)	21 (72.4)		
Caverns					
Yes	21	6 (28.6)	15 (71.4)	0.97 (0.33–2.84)	0.957
No	25	8 (32.0)	17 (68.0)		
Pleural effusion					
Yes	6	2 (33.3)	4 (66.7)	0.62 (0.13–2.91)	0.542
No	40	12 (30.0)	28 (70.0)		
Pulmonary destruction					
Yes	25	9 (36.0)	16 (64.0)	0.34 (0.10–1.12)	0.063
No	21	5 (23.8)	16 (76.2)		

TABLE 3	Continued				
	Cases[¶] n	Events	Censored	HR (95% CI)	p-value
Pleural thickening					
Yes	25	8 (32.0)	17 (68.0)	0.46 (0.15–1.48)	0.185
No	21	6 (28.6)	15 (71.4)		
Bronchopleural fistula					
Yes	10	1 (10.0)	9 (90.0)	3.26 (0.43–24.98)	0.228
No	36	13 (36.1)	23 (63.9)		
Granulomas					
Yes	14	5 (35.7)	9 (64.3)	0.94 (0.31–2.83)	0.905
No	32	9 (28.1)	23 (71.9)		
Pulmonary infiltrates					
Yes	32	10 (31.2)	22 (68.8)	1.05 (0.33–3.38)	0.936
No	14	4 (28.6)	10 (71.4)		
Pachypleuritis					
Yes	24	7 (29.2)	17 (70.8)	0.67 (0.22–2.05)	0.475
No	22	7 (31.8)	15 (68.2)		
Calcium plaques					
Yes	10	3 (30.0)	7 (70.0)	0.85 (0.23–3.10)	0.806
No	36	11 (30.6)	25 (69.4)		
TB active					
Yes	21	5 (23.8)	16 (76.2)	1.04 (0.34–3.15)	0.951
No	25	9 (36.0)	16 (64.0)		
Treatment of TB					
Complete	20	4 (20.0)	16 (80.0)	1.19 (0.36–3.94)	0.772
Incomplete	25	9 (36.0)	16 (64.0)		
Surgery					
Yes	13	1 (7.7)	12 (92.3)	4.38 (0.57–35.60)	0.120
No	33	13 (39.4)	20 (60.6)		
Recurrence					
Yes	11	3 (27.3)	8 (72.7)	0.83 (0.23–3.01)	0.782
No	35	11 (31.4)	24 (68.6)		

Data are presented as n (%) or mean±SD, unless otherwise stated. Event: death during the follow-up period; censored: number of patients who were still alive at the end of the last interval of the follow-up. HR: hazard ratio; COPD: chronic obstructive pulmonary disease; HBV: hepatitis B virus; HCV: hepatitis C virus; NC: not computable. [#]: using the log-rank test, with the exception of age. [¶]: n=46, as one patient was lost during follow-up. [+]: estimated by Cox regression.

this problem [6], a fact that should be taken into account by all national programmes for TB control. It is very likely that many of them go unnoticed, especially in low-income countries, where >85% of the TB patients in the world are living.

It is surprising that SSP as a complication of TB, which has been so accepted for so long, has been the topic of so few publications. There are also very few reports analysing the place that SSP secondary to TB occupies in the total gamut of SSP occurrences. Moreover, the few works that have analysed the relationship between TB and SSP are sometimes conflicting. WILDER et al. [6], in 1962, stated that 78% of SSPs studied were due to TB, which supposes that 1.4% of patients with TB suffer this complication. While the data from the present study are close to those found by WILDER et al. [6] regarding the percentage of patients with TB suffering this type of
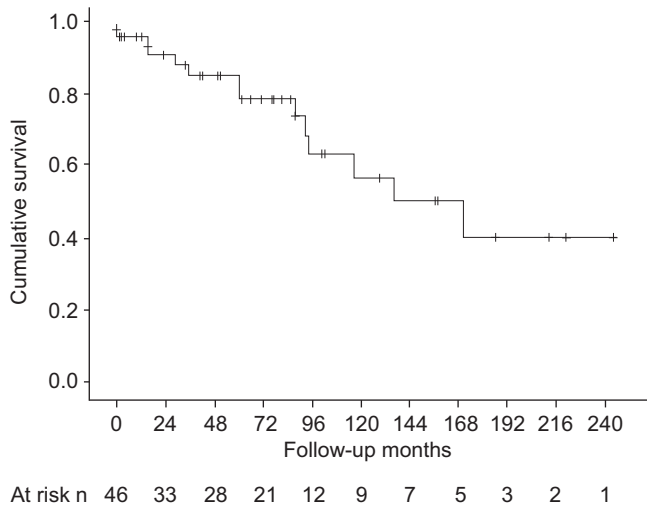
FIGURE 1. Actuarial Kaplan–Meier survival analysis of patients with pulmonary tuberculosis after pneumothorax.

complication, they are very low when the causes producing SSP are analysed. Later publications analysing the aetiology of SSP found that TB was decreasing progressively among causes [9, 10], which was linked to earlier diagnosis of TB cases and the possibility of cure with adequate drug treatment. Nevertheless, in a recently published study by BOTIANU [11], who retrospectively analysed all the cases of SSP at his institution between 1985 and 2004, a marked increase in TB as a cause of SSP during the period of study was found. Despite these discrepancies regarding whether TB is an increasingly or decreasingly frequent cause of SSP, it does seem to be accepted by most authors that TB is one of the most frequent causes of SSP [2, 12, 13], with COPD being the most frequent [14].

In any case, as has been stated, there are very few studies that specifically analyse tuberculous SSP [11, 15–17] and to date, the clinical characteristics of this type of SP, as well as its treatment, evolution, prognosis and survival, have not been sufficiently studied. In fact, a detailed analysis of the clinical and radiological presentation of these SSPs, such as possible factors associated with the recurrence of SSP and the survival of the patients, has yet to be reported. The present study provides new information that can be very useful for the future and should be tested by other authors.

The definition of SSP caused by TB is not a clear concept in the published studies addressing this issue. Many authors consider it in cases of active TB. In our study, we decided to include patients with TB antecedents, although they did not present activity at the time their SSP appeared. It is our opinion that SSP can occur at any moment of the disease, including at the stage of sequelae, because of the associated lung destruction that would be responsible for SSP, as was evident in our patients. In any case, many of the active TB cases probably also have TB sequelae, facilitating the possible SSP.

Radiological signs of destroyed lung are very frequent in this group of patients, reflected by the high percentage (53.2%) found radiographically. Despite these data, the SSP treatment was not especially problematic and a resolution of episodes was obtained in most cases. It was only necessary to place

more than one PD in 13% of cases; recurrences occurred in 23% and the total of patients that required surgery was 28%. These figures can be considered acceptable, and are even lower than these in patients with primary SP [18] and for those recognised in general for SSP [19, 20]. However, one cannot conclude that active TB will worsen the evolution of SSP, because there were no significant differences in this regard. The only inference that can differentiate the management of tuberculous SSP from that of primary SP is the number of days for which the PD was left in prior to indicating surgery for air leakage (12.9±11.3 days), which is usually 5–7 days in primary SP [18]. This is indicative of a more conservative approach in tuberculous SSP. As for surgical interventions, all of these were performed using conventional procedures, with segmental resections and complementary procedures that were, in general, aimed at obtaining a correct pleural abrasion. This suggests that, in our experience, tuberculous SSP has a clinical behaviour that is not especially aggressive and that can be solved, in nearly all cases, with conventional surgical measures.

There are, however, a small number of cases in our series (4%) with a fatal intrahospital evolution. All of those presented with poor general health and serious respiratory insufficiency that lead to death. As with previous cases, evolution during follow-up has shown mortality because of respiratory insufficiency, but without demonstrating significant differences in survival. It has been related only to the patients' age and the presence of HIV.

The present study analysed a group of patients affected with SSP and TB in detail, allowing a better characterisation of this type of patient. There are, however, some limitations to this study, which come from its retrospective character and small number of cases, although it has the largest number of cases that have been studied so far.

We conclude that SSP in patients with TB occurs especially in cases that demonstrate destroyed lung. PD is usually a good response, with only a small number of cases that require more than one PD and/or surgery, although it requires a high number of days on PD, a longer hospital stay and mortality in some cases of severe pulmonary affectation. Surgical treatment is standard, in general, and does not require major surgical manoeuvres. The survival of this group of patients is dictated more by age and the presence of HIV than by TB.

STATEMENT OF INTEREST
None declared.

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