presentamos el caso de una señora de 62 años, sin hábitos tóxicos, diagnosticada de fibromialgia y osteoporosis con infección por herpes zoster torácico en 2011 apareciendo como complicación tardía una neuralgia postherpética. Estaba en seguimiento por la Unidad del dolor, desde 2012 había recibido diferentes tratamientos médicos sin éxito, incluyendo un bloqueo nervioso con anestésicos locales y esteroides en el 2015. En abril del 2016 se inicia tratamiento con radiofrecuencia intercostal convencional, en la que se estimula el área dolorosa sobre el 9.º espacio intercostal derecho a 80° durante 90 seg, sin complicaciones. Se realiza una segunda radiofrecuencia sin incidencias a las 12 semanas y en diciembre se lleva a cabo una tercera radiofrecuencia convencional sobre el 5.º espacio intercostal. En esta última sesión de radiofrecuencia la paciente presenta disnea, taquicardia a 110 lpm e hipotensión, por lo que se realiza una radiografía de tórax urgente en la que se observa un neumotórax derecho (fig. 1). Se coloca un drenaje pleural tras el cual se observa la reexpansión completa del pulmón derecho. La paciente es dada de alta de neumología a las 48 h sin complicaciones.

La neuralgia postherpética por lo general responde a la farmacoterapia, debiéndose emplear antes de intentar cualquier intervención. Los casos refractarios pueden ser tratados con procedimientos mínimamente invasivos no exentos de riesgos como el bloqueo nervioso, tras el cual en un 0,09% de los pacientes se produce un neumotórax, cifra que asciende al 0,42% si se realiza de forma rutinaria a todos los pacientes una radiografía de tórax[12], sin embargo esta no es una complicación habitual de las técnicas de radiofrecuencia donde series más amplias de hasta 96 pacientes en las que fue usada para el tratamiento de la neuralgia postherpética no fue descrito ningún neumotórax[9].

La radiofrecuencia es una técnica intervencionista mínimamente invasiva que ofrece una alternativa en el manejo del dolor crónico y está siendo cada vez más utilizada en los últimos años en las unidades de dolor crónico.

Dada la localización habitual de la neuralgia postherpética en la región torácica junto al desarrollo de nuevas técnicas para el control del dolor obliga a tener en cuenta posibles complicaciones no contempladas inicialmente, como en el caso de nuestra paciente que presentó un neumotórax en un pulmón sano. El manejo de estos casos comienza por la sospecha clínica, sobre todo en casos con factores de riesgo. El tratamiento dependerá del tamaño del neumotórax. En el caso de nuestra paciente, con repercusión hemodinámica, el drenaje fue colocado de forma urgente.

Todo ello obliga a pesar de la baja incidencia de estas complicaciones a disponer de los medios en estas unidades para su diagnóstico y proceder en el caso de que se produzcan.

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https://doi.org/10.1016/j.arbres.2017.05.026
0300-2896/

Effect of Isoniazid Resistance on the Tuberculosis Treatment Outcome

Efecto de la resistencia a la isoniazida en el resultado del tratamiento de la tuberculosis

Dear Director,

Tuberculosis (TB) remains a serious public health problem, and about one-third of world's population has active or latent TB. In Europe, there are 49 new cases and 7 deaths from TB every hour.[1] In Portugal, the incidence has been decreasing in recent years, and in 2014 the annual incidence was 20/1,00,000.[2]

Drug-resistant *Mycobacterium tuberculosis* has become a major threat to the control of TB and, among all first-line drugs, resistance is greatest to isoniazid (INH).[3,4] In Portugal, INH resistance was 10.5% in 2014 among TB cases in whom susceptibility testing was performed.[2] In fact, there has been an increasing resistance to INH, despite the decreasing number of TB cases.[2]

INH is a first-line anti-TB drug because of its potent early bactericidal activity against rapidly dividing cells.[3,5] However, treatment of active TB requires multiple anti-TB drugs along with INH to prevent selection and emergence of a drug-resistant population of *M. tuberculosis*. According to current World Health Organization (WHO) recommendations, INH mono-resistant TB should be treated with 6–9 months of rifampicin, ethambutol, and pyrazinamide, plus or minus a fluoroquinolone.[6] These are also the current treatment guidelines in Portugal.

TB is a notifiable disease in Portugal, so clinicians report all cases to National-Tuberculosis-Surveillance-System (SVIG-TB) that has data on patient demographics, comorbidities, risk behaviors, and clinical, radiological, and microbiological information, as well as treatment outcomes.[7]

Table 1
Demographic and clinical characteristics of enrolled TB patients.

Variable	Total (n = 7345)	INH mono-resistant (n = 242)	Drug-susceptible (n = 7103)	p-Value
Male	5013 (68%)	160 (66%)	4853 (68%)	0.512
Age (years), median (IQR)	44 (24)	44 (24)	44 (23)	0.713
Country of origin				
Portugal	6099 (83%)	191 (79%)	5908 (83%)	
Other country	1246 (17%)	51 (21%)	1195 (17%)	0.100
HIV positive	796 (11%)	18 (7%)	778 (11%)	0.104
Alcohol use	1077 (15%)	36 (15%)	1041 (15%)	1.000
IV-drug use	535 (8%)	11 (8%)	524 (8%)	0.116
Other drug use	655 (9%)	14 (6%)	641 (9%)	0.094
Correctional facility residence	130 (2%)	1 (0.4%)	129 (2%)	0.165
Homeless	134 (2%)	4 (2%)	130 (2%)	1.000
Clinical history				
Diabetes	479 (6%)	16 (7%)	463 (6%)	1.000
Silicosis	101 (1%)	2 (1%)	99 (1%)	0.642
Lung cancer	65 (1%)	0	65 (1%)	0.252
Other cancer	205 (3%)	7 (3%)	198 (3%)	1.000
Hepatic disease	334 (4%)	10 (4%)	324 (5%)	0.874
COPD	229 (3%)	3 (1%)	226 (3%)	0.128
X-ray				
Non-cavitary TB	2518 (40%)	84 (41%)	2434 (40%)	
Cavitary TB	3559 (57%)	114 (56%)	3485 (57%)	
Normal	159 (3%)	5 (3%)	154 (3%)	0.933
Pulmonary TB	6510 (89%)	210 (87%)	6300 (89%)	0.411
Other localization TB	835 (11%)	32 (13%)	803 (11%)	
Previous treatment	610 (8%)	17 (7%)	593 (8%)	0.538
Positive initial microscopy	4812 (69%)	154 (66%)	4658 (69%)	0.494
Positive initial culture	6819 (97%)	221 (97%)	6598 (97%)	1.000
Toxicity during treatment	158 (3%)	7 (3%)	151 (3%)	0.579
Outcome				
Successful (n = 7185)	6385 (89%)	210 (90%)	6175 (89%)	
Unsuccessful (n = 7185)	800 (11%)	24 (10%)	776 (11%)	0.743
Death (n = 7185)	461 (6%)	14 (6%)	447 (6%)	0.889
Treatment duration (days), median (IQR)	254 (104)	279 (94)	251 (104)	<0.001

TB: tuberculosis; INH: isoniazid; IQR: interquartile range; HIV: human immunodeficiency virus; IV: intravenous; COPD: chronic obstructive pulmonary disease.

The objectives of this study were identify factors associated with INH mono-resistance, compare treatment outcomes of INH mono-resistant patients with drug-susceptible patients and understand the causes of unsuccessful treatment among INH mono-resistance TB cases.

To achieve the goals, data from Portuguese SVIG-TB were retrospectively analyzed from 1/January/2008 to 31/December/2014. INH mono-resistant cases were compared with drug-susceptible cases.

Culture-confirmed cases tested against first-line anti-TB drugs were included. INH mono-resistant TB cases were defined as having resistance to INH, but susceptibility to all other first-line anti-TB drugs. Drug-susceptible cases were those that had documented sensitivity to INH, rifampin, pyrazinamide, and ethambutol.

Susceptibility testing was carried out according to international standards; the method of proportions (liquid medium in the MGIT 960 system) was used to determine susceptibility to anti-tuberculosis drugs on: isoniazid (0.1 μg/ml), rifampicin (1 μg/ml), ethambutol (5 μg/ml), pyrazinamide (100 μg/ml) and streptomycin (1 μg/ml).

The WHO standard definitions were used for treatment outcomes.[6] Unsuccessful treatment includes failure, death during treatment and default. Cured patients and those with completed treatment were defined as treatment success. Cases with unknown outcomes ("in treatment" and "transferred") were excluded.

Data were summarized by descriptive statistics, consisting of absolute (relative) frequencies or median (minimum–maximum), according to nature of variables.

Comparisons of demographic and clinical variables between two groups used the Chi-squared test (or Fisher's test, as appropriate) for categorical variables and the Mann–Whitney U-test for continuous variables. A univariate analysis evaluated the effect of demographic characteristics and risk factors on treatment outcome for INH mono-resistance TB, through simple logistic regression.

Statistical analyses were performed with SPSS version 18.0 (PASW Statistic 18). Significance level was set at 0.05.

This study used surveillance data, with no possibility of linking patient records to patient personal data, so ethical approval was considered unnecessary.

Between 2008 and 2014, 18,429 TB cases were reported to SVIG-TB, from them 12,031 had culture confirmation and 10,588 of them were tested for sensitivity to first-line anti-TB drugs. According to drug susceptibility test, 7103 cases were susceptible to all first-line anti-TB drugs and 242 cases were INH mono-resistant. 3243 cases were excluded: 657 cases had resistance to rifampin and/or pyrazinamide and/or ethambutol and/or streptomycin, and 2061 had susceptibility to isoniazid and rifampin and no results for pyrazinamide, ethambutol and streptomycin.

Table 1 shows the characteristics of INH mono-resistant, drug-susceptible, and all eligible TB cases. In all groups, median patient age was 44 years, men were more likely to be cases than women, and most patients were born in Portugal. Most of studied clinical factors were not statistically different between INH mono-resistant and drug-susceptible groups. Although the two groups had no significant difference in treatment outcome and toxicity during treatment, the median treatment duration was longer in INH mono-resistant group (279 *vs.* 251 days, $p < 0.001$).

Several previous studies identified risk factors for INH mono-resistant TB, like previous TB treatment,[5,8] younger age,[9,10] foreign birth,[10,11] and various social factors.[11] However, as in Birmingham study,[12] no predictive factors for INH mono-resistant TB were found.

Table 2
Univariate analysis of the risk (odds ratio, OR) for unsuccessful treatment of TB patients with INH mono-resistant disease.

Variable	Successful ($n=210$)	Unsuccessful ($n=22$)	Unadjusted OR (95% CI)	p-Value
Age, median (IQR)	44 (23)	52 (43)	1.04 (1.01–1.06)	0.005
Sex				
Female	73 (35%)	7 (32%)	1.0	
Male	137 (65%)	15 (68%)	1.14 (0.45–2.93)	0.782
Alcohol use				
No	174 (86%)	14 (74%)	1.0	
Yes	29(14%)	5 (26%)	2.14 (0.72–6.40)	0.172
IV-drug use				
No	198 (97%)	16 (80%)	1.0	
Yes	6 (3%)	4 (20%)	8.30 (2.11–32.27)	0.002
Other drug use				
No	194 (94%)	17 (90%)	1.0	
Yes	12 (6%9	2 (10%)	1.90 (0.39–9.21)	0.424
Homeless				
No	203 (99%)	19 (95%)	1.0	
Yes	2 (1%)	1 (5%)	5.34 (0.16–61.66)	0.179
HIV status				
Negative	198 (94%)	17 (77%)	1.0	
Positive	12 (6%)	5 (23%)	4.85 (1.53–15.40)	0.007
Previous TB treatment				
No	195 (93%)	21 (96%)	1.0	
Yes	15 (7%)	1 (4%)	0.62 (0.08–4.92)	0.650
Site of TB				
Extrapulmonary	27 (13%)	4 (18%)	1.0	
Pulmonary TB	183 (87%)	18 (83%)	0.66 (0.21–2.11)	0.488
Toxicity during treatment ($n=196$)				
No	173 (98%)	16 (80%)	1.0	
Yes	3 (2%)	4 (20%)	14.42 (2.96–70.14)	0.001

TB: tuberculosis; INH: isoniazid; IQR: interquartile range; IV: intravenous; HIV: human immunodeficiency virus; TB: tuberculosis.

We did find that INH mono-resistant cases had a longer mean treatment duration, in agreement with some previous studies.[5,8] For example, a study in Georgia[4] reported that the extended length of regimen may be associated with reduce adherence. A study in San Francisco[5] suggested that new short-course treatment regimens are needed because of high incidence of drug toxicity. In contrast, a study in southern Mexico reported that treatment outcomes were similar for patients receiving a 6 month course and an extended course.[13]

In contrast to some studies,[4,13,14] but in agreement with San Francisco study,[5] we found no difference in the outcome of the two groups patients. This is in agreement with a previous report that INH mono-resistant treatment regimens have success rates of 95% or more.[15]

The treatment outcome of 232 INH mono-resistant cases were also analyzed (Table 2). Following the exclusion criteria, 10 of the INH mono-resistant cases without treatment results were excluded. The profile of these patients was similar to studied cases: median age was 46 years, 8 (80%) were male, 1 (10%) was HIV-positive, and 1 (12.5%) was an IV-drug user.

A univariate analysis showed that age (OR = 1.04; 95%CI = 1.01–1.06; $p=0.005$), intravenous (IV)-drug use (OR = 8.30; 95%CI = 2.11–32.27; $p=0.002$), HIV-positivity (OR = 4.85; 95%CI = 1.53–15.40; $p=0.007$) and toxicity during treatment (OR = 14.42; 95%CI = 2.96–70.14; $p=0.001$) were significantly associated with unsuccessful treatment outcome.

The Georgia study[4] reported treatment outcomes were worse for older patients among those who had INH mono-resistance. The Tanzania study[14] reported similar results, and also that HIV-infected individuals were more likely to experience unsuccessful treatment. The increased toxicity during treatment may be related to longer treatment duration, as described in San Francisco study.[5]

The main limitation of our study was the low number of unsuccessful treatments ($n=22$), which made multivariate analysis unfeasible.

Our findings reinforce the need for susceptibility testing and monitoring the treatment of INH mono-resistant cases, especially among the elderly, cases with HIV co-infection, IV drug users, and cases with toxicity during treatment.

Acknowledgment

Rita Gaio was partially supported by CMUP (UID/MAT/00144/2013), which is funded by FCT (Portugal) with national (MEC) and European structural funds (FEDER), under the partnership agreement PT2020.

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https://doi.org/10.1016/j.arbres.2017.06.009
0300-2896/

Actinomicosis pulmonar en paciente con neumonía eosinófila crónica en tratamiento con omalizumab

Pulmonary Actinomycosis in a Patient with Chronic Eosinophilic Pneumonia treated with Omalizumab

Estimado Director:

La actinomicosis pulmonar es un diagnóstico muy infrecuente. Se trata de una infección crónica, no contagiosa y supurativa, causada por microorganismos del género *Actinomyces*. Son bacilos gram positivos, de forma filamentosa y ramificada[1]. El agente etiológico más frecuente es el *Actinomyces israelii*. Se han identificado 8 especies patógenas para el hombre y una de ellas es el *Actinomyces odontolyticus (A. odontolyticus)*, que muy raramente produce infecciones pulmonares. La actinomicosis pulmonar se produce, principalmente, debido a la aspiración de estos organismos desde la cavidad oral. Presentamos el caso de un paciente con asma grave y neumonía eosinófila crónica (NEC), con diagnóstico de actinomicosis pulmonar.

Se trata de un varón de 43 años con asma grave, que tuvo varios ingresos por infiltrados pulmonares que se etiquetaron de NEC (diagnóstico realizado por lavado broncoalveolar [LBA] con el 52% de eosinófilos). Estuvo en tratamiento con corticoides orales durante un año, y al reducirse a 10 mg/día, presentó nueva recidiva. Se descartó síndrome hipereosinofílico mediante estudio hematológico. Se decidió iniciar tratamiento con omalizumab como ahorrador de corticoides, dado el importante componente alérgico asociado que presentaba el paciente (IgE 1.089 KU/l). En las analíticas de control se apreciaba eosinofilia periférica persistente (pico máximo: 50,3%-11.300 mm^3) y ANCA repetidamente negativos. Desde el inicio del tratamiento con omalizumab tuvo buena respuesta, sin recidivas de asma ni de infiltrados pulmonares, lo que permitió la reducción progresiva de los corticoides hasta suspenderlos.

Tras 18 meses del inicio de omalizumab y 12 meses después de haber suspendido el tratamiento con corticoides, el paciente consultó por síntomas catarrales y disnea. En la radiografía (Rx) de tórax se apreciaban infiltrados pulmonares y pequeño derrame pleural izquierdo. En la analítica destacaba eosinofilia (38%-3.880 mm^3). Se realizó una broncoscopia en la que se observaba a nivel segmentario una mucosa con lesiones de aspecto granular, sobreelevadas, de fondo blanquecino con punteado eritematoso, de donde se tomaron biopsias (fig. 1A). En estas se apreciaba mucosa con metaplasia escamosa e intenso infiltrado inflamatorio agudo y crónico en corion. El recuento celular del LBA mostraba el 0% de eosinófilos. Además, en la broncoscopia pudimos ver como el segmento 8 izquierdo estaba obstruido por un material blanquecino muy denso y filante, que también se biopsió (fig. 1B). Ante estos hallazgos, solicitamos una tomografía computarizada de tórax que demostró la existencia de un infiltrado en el segmento 8 izquierdo (fig. 1C), que correspondía con la imagen endoscópica alterada y biopsiada, y en la que posteriormente creció *A. odontolyticus* en el cultivo. La identificación del microorganismo se realizó mediante espectrometría de masas, utilizando la tecnología MALDI-TOF (desorción/ionización mediante láser asistida por matriz), con un *score* de 1.976. Tras estos resultados se inició tratamiento con amoxicilina-clavulánico durante 6 meses, y corticoides (1 mg/kg). El paciente se mantuvo estable, sin agudizaciones y con Rx de tórax a los 2 meses del ingreso, con resolución del infiltrado.

Dos meses después de finalizar el tratamiento antibiótico, el paciente comenzó con opresión centrotorácica y disnea. Se realizó Rx de tórax, objetivándose un nuevo infiltrado en lóbulo inferior izquierdo. Realizamos nueva broncoscopia, en la que se apreciaban las mismas lesiones que en la prueba anterior, de donde se volvieron a tomar biopsias. En esta ocasión en la biopsia se observaba intensa inflamación de predominio eosinofílico, y en el LBA de nuevo el 0% de eosinófilos. Nuevamente creció *A. odontolyticus* en el cultivo. Iniciamos tratamiento con ceftriaxona hasta completar un mes, seguido de amoxicilina durante 12 meses. Ante la presencia de eosinófilos en sangre (35%-2.860 mm^3) y de intenso infiltrado eosinofílico en biopsia bronquial se inició nuevo ciclo de corticoides. El paciente presentó buena evolución clínica y continúa en seguimiento en nuestra consulta.

A. odontolyticus es un comensal habitual, en personas sanas, de la flora oral, gastrointestinal y genital femenina[2]. Los factores predisponentes para desarrollar la enfermedad son alcoholismo crónico, mala higiene bucal, diabetes mellitus y enfermedad