

3. Tuberculosis care with TB-HIV co-management: Integrated Management of Adolescent and Adult Illness (IMA). "WHO/HTM/HIV/2007.01".
4. National Institute for Health and Clinical Excellence. Clinical Diagnosis and Management of Tuberculosis, and Measures for Its Prevention and Control. London: National Institute for Health and Clinical Excellence; 2006.
5. American Thoracic Society, Center for Disease Control and Prevention, and Infectious Diseases Society of America. Treatment of tuberculosis. MMWR Recomm Rep. 2003;52:1-77.
6. Nahid P, González LC, Rudoy I, De Jong BC, Unger A, Kawamura LM, et al. Treatment Outcomes of Patients with HIV and Tuberculosis. Am J Respir Crit Care Med. 2007;175:1199-206.

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Guillain-Barre Syndrome as an Initial Manifestation of Small Cell Lung Carcinoma

Síndrome de Guillain Barré como forma de debut de un carcinoma microcítico de pulmón

To the Editor:

Lung neoplasia can debut as paraneoplastic neurological syndromes, among these being Eaton-Lambert syndrome, encephalomyelopathy, cerebellar degeneration, subacute sensory neuropathy and autonomic neuropathy¹ but rarely has Guillain Barré syndrome been described. It is an autoimmune demyelinating polyneuropathy that affects the peripheral nervous system and frequently presents as ascending paralysis together with complete loss of deep tendon reflexes.² Guillain Barré syndrome as an initial manifestation of lung neoplasia is quite exceptional.

Our patient was a 45-year-old male who smoked 65 packs/year and was diagnosed with schizophrenia. He was admitted for anorexia, asthenia, low-grade fever and weight loss over the previous three months, in addition to dyspnea upon mild effort. Two months before, he had begun with symptoms of ascending generalized progressive muscular weakness, and was incapable of walking during the prior week. Physical examination discovered abolition of the vesicular murmur in the upper 2/3 of the right hemithorax, intense tetraparesis (proximal 1/5 and distal 2/5) with areflexia and generalized amyotrophy with maintained sensitivity. Posteroanterior and lateral chest radiograph (fig. 1A) showed a well-defined mass occupying the right upper lobe (RUL). Thoracic computed axial tomography was ordered (fig. 1B), which confirmed the presence of a lung mass of 15 × 11 cm in the RUL, with infiltration of the right pulmonary artery, tracheal compression and pathological

adenopathies in the mediastinum. Bronchoscopy revealed an obstruction of the RUL bronchus with tumor infiltration. Bronchoaspiration, brushing and bronchial biopsy were compatible with small-cell carcinoma. Electromyogram showed neurographic findings compatible with Guillain Barré syndrome while the anti *Hu*, anti *Yo*, anti *Ri*, and anti calcium channel antibodies were negative. Treatment was initiated with chemotherapy and intravenous immunoglobulin and the neurological symptoms progressively improved for 15 days. The patient died three months later due to tumor progression.

Patients with cancer can develop signs and symptoms of peripheral nervous dysfunction, although more frequently related to chemotherapy; in some cases, neuron antigens expressed by the tumor stimulate an immune response characterized by T-cells, antibodies or both, that attack not only the tumor but also the nervous tissue.³ The Guillain Barré syndrome is a very infrequent initial manifestation and is rarely associated with lung carcinoma. To our knowledge, just one case has been published in Spain associating squamous carcinoma, and in the literature 3 sporadic cases of small-cell carcinoma are described. Although infrequent, the syndrome is estimated to appear in 1% of cancer patients. Non-Hodgkin lymphoma is the cancer that is most frequently associated, but the relationship with other malignant diseases is less clear.⁴ In a series of 435 patients with Guillain Barré syndrome, 9 developed neoplasia in the following 6 months, 3 of which were non-small cell lung cancer while the others were chronic lymphocytic leukemia, non-Hodgkin lymphoma, kidney cancer, esophageal cancer, cancer of the vocal chords and metastatic disease of unknown etiology.⁵ The prognosis of patients with neoplasia and Guillain Barré was poorer, with higher mortality than those with Guillain Barré syndrome alone.⁶

We conclude that, given a patient with Guillain Barré syndrome, lung cancer should be included in the etiological study.

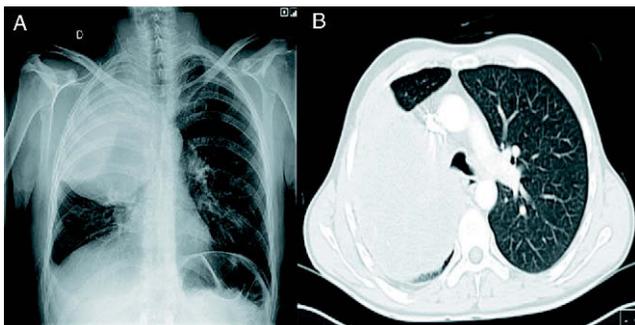


Figure 1. A) Posteroanterior chest radiography showing a large mass in the right upper lobe. B) Thoracic computed tomography of the patient.

References

1. Silvestri GA, Jett J. Bronchogenic carcinoma. In: Murray and Nadel's, editors. Textbook of respiratory medicine. Philadelphia: Elsevier Saunders; 2000; 3rd ed. p. 1357-82.
2. Alter M. The epidemiology of Guillain-Barré syndrome. Ann Neurol. 1990;27:S7-12.
3. Darnell RB, Posner JB. Paraneoplastic syndromes involving the nervous system. N Engl J Med. 2003;349:1543-54.
4. Vallat JM, De Mascarel HA, Bordessoule D, Jauberteau MO, Tabaraud F, Gelot A, et al. Non-Hodgkin malignant lymphomas and peripheral neuropathies: 13 cases. Brain. 1995;118:1233-45.
5. Vigliani MC, Magistrello M, Polo P, Mutani R, Chiò A, et al. Piemonte and Valle d'Aosta Register for Guillain-Barré Syndrome. Risk cancer in patients with Guillain-Barré syndrome. J Neurol. 2004;251:321-6.
6. Rudnicki SA, Dalmau J. Paraneoplastic syndromes of the peripheral nerves. Curr Opin Neurol. 2005;18:598-603.

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Preliminary Study of the Effect on the Lungs Due to the Consumption of Cocaine Base Paste

Estudio preliminar de la repercusión pulmonar por consumo de pasta base de cocaína

To the Editor:

In the last ten years, there has been a significant increase in the consumption of cocaine base paste (CBP) in Uruguay, Chile and Argentina, which has become a real public health problem given its biological, psychological and social repercussions. Cocaine paste is an intermediate product in the preparation of cocaine chlorohydrate and it has a variable percentage of cocaine, other alkaloids, impurities and fillers.¹ It is smoked using either tin cans or plastic pipes, or in a cigarette with marijuana or tobacco. Its composition is different from that of crack, another form of smokable cocaine that is considered to have a higher purity than CBP. Based on reports of clinical cases analyzing the consumption multi-drug abuse, authors have related the use of crack, widely consumed in the US and Europe, with asthma exacerbation, bronchitis, bronchiolitis, pulmonary edema, alveolar hemorrhage, eosinophilic pneumonitis and barotrauma.^{2,3} There are, however, no studies that characterize the respiratory toxicity of cocaine paste.

We present a preliminary study with the aim of determining the main clinical manifestations and lung complications related to CBP abuse. Ours is a single-center observational retrospective descriptive study of CBP users admitted to the "Portal Amarillo" Center for detoxification for a period of three months. Excluded from the study were patients who had been smoking CBP for less than one year and those with previous lung disease or human immunodeficiency virus (HIV) infection. Data were taken from their medical history and patients underwent: chest radiography, thoracic high-resolution computed tomography (HRCT), respiratory function and post-effort testing, Doppler ultrasound and hemogram. None of the patients presented clinical symptoms of cocaine overdose. Seventeen male patients were studied, with a mean age of 26. Multi-drug use was a factor in all cases, with current associated use of marijuana and tobacco. CBP consumption time ranged from 2 to 6 years, and 82% of the subjects had a history of cocaine chlorohydrate use. All patients presented respiratory symptoms; prevalent clinical manifestations were: cough with carbonaceous expectoration, dyspnea and bronchospasm. Chest radiography revealed bilateral perihilar radiopaque infiltrates in every case. On CT, 6 patients presented nodular images occupying the air space, with areas of air-trapping (in two cases with obstruction of the small airway). Respiratory function tests and Doppler ultrasound were normal in all patients. Three patients presented eosinophilia.

Clinical manifestations in CBP users are frequent and do not seem to be related with specific lung disease. According to the literature, these symptoms have been reported in crack users a few

hours after the last consumption,³ while in these users there was an abstinence period of two to three weeks. Carbonaceous expectoration is a result of the inhalation of impurities and products from the combustion of this type of drug use.⁴ The radiological findings were consistent with chronic tracheobronchitis and pneumonitis, associated with eosinophilia in three cases, which may suggest an immunological response as described in "crack lung".⁵ CT alterations demonstrated the chronic irritation of cocaine and products of its combustion in the lower respiratory apparatus. Although many of these findings can be explained by concomitant use of tobacco and marijuana, Tashkin et al. hypothesize that crack consumption could aggravate the damage caused by tobacco habit.⁶ We found no pulmonary hypertension, described in cocaine users, nor any respiratory function alterations, coinciding with studies in crack users.³ This preliminary study reveals similarities in lung repercussions of CBP with the crack study, both in the context of multi-drug use and in cases reported. "CBP lung" should be evaluated and characterized with a greater number of patients, selecting user groups that do not consume either tobacco or marijuana, and including other studies such as bronchoalveolar lavage, evaluation of the pulmonary alveolar-capillary permeability and local immunological response.

References

1. Jeri FR. Coca paste smoking in some Latin American countries: a severe and unabated form of addiction. *Bull on Narc.* 1984;XXXVI:15-31.
2. Terra Filho M, Yen CC, de Paula Santos U, Romero Muñoz D. Pulmonary alterations in cocaine users. *Sao Paulo Med J.* 2004;122:26-31.
3. Haim DY, Lippmann ML, Goldberg SK, Walkenstein MD. The pulmonary complications of crack cocaine: a comprehensive review. *Chest.* 1995;107:233-40.
4. Klinger JR, Bensadoun E, Corrao WM. Pulmonary complications from alveolar accumulation of carbonaceous material in a cocaine smoker. *Chest.* 1992;101:1171-3.
5. Kissner DG, Lawrence WD, Selis JE. Crack lung pulmonary disease caused by cocaine abuse. *Am Rev Respir Dis.* 1987;136:1250-2.
6. Tashkin DP, Simmons MS, Coulson AH, Clark VA, Gong H. Respiratory effects of cocaine freebasing among habitual users of marijuana with or without tobacco. *Chest.* 1987;92:638-44.

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