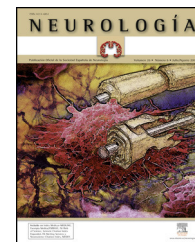




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LETTERS TO THE EDITOR

Cerebral abscess due to *Rhodococcus equi* with pseudotumour presentation in an immunocompetent patient[☆]

Absceso cerebral por *Rhodococcus equi* con comportamiento seudotumoral en paciente inmunocompetente

Dear Editor,

Infections by *Rhodococcus* species are very rarely seen in humans.^{1,2} We present the case of a 52-year-old Spanish-born male construction worker with a history of hypertension and moderate heart failure and no history of habitual contact with animals. Over a period of 2 weeks, the patient had experienced progressive symptoms of disorientation in time and space, mobility limitations, and language difficulties. He was transported to our hospital's emergency department for those reasons. Physical examination upon admission revealed disorientation in time and space, frank bradypsychia, mild right brachial paresis, motor aphasia, apathy, and aboulia. The general examination, including cardiopulmonary auscultation, yielded normal results. Vital signs recorded by the emergency department were also normal (blood pressure, heart rate, temperature, and oxygen saturation). Emergency laboratory analyses and chest radiography showed no abnormalities. An emergency CT study (Fig. 1) revealed a left frontal–temporal mass 45 mm in diameter. The mass was cavitated, with a small haemorrhagic area and perilesional oedema. Since doctors suspected a malignant tumour, they began corticosteroid treatment with dexamethasone dosed at 8 mg every 8 h, intravenous analgesics, and low molecular-weight heparin at a prophylactic dose. The blood test performed as part of the study delivered the following results. Complete blood count (haemoglobin 16 g/dL, WBC count 9000/mm³, neutrophils 70%, platelets 282 000/mm³); coagulation study (normal); cellular immunity study (CD4 lymphocytes 45%, CD8 lymphocytes 24%, CD4–CD8 ratio 1.9); immunoglobulins test

(normal); negative for HIV. The magnetic resonance study (Fig. 2) using normal sequences (T1, T2, and proton-density weighted) revealed a lesion that was hypointense in T1 and hyperintense in T2. It was located in the left frontal-temporal area and surrounded by an extensive hyperintense perilesional halo with collapsed sulci and slight subfalcine herniation. Contrast uptake was ring-shaped; this pattern, together with clinical data, was compatible with highly malignant glioma. Diffusion sequence and spectroscopy results are not available since we did not have access to those techniques.

Focal motor signs and bradypsychia improved partially in the first few days of hospitalisation. One week later, the patient experienced sudden-onset chest pain, associated dyspnoea, and low oxygen saturation. Based on a suspected diagnosis of pulmonary embolism, we performed an emergency thoracic CT study that revealed multiple bilateral pulmonary nodules that were cavitated. These data, plus the patient's medical history and the previous laboratory analyses and imaging studies, suggested a secondary disease. The patient required orotracheal intubation and was moved to the intensive care unit. In the following days, his condition deteriorated rapidly and he developed a fever. Doctors took blood and urine cultures, bronchoalveolar lavage (BAL) sample, and a tracheal aspirate culture. The brain lesion was biopsied and the analysis revealed cystic tissue containing unrecognisable cellular material. The microbial study identified *Rhodococcus equi*. The BAL sample also tested positive for *Nocardia* and *Aspergillus*. Likewise, the tracheal aspirate culture showed nocardiform bacteria and the blood culture confirmed the presence of *Rhodococcus equi*. Intravenous antibiotic treatment with amikacin and meropenem elicited no improvement. Later neuroimaging studies showed no changes and the following chest CT confirmed the presence of necrotising invasive aspergillosis. The patient died several days later with a final diagnosis of adult respiratory distress caused by a mixed infection (*Aspergillus/Rhodococcus/Nocardia*) and cerebral abscess caused by *Rhodococcus equi*.

Brain abscesses are suppurative processes of the cerebral parenchyma with high morbidity and mortality rates.³ They are slightly more prevalent in men and most often occur during the 6th and 7th decades of life. A compromised immune system and ENT surgery or neurosurgery are known risk factors for these abscesses. The causal agents may be aerobic or anaerobic, although studies are most

[☆] Please cite this article as: Velázquez Benito A, Tejero Juste C, Pérez Lázaro C, Santos Lasasa S. Absceso cerebral por *Rhodococcus equi* con comportamiento seudotumoral en paciente inmunocompetente. Neurología. 2013;37:522–524.

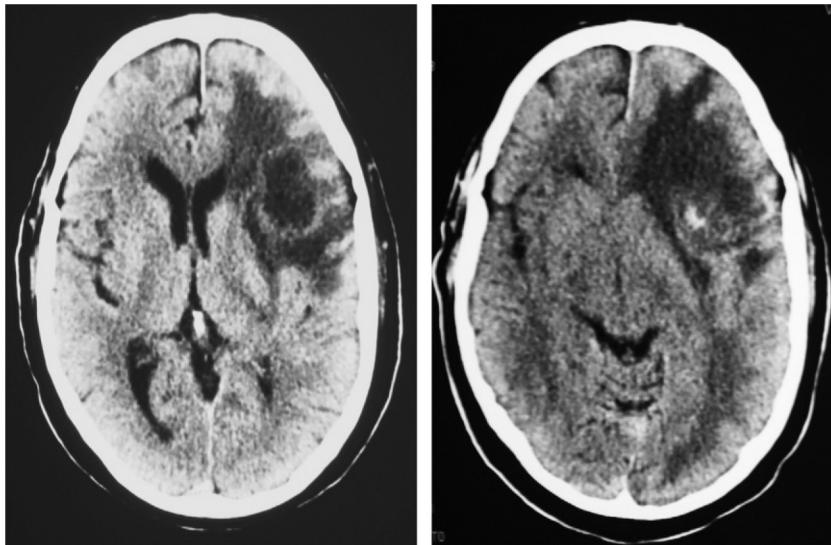


Figure 1 Brain CT without contrast showing a space-occupying lesion in the left frontal–temporal area with significant perilesional oedema and a midline shift.

likely to identify mixed polymicrobial flora. In cases of weakened immune system, the microbes may be uncommon (opportunistic micro-organisms). *Rhodococcus equi* is one such micro-organism.^{4,5} This environmental zoonotic pathogen belongs to the *Nocardiaceae* (along with the *Nocardia* and *Gordona* genera, a fact which explains the presence of the multiple types of micro-organisms in the patient's respiratory system samples). The most common infection route is respiratory contamination after contact with carrier animals. The prevalence of infection with this microbe is low, but it has risen as the HIV infection rate has increased. The most reliable diagnostic tests are sputum sample and blood culture. In humans, *Rhodococcus equi*^{6,7} produces cavitated lesions containing abundant

intracellular bacteria. Its main manifestation is insidious respiratory infection; less frequently, it may infect other organs, including the brain, liver, kidneys, or eyes (endophthalmitis). There is no consensus regarding what treatment should be used, or the treatment duration. The most common recommendation^{5,8} is to administer erythromycin with vancomycin or rifampicin during at least 3 weeks. The literature describes only a few cases, but infection with *Rhodococcus equi* may also present in immunocompetent patients, as in our case. Early diagnosis and effective treatment are therefore crucial to minimise the complications of this disease.



Figure 2 Brain MRI, T2-weighted sequence.

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Precision in historical quotations[☆]

Exactitud en las citas históricas

Dear Editor,

If historical articles are to have the same level of quality and prestige as medical and technical articles, any quotations must be meticulously researched. Otherwise, the article does not achieve its purpose. This comment is a response to the most recent article by Dr. Arboix and Dr. Fabregas, 'The first Catalan neurological societies and their protagonists', published in *Neurología*. In just the first paragraph describing the French school of neurology as a precursor of the Catalan school, we find the following inaccuracies:

- 1) Charcot's name was Jean Martin, not Jean Marie.
- 2) Duchenne de Boulogne was not in fact Charcot's professor, although the latter chose to call him by that honorific. Duchenne was an unaffiliated genius whose studies of the effects of electricity on neuromuscular systems frequently led him to La Salpêtrière and other hospitals. As the story is told in France, the patients feared him and called him 'l'homme à la petite machine mechante' [the man with the vicious little machine]. Be that as it may, Duchenne was a rigorous observer and researcher who also introduced needle-stick muscle biopsies. While his ground-breaking contributions earned the respect of Charcot and many others, he was never entrusted with a hospital or university position.
- 3) Charcot was succeeded by Fulgence Raymond, not by Dejerine, whose time came later. Dejerine's first

initial, given as L, is really J (for Jules, as he was best known) or JJ (Joseph Jules, his full name). The Dejerine surname has been accented in every imaginable way, but Dejerine's daughter, Yvonne Sorrel-Dejerine who was the secretary of the Société Française de Neurologie for many years, stressed that the correct form takes no accent marks.

- 4) Pierre Marie was not the founder of a neurology department at 'Bizet'. That department, if we can call it such, already existed at Hospice Bicêtre (Le Kremlin-Bicêtre). Hospice Bicêtre, which housed disabled (and convicted) men and boys, was analogous to the women's hospital La Salpêtrière. Apart from Dejerine, another of Hospice Bicêtre's illustrious figures was Pinel, who freed the shackled madmen as he had freed the chained women in La Salpêtrière. Pierre Marie did not take a position at Bicêtre of his own volition; rather, Dejerine pushed him out of the Salpêtrière and ended his hopes of succeeding Charcot. Pierre Marie would later return to exact his vengeance. Prior to that, Babinski himself never chose to work at La Pitié. He had been forced out of La Salpêtrière by Bouchard, who in doing so buried Charcot's legacy in a very Freudian way: denying Charcot's favourite student the department chair. Feuds and rivalries sprung up on all sides.
- 5) La Société de Neurologie de Paris was founded in 1899, not 1900; its 100th anniversary was celebrated in 1999, according to the webpage of its direct descendent, the modern Société Française de Neurologie.

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[☆]Please cite this article as: Zarranz JJ. Exactitud en las citas históricas. *Neurología*. 2013;28:524.

Precision in historical quotations. Reply[☆]

Exactitud en las citas históricas. Réplica

Dear Editor:

We express our sincere thanks for the very judicious comments made in response to our recent article in *Neurología*.¹

Our purpose in writing the article was to shed light on a lesser-known moment in the history of Neurology: the activities of the first Catalan societies of neurology, the first of which was founded by Dr Artur Galcerán i Granés in 1911, under the name of *Sociedad de Psiquiatría y Neurología de Barcelona*.² As stated in our article, Dr Martí i Julià (1860–1917) succeeded him as president in 1915. In 1934, the Society adopted a Catalan name, *Societat Catalana de Psiquiatría i Neurologia*; its president at the time was Dr Belarmino Rodríguez Arias. That society was dissolved during the Spanish Civil War. In 1941, the society was reinstated under the Spanish name *Asociación de Neurología y Psiquiatría*, with Dr José Córdoba Rodríguez as its president. In 1968, it became *Asociación de Ciencias Neurológicas*

[☆] Please cite this article as: Arboix A, Fabregas M. Exactitud en las citas históricas. Réplica. *Neurología*. 2013;28:524–525.