

NEUROLOGICAL PICTURE

Trigeminallepsy

A 39-year-old-woman was admitted to the hospital after attempting to commit suicide by jumping off a bridge. She had been diagnosed with trigeminal neuralgia at the age of 19 years, which was refractory to medical therapy with carbamazepine and high doses of amitriptyline. She preferred to be self-medicated with

benzodiazepines and alcohol to relieve her pain since she was 21 years old; nevertheless, such substance abuse led to impairment of depressive disorder induced by her rebel facial pain.

On admission, our patient fulfilled the criteria for major depressive disorder according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000), presenting with depressed mood, insomnia, psychomotor retardation and inappropriate guilt. Moreover, she experienced intense hopelessness due to her history of facial pain resistant to medical

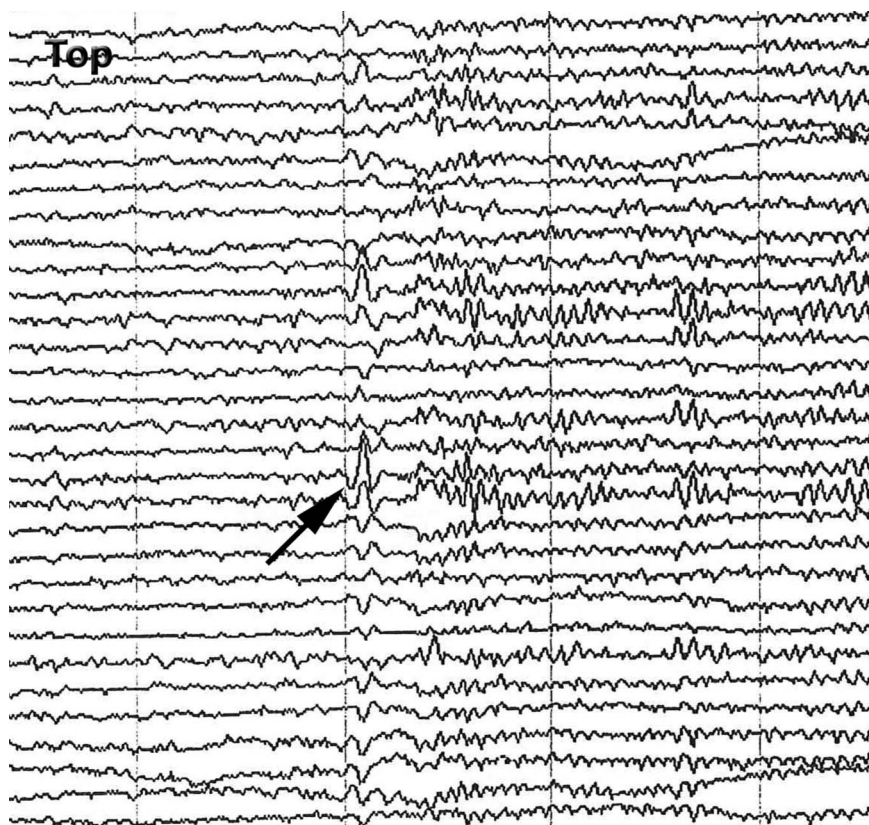


Figure 1 Video-EEG trace revealing spike discharges, which were elicited by the touch of the cheek (arrow). Such finding dramatically improved our patient's outcome.

therapy, which led her to refuse other therapeutic approaches. Furthermore, years back she had been told that other neurological diseases had been ruled out, since both CT and brain MRI were normal. Her intense left-sided facial pain attacks were mainly triggered by local tactile stimuli, and became partially controlled with benzodiazepines and alcohol intake. After complete alcohol detoxification was granted, a test that had never been elected before finally led to curative management.

A video-EEG showed spike discharges induced by the touch of her left cheek (figure 1). The final diagnosis was reflex epilepsy with partial simple seizures (sharp pain) elicited by tactile stimuli. A functional MRI of the brain localised the epileptic focus in the right parietal lobe. Initial response was reported with levetiracetam plus pregabalin plus venlafaxine plus trazodone. A complete remission was achieved after stereotactic resection of the epileptic zone.

COMMENT

Reflex epilepsies, characterised by seizures that are provoked by specific stimuli, are rare conditions that may be underdiagnosed and, thus, may mislead to other diagnoses.¹ Indeed, our patient had been suffering from sensorial seizures which mimicked trigeminal neuralgia for 20 years.

Tricyclic antidepressants such as amitriptyline are widely used to treat neuropathic pain; nevertheless, tricyclic antidepressants may lower seizure threshold, which initially impaired our patient's quality of life.² Conversely, benzodiazepines characteristically raise seizure threshold, thus reducing seizure frequency. Otherwise, alcohol withdrawal syndrome lowers seizure threshold and increases pain sensitivity, which led to abuse from alcohol and benzodiazepine consumption to relieve her seizures.³

To our knowledge, this is the first case reported in English-language literature that considers epilepsy as a differential diagnosis of trigeminal neuralgia. Despite controversies on the term migralepsy, we have entitled our case as *trigeminalesy* to claim that refractory trigeminal pain, or any rebel idiopathic neuralgia localised elsewhere, may be electable for a simple EEG to rule out atypical epilepsy.⁴

Carlos Miró,¹ Tomás Ortiz²

¹Department of Neuropsychiatry, University Hospital of La Ribera, Valencia, Spain

²Magnetoencephalography Unit, Complutense University of Madrid, Madrid, Spain

Correspondence to Dr Carlos Miró, Department of Neuropsychiatry, University Hospital of La Ribera, Carretera de Corbera Km. 1, Alzira, Valencia 46600, Spain; cmiro@hospital-ribera.com

Contributors CM has made substantial contributions to the case reported. TO has participated in critical revision of the manuscript for important intellectual content.

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