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LETTERS TO THE EDITOR

Male anorgasmia as initial symptom of transverse myelitis☆

Anorgasmia masculina como síntoma inicial de mielitis transversa

Dear Editor:

Anorgasmia is the inability to reach orgasm after any type of sexual stimulation.[1] Sexual desire, psychological and physical stimulation, penile erection, and ejaculation typically precede the male orgasm.[2] Erections are regulated by the parasympathetic nervous system through the inferior hypogastric plexus and by the somatic nervous system through pudendal nerve fibres, both of which arise from the sacral nerves S2 to S4.[3] Ejaculation is conveyed through the hypogastric plexus originating in the sympathetic chain ganglia of the spinal cord segments T11 to L2.[4,5] Involuntary smooth muscle contractions of the seminal vesicles and the striated muscles of the pelvic floor give rise to the release of semen associated with orgasm. The afferences involved in sexual pleasure activate such brain areas as the mesodiencephalic transition zone (which includes the mesencephalic tegmentum and the hypothalamus), subcortical structures (caudate nucleus, thalamus), the cerebral cortex (primarily the amygdala and the right neocortex), and even the cerebellum.[6]

Sexual function disorders as a result of such neurological conditions as head trauma, stroke, epilepsy, Parkinson's disease, multiple sclerosis, myelopathy, and peripheral neuropathy have previously been described in the literature.[7]

We present the case of a patient with isolated anorgasmia as an initial symptom of myelitis.

The patient was a 30-year-old man with no relevant medical or surgical history who presented at the urology department with anorgasmia. From adolescence to 20 years of age, the patient described satisfactory sexual desire, no erectile dysfunction, and normal ejaculations accompanied by orgasm. At the time of the consultation, however, he disclosed a complete loss of sexual pleasure over the previous 10 years, although all other sexual functions remained intact. He reported no urethral or anal sphincter dysfunction. Physical examination of the pelvic floor and the external genitalia, a scrotal Doppler ultrasound, a spermiogram, and a hormonal analysis (including testosterone, estradiol, prolactin, LH, and FSH) yielded normal results. The patient was referred to the neurology department, where an examination revealed normal results except for pallesthesia in the lower limbs.

The neurophysiological study included an electromyography of the bulbocavernosus muscle and the external anal sphincter using concentric bipolar needle electrodes, a study of somatosensory evoked potentials (SEP) from

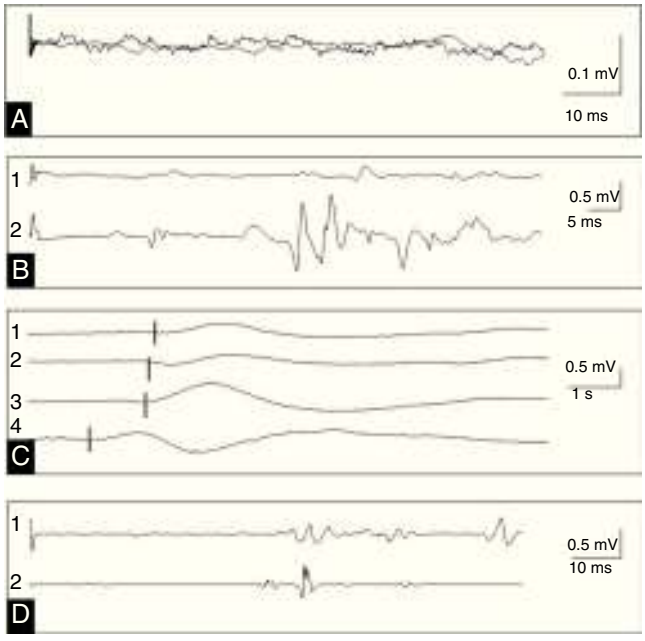

Figure 1 (A) Absent somatosensory evoked potentials in the internal pudendal nerve. (B) Registered transcranial magnetic stimulation of the bulbocavernosus muscle in relaxed state (1) and after motor facilitation (2) showed normal latency. (C) Sympathetic skin response to nociceptive stimulation of the right sole (1), the left sole (2), the right palm (3), and the perineum (4). (D) Right (1) and left (2) bulbocavernosus reflex with normal latency and symmetry.

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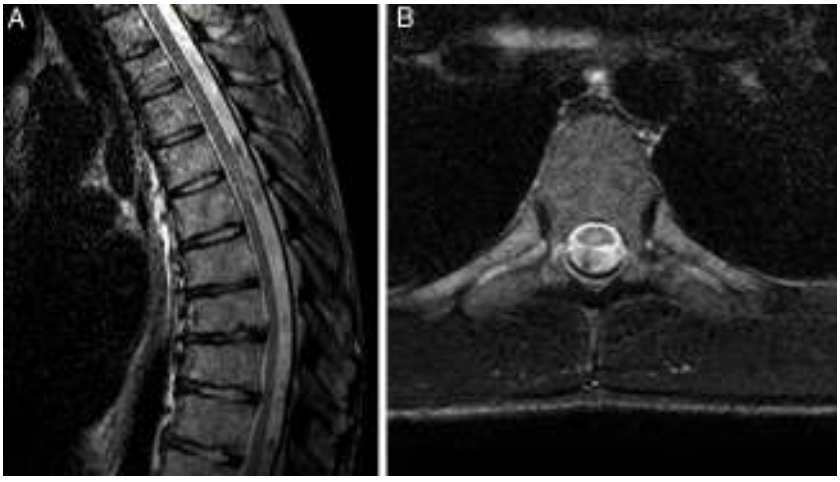

Figure 2 Axial (A) and transversal (B) T2-weighted MRI sequences of the dorsal column showing hyperintensities in the centre of the spinal cord at T5-T6 and no mass effect, uptake, or oedema, which is compatible with residual signs of myelomalacia.

the internal pudendal nerve to the posterior tibial nerve, a registered transcranial magnetic stimulation of the bulbocavernosus muscle, a sympathetic skin response test of the perineum and limbs, a sacral reflex test (bulbocavernosus and anal), and a sural nerve sensory neurography. These studies revealed absence of SEP in the pudendal and posterior tibial nerves and normal results for the remaining parameters, which is indicative of injury to the somatosensory pathway at the level of the posterior column above the L1 segment (Fig. 1).

Cranial, cervical, dorsal, and lumbar MRI scans showed myelomalacia in the posterior spinal cord at T5-T6 level which was compatible with residual complications from myelitis (Fig. 2).

Blood analyses (including thyroid function and vitamin B_{12} levels), serological tests (HIV, HTLV, CMV, EBV, borrelia, syphilis), and autoimmune tests (ANA) did not reveal the aetiology of transverse myelitis.

The correlations between transverse myelitis and sexual syndromes has previously been described in the literature.[8,9] Male anorgasmia as the sole sexual disorder after spinal cord lesions is unusual. Sensory alterations, motor symptoms, and sphincter dysfunction are concomitant symptoms that depend on the location and size of the lesion.[10] In this particular case, the normal sympathetic skin response of the perineum is compatible with the preservation of the sympathetic pathway from the sympathetic chain ganglia at T11-L2 to pelvic nerves and, consequently, intact erectile function. Anal and bulbocavernosus reflex studies indicated no pelvic floor sensorimotor or reflex arc impairment. These systems regulate the striated sphincters, sensitivity of dermatomes S2-S4, ejaculation, and the transmission of sexual pleasure. The absence of SEP and myelomalacia limited to the thoracic portion of the posterior spinal column explain the lack of afferent transmission of the orgasm as well as the pallesthesia. Preservation of the corticospinal motor pathway is confirmed by the results of the transcranial magnetic stimulation. The electromyography study and the neurography ruled out focal and systemic sphincter neuromuscular disorders, respectively.

Taking sexual histories and analysing sexual function is not common practise in neurology consultations. A basic sexual history and knowledge of the complementary tests available can help manage patients with sexual syndromes of possible neurological origin.

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Altered neurocognitive functions in a patient with carbon monoxide poisoning☆

Funciones neurocognitivas alteradas en paciente intoxicada por monóxido de carbono

Dear Editor:

Carbon monoxide (CO), released into the environment by the incomplete combustion of hydrocarbons, forms carboxyhaemoglobin (HbCO) when it comes into contact with haemoglobin (Hb) in the blood. HbCO reduces the formation of oxyhaemoglobin, impeding the transportation of oxygen to body cells and leading to hypoxia, anoxia, and death when the concentration increases. As CO is an invisible and highly toxic gas, death can arrive before the subject realises anything is happening (silent killer). In industrialised countries, CO is the principal cause of death by poisoning (suicides are 3.4 times more frequent than accidents). In Mexico, an estimated 233 deaths annually are due to toxic gases, of which 166 are accidental and 5 are suicides; however there is reason to believe that CO deaths are under-reported, given the non-specific nature of clinical manifestations, the lack of pathognomonic signs or symptoms, and the dearth of diagnostic equipment.[1–3] In addition to underdiagnosis, neuropsychological symptoms of CO poisoning are largely unknown, even though damage may remain for several years after the event.

Our patient was a 30-year-old woman with a bachelor's degree who was poisoned by CO emanating from a malfunctioning gas heater, which caused the death of her partner and diverse sequelae to herself. Her cardiovascular system was unscathed, but her vision was damaged and her central nervous system (CNS), especially neurocognitive function, was severely affected. She was diagnosed with upper motor neuron syndrome, right hemiparesis secondary to hypoxic—ischaemic encephalopathy, and disability in activities of daily living.

Results of the neurocognitive evaluation. INTELLIGENCE QUOTIENT: Verbal IQ = 66; performance IQ = 60; full-scale IQ = 60; verbal comprehension index = 66; perceptual organisation index = 69; processing speed index = 62; working memory index = 86. These scores are extremely low, which means these aspects of IQ were severely affected. Working memory, though less affected, was below average, indicating an inability to learn new information and communicate with others. *LURIA NEBRASKA NEUROPSYCHOLOGICAL BATTERY*: Sensory aphasia, predominantly auditory aphasia and anomia. *Abbreviated Barcelona Test*: Performance on the various tasks assessed was poor or very poor. *REY-OSTERRIETH COMPLEX FIGURE TEST*: Left hemispatial neglect and apperceptive visual agnosia; the patient was unable to draw a line of the figure. *Orientation*: severely affected in all 3 spheres. *Higher mental functions*: **Attention*: short attention spans, easily distracted; auditory attention: fluctuating; visual attention: incapable of perceiving a picture according to Gestalt laws, image segmentation. **Language comprehension*: fluctuating and severely impaired. The relationship between 'signifier' and 'signified' was not preserved. *Language fluency*: not very fluent, unspontaneous, only answered questions. The patient provided incorrect, unreal, and incoherent information but was able to complete some familiar series (days of the week, months of the year, numbers 1-10). The patient presented anomia, paraphasia, perseverations and confabulations, but not dysarthria; *grammatical structures*: she produced poorly structured sentences containing the 3 main components (subject, verb, and predicate). **Writing*: visuospatial problems, left hemispatial neglect, micrography, automatisms, with structural defects. Numbers and letters crowded together and overlapped. During the dictation test, the patient wrote only mono- and bisyllabic words, and when she was required to copy some text, she was only able to identify one consonant from a sentence. Dissociative writing: in regards to reading and writing, the patient was incapable of connecting signifiers with signifieds. **Reading* and **executive functions*: severely impaired. **Calculations*: the patient's ability to do mental and written arithmetic was severely affected. She was unable to understand numerical abstraction and had no understanding that a 1 or a 0 could be either a unit or a decimal, as in 0.01, 10, 100, or 1000. She was able to write the numbers from 1 to 10 but did not identify them as symbols or do mathematical operations with them, and could not classify one as 'more' or 'less' than another. **Memory*: The patient's episodic memory was severely impaired and she produced confabulations with unreal content. Her semantic memory was also severely impaired and characterised by anomic aphasia. **Thinking*: concrete. **Visuospatial ability*: severely impaired. Incapable of distinguishing 'right', 'left', 'in front', or 'behind', the patient was only able to identify 'up'. In addition, she could only visualise the top third of the right side of a piece of letter-size paper in a horizontal position. **Body*

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