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DIFFUSE TOXIC LEUKOENCEPHALOPATHY SECONDARY TO COCAINE USE: A CASE REPORT

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Abstract: A 27-year-old girl, presented with weakness of lower and upper limbs, acute and progressive onset, evolving with inability to walk, besides excessive sleepiness, unmotivated laughter and personality alteration after 7 days of exacerbated Cocaine use. Physical examination admission, she had Babinski, Tromner and Hoffman signs bilaterally, tetrasegmental grade 3 strength, apraxic speech, bradypsychism, and a puerile and uninhibited attitude. General laboratory tests, including infectious, were normal, and the CSF showed only mild proteinorrhea. MRI of the skull demonstrated axial FLAIR image with extensive bilateral supratentorial white matter hypersignal. No previous neurological or systemic complaints were reported. The patient spontaneously reported recreational use of marijuana and cocaine, with 25g of the latter being used approximately 7 days prior to the onset of symptoms. During hospitalization the patient evolved favorably, and after 9 months of the occurrence, she was living independently, with significant recovery of motor and behavioral symptoms. Discussion: Cocaine use is associated with a variety of neurological complications, classically involving cerebrovascular events. The cocaine-related toxic leukoencephalopathy (TL) is a rare condition, with few reports in the medical literature, and that may be related to an immune-mediated response mechanism. The onset of symptoms is usually subacute, and the most common symptoms being motor deficit, altered cognition and behavior. There appears to be no significant difference in risk between chronic cocaine abusers and first time users, as well as between individuals who snort cocaine and those who smoke it in the form of crack. Although, the patient presented a good evolution, the risk of serious neurological sequels is a high possibility, moreover, and the mortality of TL is approximately 23%, it is important to remove the use of the toxic substance ahead for a better future prognosis. Final comments: we report acute TL after recreational cocaine abuse in a young woman, its relevant this case for additional etiological knowledge to the neurologist, because the widespread substance use in today's society, should always be remembered in non-focal neurological symptoms, with compatible MRI, and no other commemorative, unless, the report of toxic substance use/abuse.
Keywords: Leukoencephalopathy, drugs, brain, neuroimunology.

CASE PRESENTATION

A 27-year-old single girl was admitted to our hospital due to weakness of lower and upper limbs, with acute and progressive onset, evolving in a few days with inability to walk, besides excessive sleepiness, unmotivated laughter and personality alteration with puerile behavior, after 7 days of exacerbated cocaine use. She reported sporadic and recreational use of this drug. On general admission examination: she presented bilateral Babinski, Tromner and Hoffman signs, grade 3 tetrasegmental strength, slurred speech, slow thinking, puerile and uninhibited attitude, and altered sleep-wake cycle. General laboratory tests, including infectious, were normal, and CSF showed only mild protein increase. Skull MRI demonstrated axial FLAIR imaging with extensive bilateral supratentorial white matter hypersignal. No neurological or systemic complaints were previously reported. He denied vaccination and previous infection.

The patient reported recreational marijuana use and recreational cocaine abuse, with reported use of “25g” , approximately 7 days prior to the onset of the aforementioned symptoms. During hospitalization, the patient progressed favorably with only supportive treatment, without any specific treatment, and 9 months after the illness, she was living independently, with significant recovery of

motor and behavioral symptoms. The patient maintains outpatient follow-up with our team, and reports no further recreational cocaine use. She maintained a more expansive attitude, but without any other impairment.

DISCUSSION

Cocaine use is proven to be associated with a variety of neurological complications, most commonly involving cerebrovascular events. Toxic leukoencephalopathy is an already well known entity, however, specifically cocaine-related (TL) is still an uncommon condition with few reports in the current medical literature.The pathophysiology of cocaine toxic leukoencephalopathy remains unknown, however, some pathophysiological hypotheses may be related as mechanisms of immune-mediated inflammatory response and mitochondrial metabolism after the use of this illicit stimulant. The signs/ symptoms are usually atypical, non-specific, with the most reported clinical picture being behavioral/cognitive alterations, altered level of consciousness, and motor alteration, associated with subacute and progressive onset. The neuroimaging pattern, with MRI-FLAIR showing extensive, bilaterally and diffuse symmetrical lesion in supratentorial white matter, perfectly matches similar radiological images reported in the most up-to-date literature.

The etiology of this condition is still poorly understood.And there is no general consensus on the treatment of cocaine toxic leukoencephalopathy. The course of the disease is variable according to the current medical literature, ranging with unexpected full recovery of neurological and behavioral symptoms, and in other reported cases evolving severely with neurological sequelae or death. There appears to be no significant difference in acute risk between chronic cocaine abusers and first-time users, as well as between individuals who snort cocaine and those who smoke it in crack form. It is important to remove the toxic substance use for a better future prognosis. The relapse rate is high, and therefore, it is necessary to provide adequate guidance to the patient about the potentially fatal risks and complications of substance use. In cases of reported dependence, there is a need for professional follow-up with an addiction specialist to reduce the likelihood of relapse.

FINAL COMMENTS

However, there is extreme caution in the diagnosis of LT, for presenting other differential diagnoses, even more prevalent in the epidemiology of the patient's age group, mainly due to the fact that demyelinating diseases of the cerebral white matter, as is the case of multiple sclerosis, and ADEM, may also present with spontaneous remission. Due to the fact that cocaine is an illicit toxicant of increasing use and quite common nowadays, the hypothesis of cocaine-related TL should always be rigorously elucidated, especially in cases of typical neuroimaging presentation according to other deported reports, with compatible clinical resolutions, spontaneous or recurrent, and with risk factors for cocaine use/abuse and dependence. And mainly, of a good doctor-patient relationship that corroborates with good anamnesis.

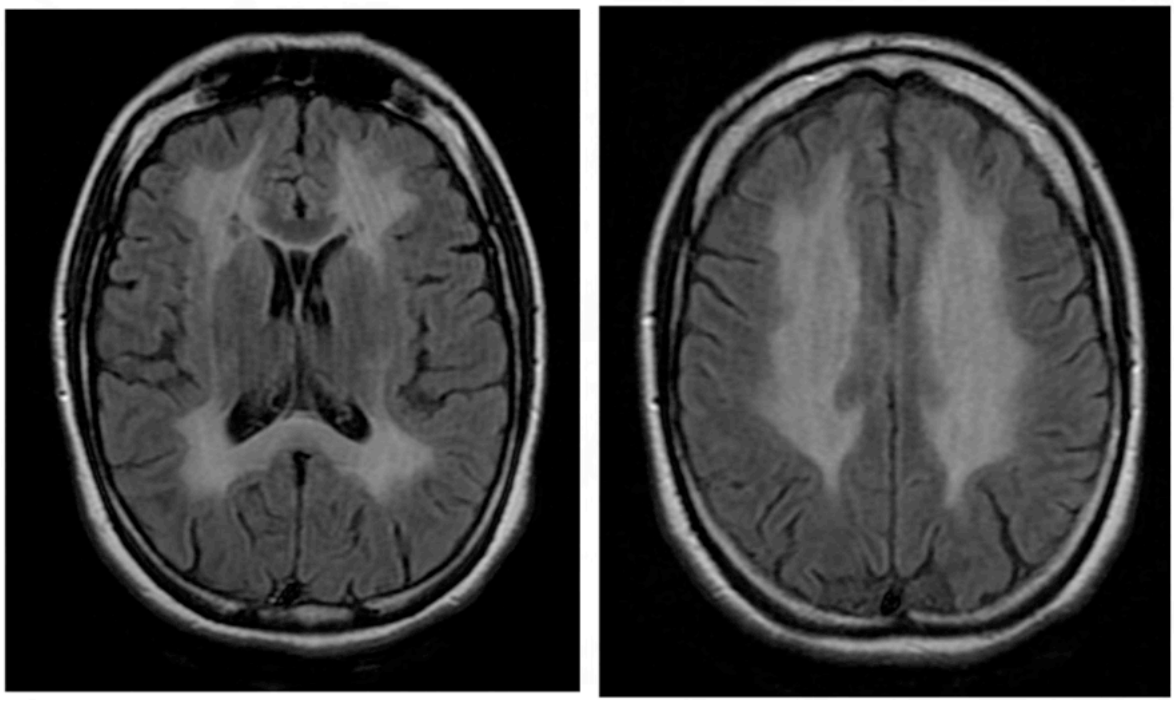

Imagem 1. Axial FLAIR images demonstrating extensive bilateral, symmetrical hypersignal on supratentorial bilateral, symmetrical hypersignal of the supratentorial white matter.

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