Toxic leukoencephalopathy: a case report

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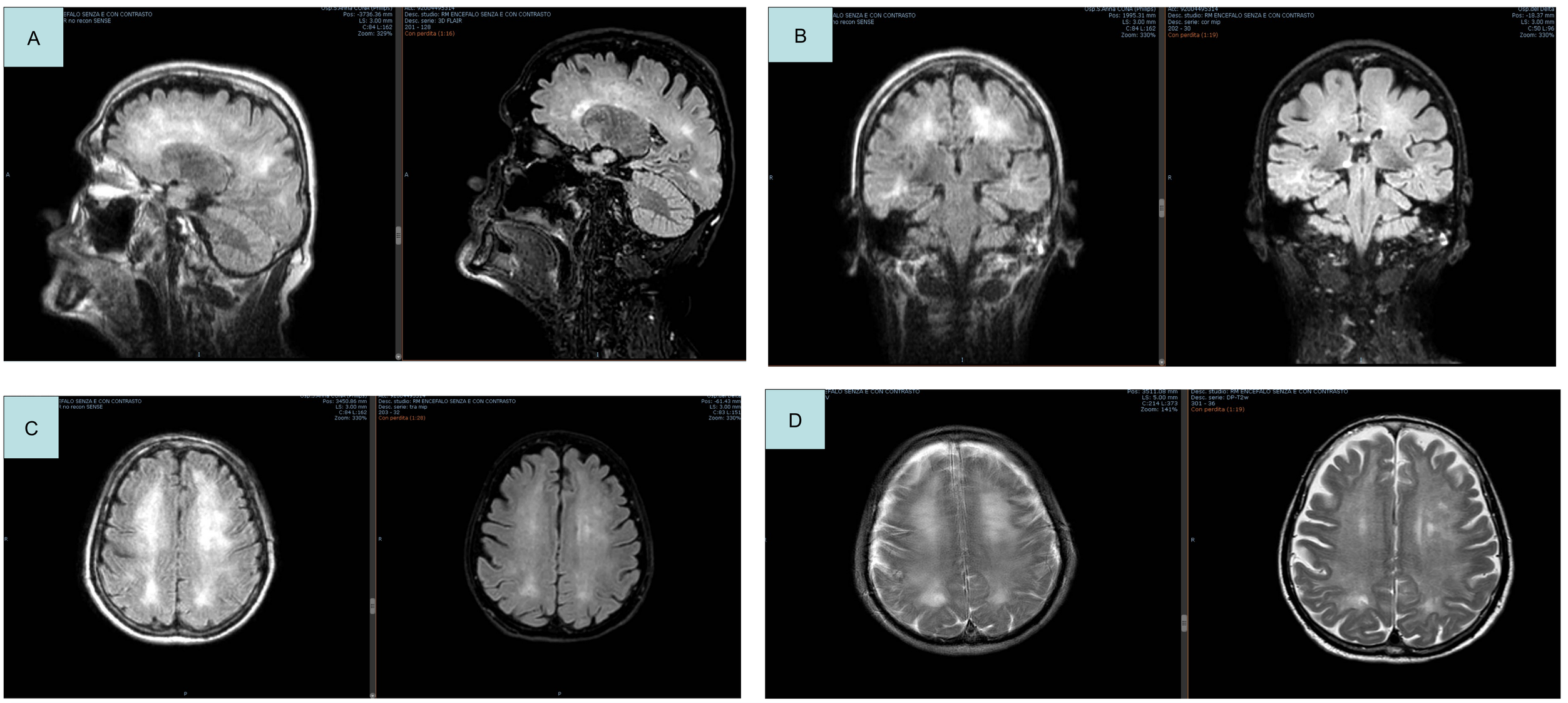
Introduction: Leukoencephalopathy caused by heroin inhalation is known by the term “chasing the dragon”. Another intriguing example of toxic brain injury is the one caused by cocaine. There may be an immune basis possibly related to levamisole, a cocaine adulterant now commonly found in cocaine and causing multifocal inflammatory leukoencephalopathy. We hereby present the case of a toxic leukoencephalopathy with substantial recovery.

Materials and Methods: B.D.,male, 58 year old. Was admitted to our Neurology Department for the subacute development of behavioural change with refusal to eat and to take his medicines and a global psychomotor slowness. There was a history of cocaine and heroin abuse and he was recently released from jail after 16 years of detention. No therapy with methadone or benzodiazepines was ongoing. Further anamnestic details include acute myocardial infarction,chronic HCV infection and benign prostatic hypertrophy. After release from prison, he developed rabdomyolysis with acute renal failure and acute hepatitis. A toxicological screen detected cocaine in urine with a significant titre. In the days after, he started to show disorientation and behavioural changes. A psychiatric evaluation excluded acute psychopathological state. In Internal Medicine department various exams were performed (brain CT, EEG and a thoracoabdominal CT), with no remarkable findings. Syphilis and HIV were excluded. On day 15 after hospital admission he was moved to our Neurology department. The patient appeared awake but no verbal interaction or cooperation were possible. There was a tethraparesis with generalized spasticity. Deep tendon reflexes were brisk. Startle myoclonus was seen. Primitive reflexes were present. CSF showed moderate hyperglycorrhachia with no oligoclonal bands. Blood tests included: cryoglobulins, quantiferon test and autoimmune panel (negative). HSV 1-2, VZV, EBV, CMV and JCV in CSF were excluded. MRI showed diffuse hyperintensity in T2 weighted sequences of the periventricular white matter, particularly of semioval centers and fronto-parietal regions with restriction of diffusion and no contrast enhancement. EEG showed theta activity bilaterally over the posterior region and delta activity over the anterior ones in a generalized slowed activity. An increase of 14-3-3 and Tau protein was detected, probably due to the dramatically fast neuronal loss. We then started bolus administration of steroid therapy with methylprednisolone: there was an improvement expressed by more cooperation and interaction with the environment and a less elusive and more concrete verbal communication. The patient was then moved to another hospital and continued steroid therapy with stabilization of his neurological assessment.



MRI comparison between baseline and month 1 after steroid treatment A) Sagittal FLAIR B) Coronal FLAIR C) Axial FLAIR D) Axial T2

Discussion and Conclusion: **although we cannot prove the levemisole exposure, the persuasive temporal correlation between cocaine assumption and the development of neurological symptoms, MRI features and improvement with steroid treatment indicate cocaine as the most probable etiological agent**

References

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