Int. j. adv. multidisc. res. stud. 2024; 4(2):1505-1506

Received: 08-04-2024
Accepted: 15-04-2024

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

Bilateral White Matter DWI Hyperintensities in a Polysubstance Addict may not be solely due to Cocaine Vasoconstriction

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DOI: https://doi.org/10.62225/2583049X.2024.4.2.2716
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We read with interest the article by Redmond *et al*. on a 23-year-old male with bilateral internal watershed infarctions attributed to cerebral vasoconstriction with consecutive cerebral hypoperfusion due to multiple substance abuse including methadone and cocaine [1]. He presented clinically with confusion and lower limb weakness [1]. It was concluded that drug abuse should be considered a specific cause of watershed infarcts [1]. The study is impressive, but some points require further discussion.

A first point is that no differential explanations for the DWI hyperintensities have been provided. The main differential cause of DWI hyperintensities is an epiphenomenon of seizure activity. Arguments for a recent seizure as the cause of the imaging abnormalities are that the patient had impaired consciousness, was leaking urine at the time of discovery, was suffering rhabdomyolysis, and that his urine was positive for methadone and cocaine. The latter could indicate that one or more seizures were triggered by either intoxication or withdrawal. Were clinical signs of withdrawal syndrome detected during the examination? A normal EEG after a seizure does not rule out that someone had a seizures a few hours ago.

The second point is that the pathophysiology of watershed infarctions is not limited to haemodynamic impairment, but can also be due to stenosis of cerebral arteries (internal watershed infarction) or embolism (external watershed infarctions) due to coagulopathy [2, 3]. In addition, normal heart rhythm monitoring does not exclude that the index patient had cardiac arrhythmias during the acute intoxication. If the cause of watershed infarctions remains unclear, it is recommended to implant a reveal recorder for long-term ECG recording. Assuming that vasoconstriction due to cocaine was indeed responsible for the internal watershed infractions in the index patient it should be explained why only some, but not all, penetrating arteries were affected by vasoconstriction.

The third point is that the location of the DWI hyperintensities does not correspond to the clinical presentation. Since the patient had bilateral lower limb weakness, one would expect a bilateral infarction involving both anterior cerebral arteries. The location of the infarctions shown in figures 1 and 2 suggests that upper extremity weakness and aphasia should also be present. How do the authors explain the discrepancy between clinical presentation and imaging?

A fourth point is that cocaine is usually not available as a pure substance, but in most cases it consists of a mixture of active ingredients and additives to artificially prolong the amount of the drug. Therefore, all molecules associated with cocaine impurities should also be considered as factors contributing to the pathophysiology of the infarcts.

A fifth point is that no follow-up MRI was performed to document the resolution of the DWI white matter hyperintensities. Knowing the outcome of the imaging lesions is crucial for assessing their pathophysiology. Complete resolution would suggest that they were postictal rather than due to cerebral hypoperfusion.

A sixth point is that there is no agreement between the ADC images in figure 1 panel d, and the DWI hyperintensities in figure 1 panels a to c [1]. Therefore, hypointensities in panel d do not indicate cytotoxic edema. What was the explanation for these ADC hypointensities shown in figure 1 panel d?

In summary, the interesting study has limitations that put the results and their interpretation into perspective. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Before DWI hyperintensities can be attributed to watershed infarction induced by cocaine-related vasoconstriction, alternative etiologies of the lesion must be thoroughly ruled out.

Declarations
Ethical approval: Not applicable.

Consent to participation: Not applicable.

Consent for publication: Not applicable.

Funding: None received.

Availability of data and material: All data are available from the corresponding author.

Completing interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author contribution: JF was responsible for the design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. SM: Contributed to literature search, discussion, correction, and final approval.

Acknowledgements: None.

Keywords: Internal Watershed Infarction, Polysubstance Abuse, Cocaine, Rhabdomyolysis, Seizure

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