Symmetric Deep Cerebellar Lesions After Smoking Heroin

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Background: Acute symmetric deep cerebellar lesions suggest toxic exposure.

Objective: To describe a patient with striking neurologic and magnetic resonance image features.

Design: Case report.

Setting: Emergency department and office.

Methods: Personal observation.

Results: A middle-aged man had a day of unsteadiness, followed by acute and pronounced cerebellar signs. The degree of disability was remarkable. Magnetic resonance imaging showed almost perfectly symmetric deep cerebellar damage that ultimately became cavitated. Serial querying of the patient revealed the use of heroin by inhalation just prior to the emergence of the ataxic syndrome.

Conclusions: An acute and purely ataxic syndrome with symmetric deep cerebellar lesions suggests toxic exposure, in this case, the smoking of heroin.

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PERFECTLY SYMMETRIC white matter lesions in the cerebrum or, less often, the cerebellum, have been reported after various toxic exposures, including illicit drug use. We describe an extreme example of acute onset that resulted in regional cerebellar destruction.

REPORT OF A CASE

A 40-year-old accountant with no medical problems or family history of neurologic disease awoke with severe unsteadiness, which he attributed to a previous inner ear problem during scuba diving. Despite the symptoms, he was able to drive to and from work, make deposits at a bank, and eat dinner. The following morning, he was more unsteady but was able to drive to the hospital and walk into the emergency department. During a period of 15 minutes, while speaking to the admitting nurse, he became increasingly unbalanced, dysarthric, and clumsy, and was no longer able to walk. There had been no fever, headache, vertigo, or vomiting.

The examination showed severe ataxia of all of the limbs and an inability to stand and walk. He became severely dysarthric in minutes. In addition to these prominent early features, we later found that there was no nystagmus, and the eyes showed only a slight tendency to overshoot with refixation, limb tone was minimally diminished, there was no static tremor of the proximal limbs, and the reflexes in the legs were brisk, without Babinski signs. He was barely able to stand, and only with assistance. His breathing rhythm and rate were erratic, with a tendency to snort. A rapid chin tremor appeared intermittently. His family noted an unnatural degree of emotional display, which was not apparent to us. His short-term memory, praxis, and visuospatial skills were normal on office testing.

Magnetic resonance imaging showed highly symmetric “C-shaped” lesions in the deep cerebellar hemispheres, encompassing the white matter and dentate nuclei (Figure). These were apparent the day following admission and became cavitated on a later scan. There was slight signal change in the central midbrain at the crossing of the cerebellar peduncles but no abnormalities elsewhere.

After protracted questioning, it was revealed that he had smoked heroin the day before and the day of illness onset. He did not use ethanol or cocaine during that
time. We could not ascertain if others who partook of the same heroin source were likewise affected. The disabling cerebellar findings were unchanged when examined 6 months later.

**COMMENT**

The entirely symmetric location of the lesions in this patient and an eventually static clinical syndrome suggested to us a toxic exposure. Extensive questioning revealed the preceding heroin inhalation. An abrupt and severe ataxic disturbance initially gave the impression of a vascular disturbance. A toxic leukoencephalopathy of this type has been described following heroin use, and less frequently, after the use of cocaine alone or in combination with other abused drugs. On the basis of clusters of these cases, it has been presumed that the heroin was adulterated with a contaminant of unknown type. It is not clear why inhalation of heroin, in contrast with injection, is particularly prone to cause toxic leukoencephalopathy; perhaps an immunologic mechanism mediated by the lungs is implicated. It is further possible that a second exposure to heroin was responsible for the rapid decline after a day of slight symptoms.

In most reported cases, the cerebral hemispheres have been prominently involved, occasionally with concurrent damage in the cerebellum.1-5 Koussa and colleagues emphasized the cerebellar aspects of the illness, as found in their second and third cases, which resemble those in our patient. There were, however, additional posterior hemisphere signal changes in their patients, whereas our patient represented an almost purely cerebellar process. In most such cases, there has been a latency of a day or more between inhalation of the drug and the first symptoms. Based on the limited pathologic evidence, the process has been described as spongiform, an aspect on which we cannot comment. In our patient, the normal findings on oculomotor examination, diminished limb tone, unimpaired mentation, and chin tremor are worth noting. The severe acute ataxia and the magnetic resonance imaging pattern are characteristic of this encephalopathy. These findings together suggest inhalation of heroin or a related drug.

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**REFERENCES**