Recovery of Cerebral Blood Perfusion from Transient Hypo-Perfusion Due to Acute Benzodiazepine Poisoning Coinciding with Generalized Convulsion as Withdrawal Syndrome

Shu-ichi Yamashita, Itaru Kyoraku, Takashi Murahara, Kazutaka Shiomi and Masamitsu Nakazato

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An 86-year-old woman was admitted to our hospital because of a deep coma. Although she had suffered from chronic bronchitis and congestive heart failure, the symptoms and signs were stable and she was in her usual state of health until the morning of the day of admission. On the evening of admission day, her husband found her unconscious when he returned home from shopping. A volume of vomit that included numerous white pills was found around her. She had been prescribed 3 mg of benzodiazepine medicine, Etizolam, as a daily dose by her primary physician, and the pills in the vomit appeared to be this drug.

On admission, she was in a deep coma. Her extremities showed no movement on noxious stimuli, but her brainstem reflexes such as light reflex were intact. Blood chemistry
Tc-99m HMPAO SPECT imaging of the brain on the 6th hospital day demonstrates recovery of cerebral blood flow to almost normal level. On that very day, the patient had generalized convulsion as a benzodiazepine withdrawal syndrome. The calculation of this image also used the same presumptive mCBF for the sake of the comparison with Picture 1.

Other metabolic conditions were excluded as the cause of her coma. Qualitative screening of her urine sample for benzodiazepine was positive by multi-immunologic assay (Triage DOA, Sysmex Co., Kobe Japan). Computed tomographic scanning and magnetic resonance imaging of the brain revealed only mild brain atrophy. Thus, stroke or anoxic brain damage seemed unlikely as her baseline condition. Electroencephalogram showed diffuse low-voltage 2-Hz delta activity without paroxysmal discharges. Technetium-99m hexamethyl-propyleneamine oxime (Tc-99m HMPAO) single photon emission computed tomographic (SPECT) imaging of the brain demonstrated markedly reduced blood flow in widespread brain areas (Picture 1) (1, 2). A diagnosis of drug-induced encephalopathy due to an alleged suicide attempt was made, though the presence of ischemic brain damage was not completely denied at the time. Intravenous fluid was administered for the treatment of moderate dehydration. Her consciousness gradually improved after admission. She was awake and alert on the 6th hospital day, when Tc-99m HMPAO SPECT showed almost normal cerebral blood flow (Picture 2). After the examination, her physician in charge found her thrashing in bed, with rhythmic movement of all four extremities, for about 5 minutes. She subsequently appeared groggy and stuporous for about 1 hour and then returned to be completely alert. Recovery of cerebral blood flow shown by Tc-99m HMPAO SPECT seemed to have a close etiologic relationship with this generalized convulsion. We concluded that this convulsion was due to benzodiazepine withdrawal since it was the only seizure.

Physicians who treat acute benzodiazepine intoxication patients who have a history of treatment with benzodiazepine medication without definite history of addiction should be alert for the possibility of withdrawal syndrome (3).

References