to selectively trigger ischemic-hypoxic damage selectively targeting these neural structures. The hippocampal artery supplies an internal anastomosis forming a link between an upper and a lower artery creating a watershed area called "the hypoxia-susceptible sector of Sommer" and it has been previously demonstrated that the CA1 region of the hippocampus is one of the most vulnerable areas of the brain to ischemia.[7]

The patient we described[1] presented no vascular risk factors with the exception of obesity and hyperhomocysteinemia. Data show that patients with TGA do not have more vascular risk factors (i.e., high blood pressure, hypercholesterolemia, diabetes) than control subjects[8] and that they do not show an increased risk of subsequent stroke or TIA[9] with respect to control subjects. Interestingly, our patient also had a phobic personality trait that might represent a predisposing factor for TGA[6] and that might cause a hyperventilation-induced vasoconstriction of cerebral resistance vessels, followed by hemodynamic changes leading to hypoperfusion in memory relevant structures.[6]

As Dr. Schott pointed out, the possibility that an amnesic syndrome of unknown cause might occur as a consequence of neuroinflammation (e.g., due to a fruste form of limbic encephalopathy) should always be considered. Moreover, we agree that MRI data in our case are similar to those seen in limbic encephalitis associated with VGKC antibodies (Abs).[3] Nevertheless, several clinical features of the described case make this diagnosis unlikely.

In the described case, symptoms developed suddenly and the amnesic syndrome resolved spontaneously within 12 hours. Conversely, patients with VGKC Abs-associated encephalopathy usually present with clinical features of a subacute (from weeks to months) amnesic syndrome. They also usually require variable regimens of steroids, plasma exchange, and intravenous immunoglobulin and often present varying degrees of cerebral atrophy and residual cognitive impairment.[2]

The patient's cognitive impairment was limited to amnesia and no additional features that may be present in VGKC-Abs-associated limbic encephalitis (e.g., seizures, hallucinations, behavioral disturbances) were observed.[2] Moreover, the patient we described had a normal EEG, which is often abnormal during VGKC-Abs-associated limbic encephalitis, showing generalized slowing with, in some cases, focal temporal sharp waves.[2,3] Finally, low plasma sodium concentrations were not found in our patient but are a common feature in VGKC-Abs-associated limbic encephalitis.[2,3]

Nevertheless, the spectrum of recognized autoimmune encephalitis (e.g., paraneoplastic limbic encephalitis, Hashimoto encephalopathy, Sjögren syndrome–associated encephalopathy) is growing and inflammatory causes should always be considered in the differential diagnosis of amnesic disorders.

Massimiliano Di Filippo, Paolo Calabresi, MD, Perugia, Italy

Disclosure: The authors report no disclosures.

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CORRECTION

Neurovascular unit dysfunction: A vascular component of Alzheimer disease?

In the Clinical Implications of Neuroscience Research review "Neurovascular unit dysfunction: A vascular component of Alzheimer disease?" by Eduardo E. Benarroch (*Neurology*® 2007;68:1730–1732), the author's name was inadvertently omitted from the byline. Dr. Eduardo E. Benarroch is both author and Section Editor. The publisher regrets the error.

CORRECTION

Neurology 2008;70;1941
DOI 10.1212/01.wnl.0000318085.35893.c6