Falling (or Ascending) Into Oblivion: Transient Global Amnesia With Paragliding

To the Editor: Transient global amnesia (TGA) is a syndrome characterized by an abrupt onset of severe anterograde, and variable retrograde, memory impairment, which is self-limited, leaves no cognitive sequelae, and usually does not recur.[1] Although some precipitants are well established (e.g., exercise and sexual intercourse), its etiology remains largely unknown.[1]

We present a patient in whom TGA occurred when paragliding. A 33-year-old healthy man was brought to our attention by two friends, with whom he had practiced paragliding for many years. On that afternoon, they were flying in a local valley, at an altitude of approximately 2,000 meters. After a 20-minute flight and a flawless descent and landing, he seemed rather anxious and started to repeatedly ask the same questions, like "What am I doing here?" and "How did I get here?". At examination, he was fully alert and well-oriented, slightly impatient, and anxious to know what was happening to him. He could not remember anything from that day or retain any new piece of information. He continued to ask the same questions, incapable of evoking any word from a given list, could not point to purposely hidden objects in the examination room after a 2-minute interval, and was unable to recall any information related to his presence in the hospital. Neurological examination was otherwise normal. Laboratory work-up, brain CT, and EEG were unrevealing. The patient soon started to recover, and was completely back to normal in about 5 hours, retaining a memory gap for this period.

In attempting to explain what happens in TGA, several mechanisms have been proposed: vascular hypoxic or ischemic damage, migraine-like cortical spreading depression, or transient epileptic phenomena, affecting brain regions known to be crucial for episodic memory, in particular, the hippocampal formation.[1] The occurrence of TGA in a high-altitude setting was first noted by Litch in 1999.[2] Since then, apart from his case series of four individuals, all developing symptoms at very high altitudes (above 3,500 meters),[3] we have found only an additional case in a man who got sick while skiing at 2,000 meters.[4] In all these instances, the same pathophysiological cascade was proposed: diminished inspiratory oxygen pressure leading to hyperventilation, which results in hypocapnia and subsequent vasoconstriction, compromising the perfusion of critical brain memory regions or, alternatively, acting as a trigger for cortical depression spreading. Also, as cerebral blood flow is controlled by a fine balance between hypoxic vasodilatation and hypocapnic vasoconstriction, autoregulation abnormalities, as previously postulated to occur in TGA,[5] can further aggravate a net disequilibrium toward cerebral vasoconstriction. Although this can well be what happened to our patient, it should be acknowledged that in this, as well as in other reported cases, it is virtually impossible to discard completely other co-occurring factors, such as physical exertion or emotional stress.

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