Cough-Induced Transient Global Amnesia

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Abstract

Two patients developed acute loss of short term memory of several hours duration while preserving autobiographic memory and exhibiting no focal signs and symptoms following a crisis of severe coughing. The first patient had recurrent allergic bronchitis; the second patient had hereditary pressure palsy neuropathy (HPPN) secondary to a mutation in the PMP 22 gene manifested by protracted cough, eventually ameliorated by the administration of Levetiracetam. Neither of the two patients had history of migraine or epilepsy. Imaging studies failed to show structural or acute vascular lesions on either patient. Symptoms were typical of transient global amnesia (TGA). Because primary TGA (no lesions apparent on imaging) may represent a migraine phenomenon according to previous reports, I propose that these two patients exhibited “cough-induced TGA”, precipitated by a similar mechanism causing cough headache but presenting as pure confusional migraine, or more formally defined, as “cough-induced persistent cognitive migraine aura without stroke.”

Introduction

Transient Global Amnesia (TGA) is a condition defined by the abrupt loss of short term memory while preserving autobiographic memory and in the absence of localizing signs on the neurological examination (1, 2). Often the patients exhibit repetitive queries. It may be confused with transient ischemic attacks (TIAs) or with complex partial seizures (2, 3). Current understanding on the pathophysiology of non-lesional or “primary TGA” is in support that it represents a migraine phenomenon, even that a good number of these patients may show subclinical signs of cardiovascular disease (4, 5). It is possible then to infer that TGA often represents “acephalgic” or “acute confusional migraine” (6). Although an early case study suggested TGA was epileptic in nature, a subsequent study employing routine and 24 hour EEG recordings established the non-epileptic origin of this disorder in the great majority of cases (3, 7). Yet, in the particular patient presenting with acute memory loss to the emergency room, only an ictal EEG will establish with certainty the diagnosis of epileptic amnesia (8).

The overall prognosis of TGA is good. Recurrence rate of TGA is approximately of 22 % in five years, and exceptionally occurs more than twice in a patient lifetime (9). Multiple triggers for TGA have been identified: from trivial head trauma and sexual activity, to exercise and exposure to cold water (10).

Exceptional patients with acute intracranial bleeding may present with symptoms of TGA (“secondary TGA”). A 68 year old male with acute onset of transient short term memory loss due to an intracranial left frontal hemorrhage was reported by Jacome and Yanez (11). A temporary disconnection syndrome affecting the basal forebrain-hippocampus pathways was suggested as the potential underlying mechanism.

The Valsalva maneuver has been adduced frequently as the pathogenic precipitant of TGA resulting from physical and sexual activity, but the development of TGA following persistent cough has not been singled out. Although altitudinal TGA, probably mediated by hypoxia and brain edema, has been described before (“Mountain TGA”), the second patient that had TGA while visiting a city in South America located at high altitude, only developed her symptoms following a severe coughing crisis (12).

Case Report(s)

Case One: A 71 year old female was seen in neurological consultation a few days after developing acute short term memory impairment without loss of self-identity and with preservation of automatic behavior, comprehension of language and fluent speech. She reported no generalized weakness, sudoration or lightheadedness with the episode. She experienced no syncope. The episode lasted several hours and was precipitated by a bout of severe persistent dry coughing. She had history of chronic allergic bronchitis, bipolar affective illness-depressive type, meralgia paresthesica, right sciatica and unilateral restless leg syndrome sometimes interfering with her sleep. She had no cardiac arrhythmia and she was hypothyroid. She had no history of epilepsy, migraine or similar episodes of short term amnesia. Her father had polyneuropathy but there were no other neurological disorders on her family. On neurological examination she had diminished sensory perception over the right thigh and brisk reflexes. Head CT scan, neck and brain MRA were normal following her...
memory loss. Her brain MRI showed limited non-specific white matter T2 hyperintensities and mild cortical atrophy. After nineteen months she has had no recurrent episodes suggestive of TGA.

Case Two: A 68 year old female developed acute short term memory loss with no other symptoms lasting for less than 24 hours following a severe coughing crisis. She reported having no lightheadedness, weakness, generalized sweating or a fainting sensation during the spell. She was visiting La Paz, Bolivia, a city located at high altitude in the Andes Mountains of South America. She had history of chronic dry cough for many years. All her detailed cardiopulmonary investigations in the past had been negative. She suffered from ataxia of gait and had left leg pain. She had chronic insomnia due to obstructive sleep apnea and depression. She had no cardiac arrhythmia. She had no history of epilepsy or migraine and no family history of neurological disorders. She reported chronic urinary incontinence, tingling of the fingers and mild tremors of the fingers with sustained action. On neurological examination she exhibited periodic blepharospasm, congenital bilateral alternating exotropia, ataxia of gait and areflexia. EEG three weeks following the incident was normal. Her brain MRI showed limited non-specific white matter T2 hyperintensities. The electromyogram (EMG) was normal. Her nerve conduction velocities (NCV) showed evidence of severe axonal polyneuropathy. Genetic testing was positive for a mutation on her PMP-22 gene compatible with Charcot Marie Tooth disease type I-A or HPPN. She was prescribed Levetiracetam at a dose of 500 mg b.i.d., with amelioration of her coughing crisis. In 2 years follow up she reported no further episodes of transient amnesia.

Discussion

According to the International Headache Classification 2 (IHC-2- ), cough headache is of sudden onset lasting from seconds to 30 minutes. It is brought on by coughing, straining and the Valsalva maneuver (13). Primary and secondary forms (for instance due to posterior fossa tumors or Chiari I malformation) are recognized (13, 14). A malignant form is exceptional, such as in cases of occult post-surgical sinus CSF fistula resulting in infectious meningitis (15). It is conceivable that a patient with patent foramen ovale (PFO) may experience CNS microembolization during coughing episodes triggering a behavioral response simulating confusional migraine and TGA (16). A positive modified Valsalva test in patients with cough headache may identify those with intracranial posterior fossa pathology, in other words, as having “secondary cough headache” (14). Of relevance, none of the two patients herein described suffered from migraine or reported headache following their transient amnesia. Persistent cough or coughing crisis may be an exceptional sign of a CNS disorder, i.e., MS or spinocerebellar degeneration (17).

Protracted cough is an unusual manifestation of hereditary peripheral neuropathy (18). It is of interest that Case 2 had genetically confirmed mutation on the PMP2 gene, probably predisposing her to suffer from chronic cough, in the absence of alternative cardiopulmonary explanations. I do not know of a similar occurrence reported previously.

Rarely premature ventricular contractions (PVCs) precede coughing in exceptional individuals (19). Coughing is triggered by the sudden increase in pulmonary blood flow as the result of the stronger ventricular contraction that follows the cardiac pause that allowed previously greater diastolic ventricular filling. The compensatory increase in blood flow stretches the pulmonary artery branches stimulating the vagus nerve cough-inducing sensors, located at the vicinity of the main arterial bifurcation (19). However, none of the two patients herein described, had cardiac arrhythmia.

Diagnostic imaging is indicated in patient with TGA since rarely TGA may be secondary to brain lesions. MRI-MRA findings reported in previous studies in non-lesion cases include delayed abnormal hippocampal hyperintensity in MRI diffusion weighted imaging (DWI), and abnormal cerebral venous return, as documented by MRA-TOF (20, 21). According to Sedlaczek, et al, delayed ischemic hippocampal damage is present in most patients with TGA when the study group is compared with a normal aging population, making TGA not always inconsequential (22).

As mentioned above, current knowledge favors the notion than TGA constitutes a form of migraine at least in a significant number of examples. In a landmark publication, Crowell, et al, described 12 patients with headache associated to TGA (4). Four of the twelve patients had history of migraine. Regional cerebral blood flow (rCBF) abnormalities in the study group consisted of vasomotor responses in the watershed areas between the middle and the posterior cerebral arteries; focal ischemia over the ventral-inferior temporal lobe was present in five patients of a sub-group of seven subjected to rCBF. These findings were atypical for TIA based on CBF criteria, hence, by
default, suggestive of a migraine process (4). An
association between TGA and migraine, mediated by
stress-induced hippocampus glutamate release
resulting in cortical spreading depression (CSD), was
previously proposed by Olesen and Jorgensen (23).

The pathophysiology of TGA has been normally
explained on basis on its trigger or underlying
disorders, i.e., intracranial bleed, epilepsy or migraine.
It is likely than several elements may participate on its
appearance, since these elements are not mutually
exclusive. For instance, migraine can precipitate
seizures, seizures can cause post-ictal TGA and
history of migraine may facilitate the eventual
appearance of TGA in a given subject. Given the
potential occurrence of migraine without headache as
a persistent aura, in particular in older patients
("migraine with late accompaniments"), and the known
association between CSD, migraine and ischemia, as
well as between cough and headache, it is legitimate
to speculate that abrupt and sustained changes in
cerebral venous return during prolonged coughing in
these two patients, resulted in unilateral hippocampus
ischemia and TGA, as an isolated persistent cognitive
migraine aura (24).

Conclusion

It is suggested that selective transient ischemia of the
hippocampus during coughing crisis may
precipitate TGA in exceptional patients, as a form of
cough-induced persistent cognitive migraine aura
without stroke.

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