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Anterograde Amnesia after Methamphetamine Lab Exposure

Abstract

Anterograde Amnesia after Methamphetamine Lab Exposure Background: Some known toxic exposures can produce detrimental cognitive deficits. Yet, very few substances have been linked to the development of anterograde amnesia. Here, we report a novel case of a middle-age man who presented with a profound and apparently permanent anterograde amnesia following his exposure to a clandestine methamphetamine laboratory. Case Report: A 47 year-old, right-handed man presented in 2013 with a history of memory loss and seizures since 2001, when he was exposed during his work as a firefighter to the fumes of spilled material intended to synthesize methamphetamine in a clandestine laboratory. Following the exposure, he suffered a major and immediate impairment of multiple cognitive domains, with anterograde memory being most impaired. A formal neurobehavioral status examination NBSE showed a profound impairment in episodic/verbal memory and lesser impairment of executive function. The patient clinical course has to date been stable without further deterioration or improvement. Discussion: This case highlights a clear temporal relationship of methamphetamine laboratory exposure and the development of deficits in memory and executive dysfunction suggestive of involvement of both prefrontal-striatal circuits and medial temporal areas. This is in concordance with prior animal studies, and warrants further investigation in the future.

Keywords: Amnesia; Anterograde; Methamphetamine; Toxicology

Case Report

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Awss Zidan, Devin Burke and Amy E Sanders*

Department of Neurology, SUNY Upstate Medical University, USA

***Corresponding author:** Awss Zidan M.D., Department of Neurology, SUNY Upstate Medical University, 750 E Adams St, Syracuse, NY, 13210, USA, Tel: (315)464-5357, (347)513-4610; Fax: (315)464-5355; Email: Zidana@upstate.edu

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Background

Medical knowledge of the anatomical and functional substrates of anterograde memory has largely been elucidated through unplanned loss-of-function cases. Classic examples include H.M, who underwent an excisional surgery of the temporal lobes [1], and R.B, who suffered a bilateral ischemic hippocampal injury. [2]. Both had a profound inability to encode new memories. In the last decade, it has been reported that toxic exposures can be another mechanism for the development of anterograde amnesia [3]. Here, we report a novel case of a 35 year-old man who developed an immediate and apparently permanent anterograde amnesia following his exposure to a methamphetamine laboratory.

Case Report

JS, A 35 year-old right-handed man who presented to our center in May 2013 with a history of memory loss and seizures since 2001, when he was exposed during his work as a firefighter to the fumes of spilled material intended to synthesize methamphetamine in a clandestine laboratory. Following the exposure, JS lost consciousness, although no trauma was involved Following his hospitalization and recovery, he started experiencing stereotypical spells of "out-of-body experience" or stating that were followed at times by generalized shaking. He also developed angry outbursts and depressed mood. He suffered a major and immediate impairment of multiple cognitive domains following the injuries, with anterograde memory being most impaired; e.g. he could not remember dates or names for more than a few minutes. His remote memory remained intact and he was able to retain all of his acquired procedural skills and knowledge up to the time of the injury. Although there has been no improvement in his memory impairment since the time of the injury, he has learned to cope with his limitations. His family provides him with written notes, organizing his chores and tasks, such as shopping or household maintenance, on a daily basis; he is able to follow these instructions. He is able to drive with the help of a global positioning system (GPS) device.

An electroencephalogram at JS' initial presentation showed frequent focal spikes of 20-40 microvolts originating from the right temporal region; a finding that correlated with the MRI appearance of bilateral flair intensity in hippocampal areas. Trials of valproic acid, phenytoin, and topiramate were undertaken and, later, discontinued due to side effects or inefficacy. At the time of his presentation to our clinic in 2013, he was managed solely on Lacosamide. His last seizure was in 2009.

A formal neurobehavioral status examination (NBSE) conducted in early 2015 showed a profound impairment in episodic/verbal memory and to a lesser extent in sustained and complex attention, executive functioning and verbal fluency (Table 1). Hold tasks (such as literacy) and fund of knowledge/

long term memory appeared to be relatively unaffected. He is almost entirely independent in the management of his activities, and shows no evidence of continuing or progressive functional decline. JS continues to be seen in follow-up in the neurology clinic, and his clinical course has to date been stable without further deterioration.

Table 1: The result of Neurobehavioral Status Exam (NBSE) of our case.

	Score	Interpretation
Blessed Information-Memory-Concentration test (BIMC) [4]. Screening test of general cognition.	14	Impaired Normal: 0-8
Digit Span (DS) [5]. Forward DS assesses simple auditory attention; backward DS also incorporates working memory	Forward: 5, Backward: 4	Borderline
Trailmaking Test Part A (Number Sequencing; sustained attention) [6].	129 sec	Severly impaired z = -12.16
Trailmaking Test Part B (Alternating Number-Letter; complex attention with set-shifting) [6].	Unable to complete	Severely impaired
Memory Impairment Screen MIS (list-learning episodic memory) [7].	0	Severaly impaired Normal: 5-8
WMS-R Logical Memory (Story A) (paragraph recall episodic memory) [8].	25-Jan	Severely impaired z = -3.56
Semantic Fluency (Animal Naming in 60 seconds) [9].	7 Animals	Severely impaired z = -3.09
Phonemic Fluency (F, A, S; 60 seconds per letter) [10].	F:5, A: 4, S: 7 Total FAS: 16	Moderately impaired z = -2.25
Rapid Estimate of Adult Literacy in Medicine (REALM) [11].	66	High-School Level Health literacy
Clinical Dementia Rating CDR [12].	1	0: No Impairment 0.5: Very Mild Impairment 1: Mild Impairment 2: Moderate Impairment 3: Severe Impairment

Discussion

The anatomical core of the episodic memory system is the medial temporal lobe and hippocampus [13]. Clandestine methamphetamine labs are known to produce phosphine, iodine, hydrochloric acid, various solvents, methamphetamine and methamphetamine intermediates [14]. A descriptive study performed by the Washington State Poison Control Center reported the health outcomes of people exposed to clandestine methamphetamine labs most commonly to be headaches, respiratory/eye irritation, nausea, and vomiting [15]. Interestingly, a retrospective chart evaluation of 66 police officers exposed to clandestine methamphetamine labs reported memory loss in up to 77%, but without further description [16].

Methamphetamine is a known neurotoxin that imposes cellular damage through numerous mechanisms, including oxidative stress, excitotoxicity, hyperthermia, neuroinflammation, mitochondrial dysfunction, and endoplasmic reticulum stress [17]. Animal models of methamphetamine use have shown a marked neurodegeneration in the limbic system, including hippocampus, in association with the limbic-originated seizures [18]. The breakdown of the blood-brain-barrier and the activation of glial cells by the released neurotransmitters are some of the hypotheses that were proposed for the degeneration [19].

A deficient strategic (i.e. executive) control of verbal encoding and retrieval has been noted by previous studies done on human subjects with history of methamphetamine dependence, but this was proposed to be the sequelae of methamphetamine-related prefronto-striatal circuit neurotoxicity rather than hippocampal involvement [20]. Yet, another small study on 12 subjects with methamphetamine dependence revealed that they matched their controls in all cognitive domains testing, except for verbal memory [21].

The results of these studies are consistent to a certain extent with JS, who manifested deficits in memory and executive dysfunction suggestive of involvement of both prefrontal-striatal circuits and medial temporal areas. Yet, our case also revealed evidence of striking– and apparently permanent-- damage to anterograde memory, which was not seen in previous studies.

We acknowledge that the NBSE may have been, at least partially, affected by exposure to some other unidentified substances at the time of the injury, or by Lacosamide which the patient was taking at the time of his cognitive evaluation. However, this striking clinical picture is unlikely to be the result of these factors alone.

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