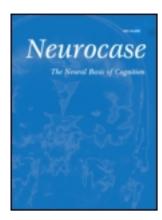
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Aurélien Benoilid $^{\rm a\ c}$, Nicolas Collongues $^{\rm a\ b\ c}$, Jérôme de Seze $^{\rm b\ c}$ & Fréderic Blanc $^{\rm c}$

 $^{\rm a}$ Department of Neurology , Strasbourg University Hospital , Strasbourg , France

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 $^{^{\}rm b}$ Strasbourg University Hospital and Centre d'Investigation Clinique INSERM 10002 , Strasbourg , France

^c Laboratoire d'Imagerie et Neurosciences Cognitives (LINC), UMR 7237, University of Strasbourg and CNRS, Strasbourg, France Published online: 25 May 2012.



Heroin inhalation-induced unilateral complete hippocampal stroke

Aurélien Benoilid^{1,3}, Nicolas Collongues^{1,2,3}, Jérôme de Seze^{2,3}, and Fréderic Blanc³

¹Department of Neurology, Strasbourg University Hospital, Strasbourg, France

A 33-year-old man presented to our clinic with amnesia 48 hours after his first heroin inhalation. Examination showed lateral tongue biting and anterograde amnesia demonstrated by impaired performance on verbal and visual Wechsler Memory Scale–Revised tests carried out 10 days after onset, suggesting hippocampal involvement. Magnetic resonance imaging (MRI) of the brain was performed 48 hours after heroin snorting and evoked cortical laminar necrosis (CLN) of the left hippocampus without vascular abnormality. This is the first description of complete hippocampal CLN as a complication subsequent to acute intranasal heroine abuse. While the pathogenic mechanism remains uncertain, our case provides a very specific MRI lesion pattern and highlights the risk of intranasal heroin uptake-induced neurological complication.

Keywords: Heroin; Stroke; Hippocampus; Seizure; Amnesia.

A right-handed 33-year-old man presented to our clinic with amnesia 48 hours after his first heroin inhalation. Examination showed lateral tongue biting and anterograde amnesia. Extensive neuropsychological examination showed impaired verbal memory with impaired free recall and recognition at the '15 mots de Rey' verbal memory test (Rey, 1958), no impaired visual memory recognition (DMS 48) (Barbeau et al., 2004). The forward verbal digit span was normal (8 figures) but the backward one impaired (4 figures) and visual-motor digit span was normal. Attention capacities were normal on test of attentional performance (TAP, including the following subtests: divided attention, flexibility, Go/No go, alertness, sustained attention) (Sturm & Willmes, 1993). Trail Making Test (TMT) A and TMT B showed a mild diminished speed of information processing (56 and 126 seconds, respectively, without any mistake). Verbal fluencies were impaired for the semantic one, but not the literal one. The Stroop test, the Rey—Osterrieth complex figure copy and capacities of language (tested with the DO80 picture naming test) (Metz-Lutz, Kremin, & Deloche, 1991), were normal. Wechsler Memory Scale—Revised (WMSR) score carried out 10 days after onset, demonstrated impaired performance in verbal (68) and visual (61) abilities. WMSR is not really able to distinguish between the right/left temporal involvement (Moore & Baker, 1996).

Thus, neuropsychological tests results with verbal memory impairment were compatible with an involvement of the left hippocampus. Biochemical examination revealed that serum creatine kinase

Address correspondence to A. Benoilid, Department of Neurology, Strasbourg University Hospital BP 426, 67091 Strasbourg, France. (E-mail: aurelienbenoilid@gmail.com).

²Strasbourg University Hospital and Centre d'Investigation Clinique INSERM 10002, Strasbourg, France

³Laboratoire d'Imagerie et Neurosciences Cognitives (LINC), UMR 7237, University of Strasbourg and CNRS, Strasbourg, France

was increased to 1643 U/L, first glucose and sodium levels were normal and no circumstantial evidence of alcohol intake was observed. Cerebrospinal fluid analysis showed a protein level of 89 mg/dL and 1 nucleated cell. Magnetic resonance imaging (MRI) of the brain was performed 48 hours after heroin snorting (Figure 1) and evoked cortical laminar necrosis (CLN) of the left hippocampus without vascular abnormality. Investigations excluded endocarditis or mycotic aneurysms.

CLN is a brain injury pattern corresponding to selective neuronal laminar pan-necrosis of affected cortical areas, usually described in association with a variety of disorders, such as cerebral hypoxia or ischemia and more rarely observed in metabolic, immunologic, infectious, or mitochondrial impairment. Intoxication-induced CLN has previously been observed after alcohol and immunosuppressor intake, but never as yet after heroin intake.

However, heroin abuse is implicated in several cerebral lesions: ischemic stroke is almost always observed further to intravenous injection (Caplan, Hier, & Banks, 1982). Intranasal administration is a route usually considered as 'safe', and even used for acute pain management. Yet, a few

publications dealing with intranasal administration have reported ischemic strokes especially in the globus pallidus region (Zuckerman, Ruiz, Keller, & Brooks, 1996).

A distinct toxic spongiform leukoencephalopathy has been described to occur after inhalation of heated heroin (chasing the dragon), evidenced as symmetrical high T2 signal involving the cerebellar hemispheres, posterior cerebrum, internal capsules, and very unusually the hippocampi (Gupta, Krishnan, & Sudhakar, 2009).

Finally, deleterious effects attributable to seizure activity have never been reported after heroin exposure, but unilateral hippocampal laminar necrosis has been previously described following complex partial status epilepticus (Heinrich, Runge, Kirsch, & Khaw, 2007), and our patient presented indirect evidence of seizure.

The major neurologic complications reported to occur with heroin abuse have been associated with intravenous administration. This is the first description of complete hippocampal CLN as a complication subsequent to acute intranasal heroine abuse. While the pathogenic mechanism may arise either directly as a result of the heroin toxicity occurring, or indirectly through a seizure or an ischemic effect,

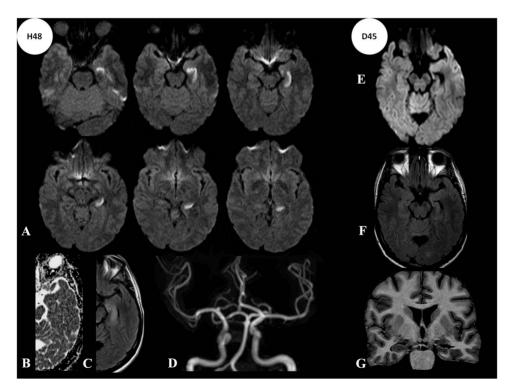


Figure 1. Initial and follow-up brain MRI.

our case provides a very specific MRI lesion pattern and highlights the risk of intranasal heroin uptake-induced neurological complication.

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