

CASE REPORT

Bilateral hippocampal stroke secondary to acute cocaine intoxication

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Abstract

Hippocampal infarction is a rare complication of cocaine use, with only two cases previously reporting this association. We present a 44-year-old male who developed a persistent amnesic syndrome following cocaine intoxication. Examination identified no other neurological deficits. Subsequent MRI brain revealed high FLAIR signals and diffusion restriction in the hippocampus and centrum semiovale bilaterally, consistent with infarction. These findings were in keeping with the results of formal neuropsychological testing where deficits in both verbal and visual episodic memory and learning capacity were identified, consistent with hippocampal dysfunction. In contrast to previous reports, this presentation occurred in the absence of other vascular risk factors or hypoxic insults.

INTRODUCTION

Cocaine is one of the most commonly used illicit drugs worldwide and is associated with a wide range of neurological complications, including ischaemic stroke. We herein report the extremely rare presentation of amnesia associated with bilateral hippocampal stroke secondary to acute cocaine intoxication.

CASE REPORT

A 44-year-old man with a past history of depression and recreational drug use was brought into the emergency department by ambulance after being found drowsy and disoriented on the floor of his home.

On initial assessment the patient was confused with a Glasgow Coma Scale of 12. He was unable to give a coherent history and denied any recollection of the events leading to his presentation to hospital. Vital signs were within normal range other than a mild sinus tachycardia (heart rate up to 120 bpm).

On examination there were no focal neurological findings, basic bloods including full blood count and biochemistry were unremarkable and non-contrast computed tomography (CT) of the brain was normal. Urine drug screen was positive for cocaine metabolites.

Several days into admission, despite being medically stable, his short-term memory remained impaired, with difficulty recalling any recent events since his admission. He was a vague historian, required regular re-orientation and had difficulty remembering visitors and regular healthcare staff. He was alert and cooperative however, and speech, language, thought content and affect were all appropriate. He had a degree of insight into his own short-term memory loss. Full peripheral and cranial neurological examination was unremarkable. Differential diagnosis at this stage included transient global amnesia, toxic leucoencephalopathy and stroke.

Given the patient's persistent cognitive deficits, further neuroimaging with MRI was performed on Day 8 of admission. This revealed high T2/FLAIR abnormality with restricted diffusion in

Received: December 19, 2014. Revised: February 5, 2015. Accepted: February 9, 2015

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hippocampus (Fig. 1) and centrum semiovale (Fig. 2) bilaterally, in keeping with acute infarction. Stroke work-up including ECG monitoring, fasting glucose and lipids, carotid ultrasound and transthoracic echocardiogram returned normal.

Formal neuropsychological assessment was performed on our patient, who prior to this event was a high functioning and educated individual, running a successful small business. Abnormalities identified included severe impairment of memory and learning, with evidence of rapid forgetting and limited benefit from recognition prompts. Episodic memory for new information was particularly impaired. On a verbal list-learning task, the patient was unable to learn past his immediate memory span; spontaneous recall was impaired and the patient could not remember being read the list. Visual memory was similarly impaired in both recall and recognition. Mild episodic retrograde memory difficulties were also noted; however, semantic memory was intact. Working memory was mild to moderately impaired, with slowed information processing with tasks of increasing complexity. Visio-perception and visio-construction ability was intact, as was verbal comprehension, expression and fluency. There was impairment of some aspects of executive function, but this was likely limited by memory deficits.

These findings are consistent with the bilateral hippocampal hypoxic damage identified on neuroimaging. The patient was discharged to a neuro-rehabilitation facility for ongoing management.

DISCUSSION

Ischaemic brain damage secondary to cocaine is a well-recognized phenomenon. In young to middle aged adults, cocaine use is associated with a relative risk of ischaemic stroke six to seven times higher than age-matched controls in the 24 h after use [1].

Various mechanisms are believed to contribute to the increased risk of ischaemic stroke associated with cocaine. Cerebral vasospasm may affect large cranial arteries as well as the cortical microvasculature, while cocaine also increases the risk of vascular thrombosis by potentiating enhanced platelet aggregation and non-laminar flow [2]. Furthermore, cocaine's well-established effects on the cardio-respiratory systems may indirectly result in cerebral ischaemia through cardio-embolic events, arrhythmias and respiratory arrest [3].

The hippocampus plays a critical role in learning, memory and emotional behaviour and is known to be particularly sensitive to hypoxic damage [4]. Hippocampal strokes are usually unilateral and generally occur in the context of posterior cerebral artery territory infarcts. Predominant clinical signs are most commonly related to associated lesions outside the hippocampus and isolated amnesic syndromes secondary to hippocampal stroke are rare occurrences. In one case series of 57 patients with hippocampal infarcts, only 19% showed evidence of hippocampal dysfunction [5].

Documentation of MRI findings and clinical presentation related to hippocampal infarcts are scarce, and our case is the first to provide both detailed neuroimaging and neuropsychological assessment in the setting of hippocampal stroke secondary to cocaine use. MRI imaging in our patient demonstrated high FLAIR signal changes with diffusion restriction in the hippocampi bilaterally, with high signal on DWI and low signal on ADC; findings extremely sensitive to acute ischaemic lesions [6]. Similar changes were noted in the centrum semiovale bilaterally, a region of the brain also highly susceptible to hypoxic damage [7]. None of these changes were appreciable on the CT brain performed earlier on our patient.

Neuropsychological assessment of our patient demonstrated findings consistent with reported effects of hippocampal damage

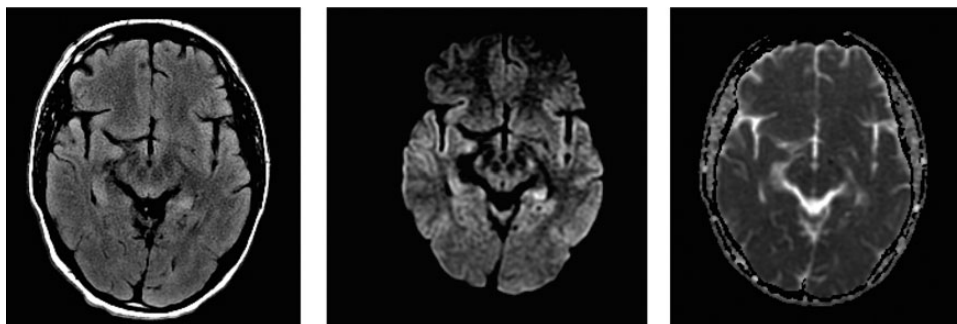


Figure 1: MRI brain—bilateral hippocampal infarcts. MRI brain (axial view) revealed high FLAIR signal changes in bilateral hippocampi and the right uncus (left image) with diffusion restriction—high signal on DWI (middle image) and low signal on ADC (right image).

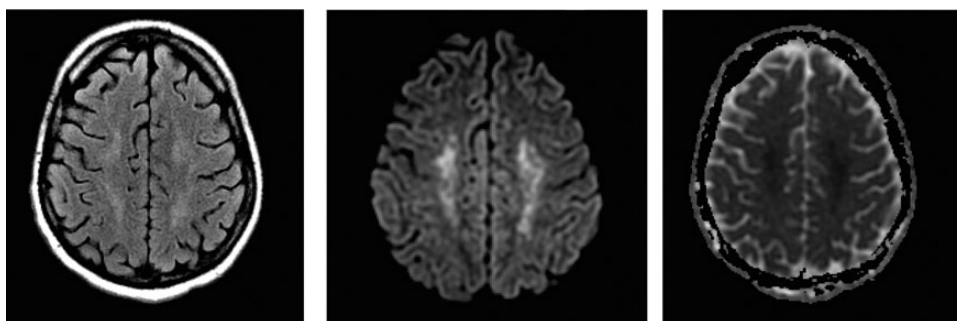


Figure 2: MRI brain—bilateral centrum semiovale infarcts. Corresponding abnormalities in the centrum semiovale bilaterally consistent with acute infarction.

from a range of pathologies [8]. Loss of episodic memory, particularly prominent in our patient, is a constant feature of hippocampal amnesia. Our patient's mild episodic retrograde memory loss with sparing of semantic memory is also in keeping with hippocampal amnesia.

To our knowledge, there are only two case reports in the literature documenting bilateral hippocampal stroke associated with cocaine use. The first case developed hippocampal and basal ganglia strokes secondary to cocaine intoxication complicated by cardiac arrest. In addition to short term memory deficits, the patient also developed dyspraxia and quadriparesis [9]. The second case, similar to ours, presented with disorientation and impaired memory in the setting of bilateral hippocampal infarcts. In contrast to our patient however, this case had multiple ischaemic risk factors in addition to cocaine exposure [10].

CONCLUSION

Hippocampal stroke is a recognised but unusual cause of acute memory impairment. Rarely, presentations of acute hippocampal amnesia secondary to bilateral stroke can be caused by cocaine use in the absence of other ischaemic risk factors, as presented in our case. MRI is a useful tool in this setting to characterise pathology and aetiology. Toxicology screening should also be considered with acute amnesia or stroke, particularly in younger patients with few vascular risk factors.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

1. Saad AQ, Ryan KA, Cole JW, Kitner SJ. Cocaine may increase stroke risk within 24 hours of use. American Stroke Association Meeting Report 2014; Abstract WMP57.
2. Johnson BA, Devous MD, Ruiz P, Ait-Daoud N. Treatment advances for cocaine-induced ischemic stroke: focus on dihydropyridine-class calcium channel blockers. *Am J Psychiatry* 2001;**158**:1191–8.
3. Brown E, Prager J, Lee HY, Ramsey RG. CNS complications of cocaine abuse: prevalence, pathophysiology, and neuroanatomy. *Am J Roentgenology* 1992;**159**:137–47.
4. Fujioka M, Nishio K, Miyamoto S, Hiramatsu KI, Sakaki T, Okuchi K, et al. Hippocampal damage in the human brain after cardiac arrest. *Cerebrovasc Dis* 2000;**10**:2–7.
5. Szabo K, Forster A, Jager T, Kern R, Griebe M, Hennerici MG, et al. Hippocampal lesion patterns in acute posterior cerebral artery stroke: clinical and MRI findings. *Stroke* 2009;**40**:2042–5.
6. Forster A, Griebe M, Gass A, Kern R, Hennerici MG, Szabo K. Diffusion-weighted imaging for the differential diagnosis of disorders affecting the hippocampus. *Cerebrovasc Dis* 2012;**22**:104–15.
7. Beckmann YY, Durakly M, Secil Y, Celebisoy M, Basoglu M. Clinical and radiologic correlations of small and large centrum ovale infarcts. *J Stroke Cerebrovasc Dis* 2010;**19**:220–4.
8. Spiers HJ, Maguire EA, Burgess N. Hippocampal amnesia. *Neurocase* 2001;**7**:357–82.
9. Bolouri MR, Small GA. Neuroimaging of hypoxia and cocaine-induced hippocampal stroke. *J Neuroimaging* 2004;**14**:290–1.
10. Morales SG, Hornik A, Morgan C. Cocaine induced hippocampal infarction. *BMJ Case Rep* 2012;**2012**:bcr0320125998.