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Cocaine-Associated Lobar Intracerebral Hemorrhage in a 42-Year-Old Woman: A Case Report and Literature Review

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Abstract **Case Report**

Spontaneous intracerebral hemorrhage in a relatively young adult warrants a search for an underlying vascular or structural cause, even when exposure to a sympathomimetic drug is documented. We report the case of a 42-year-old woman with depression, reported polysubstance use and chronic tobacco smoking, who was found at home with impaired consciousness after approximately two days without being seen. On admission, her Glasgow Coma Scale score was 11/15, with aphasia and right-sided hemiparesis. Urine screening was positive for cocaine and morphine, with trace benzodiazepines and tricyclic antidepressants. Non-contrast computed tomography showed a left temporo-insulo-parietal intraparenchymal hematoma with perilesional edema and local mass effect, and computed tomography angiography showed no macrovascular lesion. After neurosurgical evaluation, she was managed conservatively; follow-up imaging showed no hematoma expansion, and her neurological status improved gradually until discharge with a rehabilitation plan. Through this case and a focused review of the literature, we emphasize that a positive cocaine screen should not be equated with a proven hemorrhagic mechanism. In younger patients, appropriate vascular imaging, cautious interpretation of drug screening and guideline-based neurocritical care remain essential.

Keywords: Cocaine, Intracerebral hemorrhage, Hemorrhagic stroke, Computed tomography angiography, Substance abuse detection, young adult, Case report.

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INTRODUCTION

Spontaneous intracerebral hemorrhage (ICH) is a potentially devastating form of stroke requiring rapid diagnosis, stabilization, etiological evaluation and prevention of secondary brain injury. In relatively young adults, identifying an underlying vascular or structural lesion is particularly important, because a potentially treatable secondary cause may be present [1,2].

Cocaine is a potent sympathomimetic drug associated with both ischemic and hemorrhagic cerebrovascular events. A systematic review and meta-analysis reported an association between cocaine exposure and stroke, and a more recent meta-analysis estimated increased odds of hemorrhagic stroke among cocaine users (odds ratio 2.05, 95% confidence interval 1.39-3.02) [3,4]. These observational associations, however, do not prove causation in an individual patient.

We report a case of left hemispheric ICH in a 42-year-old woman with cocaine-positive urine toxicology and no macrovascular lesion on computed tomography angiography (CTA), followed by a focused literature review of hemorrhagic mechanisms, neurovascular investigation and neurocritical care in this setting.

CASE REPORT

A 42-year-old woman was admitted to the Department of Anesthesiology and Intensive Care of the Mohammed V Military Instruction Hospital, Rabat, Morocco, after being found at home with confusion and impaired consciousness. Neighbors had reportedly not seen her for approximately two days before alerting her family; the exact time of symptom onset could therefore not be established. No preceding head trauma or witnessed seizure was reported. Her history included depression under pharmacological treatment, reported recreational cocaine and other substance use, and chronic tobacco smoking. Benzodiazepine and cannabis exposure were mentioned, but a complete medication list and the timing or route of cocaine consumption were not available.

On arrival, her Glasgow Coma Scale score was 11/15, with aphasia and right-sided hemiparesis. Initial

laboratory investigations showed no major electrolyte abnormality or coagulation disorder. Qualitative urine toxicology screening was positive for cocaine and morphine, with trace reactions for benzodiazepines and tricyclic antidepressants; it was negative for amphetamines, barbiturates, cannabis, methadone, methamphetamine, phencyclidine and 3,4-methylenedioxymethamphetamine. The time of urine collection relative to possible analgesic administration was not documented, so the morphine-positive result cannot be confidently attributed to non-medical opioid use.

Initial non-contrast brain computed tomography (CT) showed a lobulated, heterogeneous intraparenchymal hematoma centered on the left temporal lobe and insula, with small satellite hyperdense foci in the posterior temporal region, perilesional hypodense edema, effacement of the left sylvian fissure and partial compression of the left lateral ventricle (Figures 1A and 1B). There was no intraventricular extension, hydrocephalus, diffuse subarachnoid hemorrhage or herniation. Contrast-enhanced CT showed normal opacification of the intracranial arteries, with no abnormal vascular structure adjacent to the hematoma (Figure 1C).

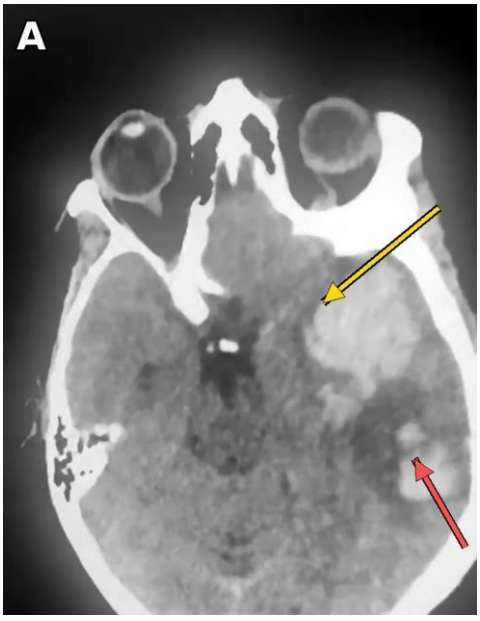


Figure 1A: Axial non-contrast CT at the level of the temporal lobes. Lobulated, heterogeneous hyperdense hematoma in the left temporal lobe (yellow arrow), with small satellite hyperdense foci in the posterior temporal region (red arrow) and surrounding hypodense edema.

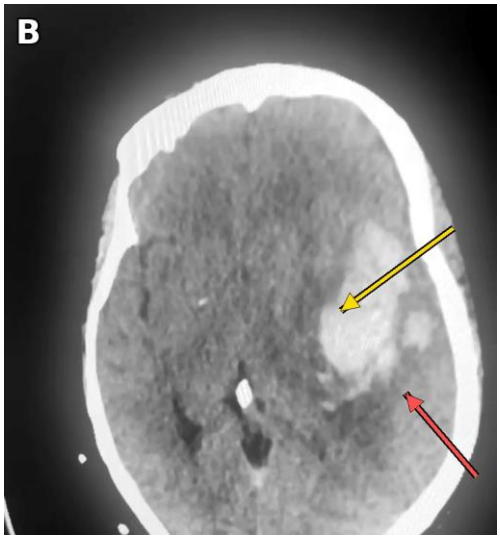


Figure 1B: Axial non-contrast CT at the insular level. Left temporo-insular hematoma at its maximal extent (yellow arrow), with perilesional hypodense edema (red arrow), effacement of the left sylvian fissure and partial compression of the left lateral ventricle, without intraventricular extension.

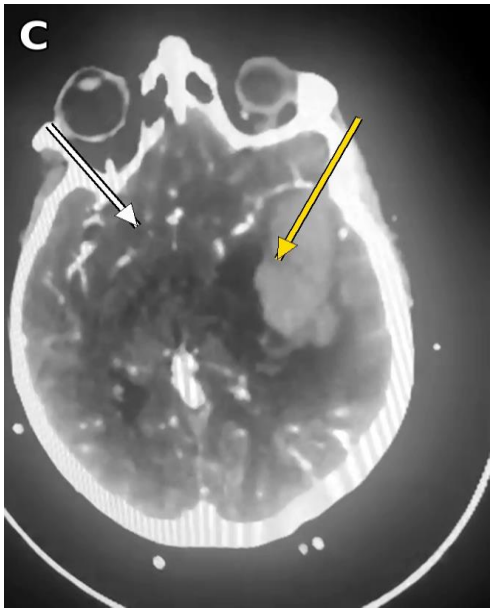


Figure 1C: Axial contrast-enhanced CT. Normal opacification of the intracranial arteries, including the middle cerebral artery branches (white arrow), with no abnormal vascular structure, nidus or early draining vein adjacent to the hematoma (yellow arrow).

Neurosurgical consultation identified no indication for emergency hematoma evacuation. The patient received conservative treatment comprising repeated neurological assessment, antihypertensive treatment for blood pressure control, analgesia, gastric protection, mechanical venous thromboembolism prevention with compression stockings, and levetiracetam. No clinical seizure occurred during admission.

Follow-up brain CT showed no hematoma expansion or new intracranial complication. Cerebral CTA was negative for aneurysm, arteriovenous malformation, dural arteriovenous fistula or other macrovascular lesion. Magnetic resonance imaging, venous imaging and catheter angiography were not performed. The patient remained clinically and radiologically stable, with gradual improvement in consciousness and motor function. She was discharged with a rehabilitation program and outpatient neurological follow-up; psychiatric and addiction follow-up were arranged.

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images. Institutional ethics committee approval was not required for a single anonymized case report in accordance with local policy.

DISCUSSION

Cocaine increases synaptic catecholamine concentrations through inhibition of monoamine reuptake. Acute sympathetic activation may cause abrupt hypertension and cerebral vasoconstriction, and altered cerebrovascular autoregulation and endothelial dysfunction are plausible contributors to vascular injury [5]. In a pathological series, hypertensive cardiovascular disease was more frequent among patients with cocaine-associated intraparenchymal hemorrhage than among those with aneurysm rupture, suggesting that underlying vascular susceptibility may modify hemorrhagic risk [6]. In our patient, the interval between cocaine consumption and hemorrhage onset was unknown, and neither a cocaine-induced hypertensive crisis nor a drug-specific mechanism was established. The term "cocaine-associated" is therefore more defensible than "cocaine-induced".

Published cohorts give heterogeneous results. Martin-Schild et al. compared 45 cocaine-positive with 105 cocaine-negative patients with spontaneous ICH: the cocaine-positive group had more frequent intraventricular extension (71.1% versus 39.0%) and higher in-hospital mortality (31.1% versus 14.3%), although the retrospective design limits causal inference [7]. In contrast, Bajwa et al. studied 102 patients, including 20 cocaine-positive individuals, and found more subcortical hemorrhages but no significant difference in mortality or intraventricular extension [8]. Our patient had a lobar hemorrhage without ventricular extension, which illustrates that no anatomical distribution is diagnostic of cocaine-mediated bleeding.

A negative CTA does not complete the etiological evaluation. A young patient with non-traumatic ICH merits consideration of aneurysm, arteriovenous malformation, dural fistula, cerebral venous thrombosis, cavernous malformation and underlying tumor. In the prospective DIAGRAM study, sequential vascular investigations improved the detection of macrovascular causes beyond CTA alone [9]. Small series have also identified vascular malformations in selected young adults presenting with

hemorrhage after recreational drug exposure [10], and magnetic resonance imaging adds diagnostic value in young adults with apparently spontaneous ICH, particularly for cavernous malformations [11]. In our patient, the negative CTA made a major macrovascular cause less likely but could not exclude occult lesions or venous pathology; additional imaging should be decided by the neurovascular team rather than omitted because urine screening was cocaine-positive [1,2].

Toxicological results also require cautious interpretation. Routine urine immunoassays are qualitative screening methods with variable detection windows and possible cross-reactivity; a positive test does not establish intoxication at stroke onset [12]. Here, cocaine positivity supports recent exposure but not its timing, and the morphine result cannot be interpreted without the medication administration record. The combined history of depression, smoking and polysubstance exposure further limits causal attribution.

Regarding management, current guidelines emphasize individualized blood pressure control, monitoring for neurological deterioration, treatment of hydrocephalus or raised intracranial pressure when present, and structured recovery planning. They do not recommend routine prophylactic antiseizure medication in patients without seizures, and favor intermittent pneumatic compression for venous thromboembolism prevention in non-ambulatory patients [1,2]. In resource-limited settings, local availability and institutional protocols may influence these choices. The main limitations of this report are the unknown timing of symptom onset and cocaine exposure, incomplete documentation of admission hemodynamic parameters and stroke severity scores, the absence of confirmatory toxicological testing, the lack of magnetic resonance or venous imaging, and the absence of long-term functional follow-up.

Conclusion

A positive cocaine screen is clinically relevant in a young patient with non-traumatic intracerebral hemorrhage, but it does not prove that cocaine caused the bleeding. A negative CTA reduces, but does not eliminate, the likelihood of a structural or vascular cause. Toxicological history should be combined with appropriate neurovascular imaging, guideline-based neurocritical care and addiction follow-up.

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