

**Case Report**

# Subarachnoid Hemorrhage after Amphetamine Exposure: A Case Report

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## Introduction

There is increasing evidence that amphetamine and methamphetamine use can predispose to hemorrhagic stroke [1,2]. While the exact mechanism remains uncertain, it is postulated that amphetamine-related compounds can lead to aneurysmal formation and early rupture.

We report a case of Subarachnoid Hemorrhage (SAH) after amphetamine exposure and hope to raise awareness of this under-recognized risk among clinicians in Asia.

## Case Report

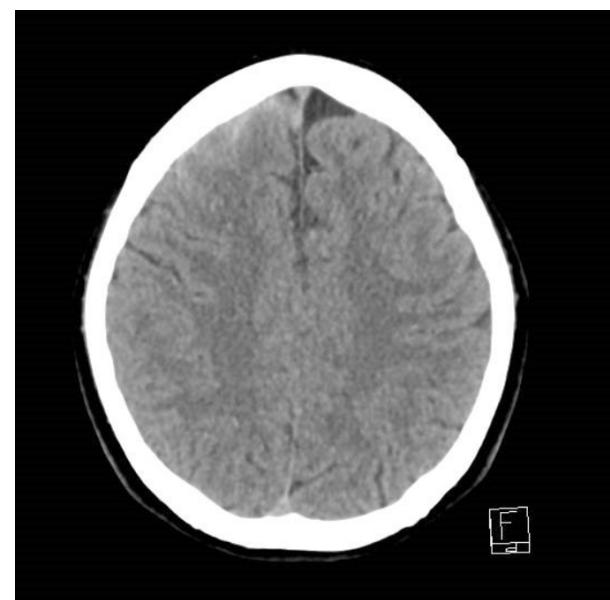
A 25-year-old lady of Chinese ethnicity presented to emergency services with a first-episode seizure after ingesting one-and-a-half tablets of amphetamine, one tablet of 3,4-methylenedioxymethamphetamine (MDMA) and 60 mg of codeine. The generalized tonic-clonic seizure lasted 1 minute and was followed by post-ictal drowsiness.

Although she had previously taken MDMA recreationally, this was her first amphetamine exposure. There was no associated fall or trauma. She had no past medical history. On admission, she was alert and calm. Her vital signs were normal. Physical examination including neurological examination was normal.

Full blood count, renal and hepatic biochemical investigations were unremarkable. Creatine kinase was raised and peaked at 1092 U/L (range: 44-201 U/L).

Chest radiograph and electrocardiogram were normal. Computed Tomography (CT) scan of the brain showed a sliver of

acute subarachnoid hemorrhage overlying the right frontal lobe (Figure 1). CT angiogram of the circle of Willis did not show evidence of vascular malformation or aneurysm. CT venogram did not show venous sinus thrombosis.



**Figure 1:** Computed tomography scan of the brain showed a sliver of acute subarachnoid hemorrhage overlying the right frontal lobe.

She was diagnosed with SAH secondary to amphetamine and MDMA use. Her Creatine Kinase levels normalized with intravenous hydration. She had no further seizures and was discharged well after conservative management.

## Discussion

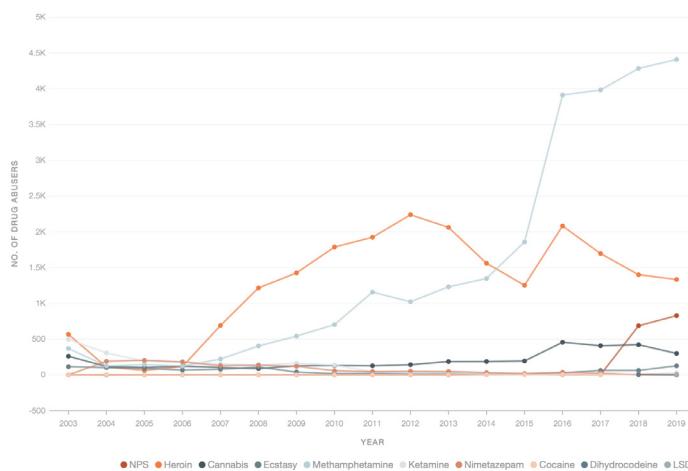
In a case series [3] of nine patients with SAH associated with methamphetamine exposure, all had intracranial aneurysms. In another review [1], 17% of methamphetamine-associated SAH cases had aneurysms, 8% had arteriovenous malformations, and a third had other vasculature abnormalities. These are attributed to chronic insults to the vasculature due to recurrent systemic hypertension from chronic exposure to amphetamine-related compounds. 47% had no vasculature abnormalities.

Although MDMA use is comparatively less commonly associated with hemorrhagic stroke [2], it has been postulated to cause acute sympathetic surges and weakening of the vessel wall leading to rupture, especially in the setting of an aneurysm [2].

Our patient was a first-time user of amphetamine, consistent with McEvoy's [2] observation that hemorrhagic stroke can occur after a single dose of amphetamine. In the absence of aneurysms or vascular malformations, amphetamine-induced SAH may be caused by necrotizing angiitis and/or sympathetic surge with resultant hypertension and tachycardia [1].

Atraumatic and non-aneurysmal sulcal SAH, as in this case, is rare and has a wide spectrum of possible etiologies [4]. This particular type of SAH is atypical in amphetamine-induced cases and hitherto there has only been one reported case of sulcal SAH caused by intravenous amphetamine use [5].

There is increasing prevalence of amphetamine-type stimulants use in East Asia and South East Asia [6], driven by unprecedented availability and increased affordability. This development poses serious challenges for public health in the region. In Singapore, methamphetamine has become the most commonly abused substance [7] (Figure 2).



**Figure 2:** Trend of substance abusers by major drugs from 2003 to 2019 (Singapore) [7].

While it is increasingly recognized in the West that exposure to amphetamine and related compounds can predispose to SAH, this association may be less widely known among clinicians in Asia.

There is a need for wider recognition of the increased risks of intracranial hemorrhage in this clinical subgroup, who are often young and otherwise healthy. Recreational drug users should be warned about this risk as part of public health education efforts.

Our patient's seizure occurred immediately after drug ingestion and she had no pre-existing intracranial pathology. Notably, she did not complain of headache. As such, the diagnosis of SAH might have been missed if she was diagnosed with an uncomplicated drug-induced seizure.

Full neurological examination should be performed for patients suspected to be intoxicated with stimulants. If neurological features such as reduced consciousness, seizures, headache, neurological deficits or signs of meningism are present, further neuroimaging is warranted.

Other than its clinical value, this case echoes calls to reconsider the arbitrary distinction of neurology and psychiatry. Both disciplines developed from a common epistemological perspective and were thought to be part of a single unified branch of medicine-neuropsychiatry. A schism emerged as understanding of the biological basis for some conditions improved, alongside advancement in neuroimaging.

In academic and clinical centers, the divergent trajectories of neurology and psychiatry has led to reduced collaboration in research, medical education and fragmented patient care. Recent research indicated that early career psychiatrists had reservations about their neurology training and felt ill-equipped to handle the neurological aspects of psychiatric practice [8].

Notwithstanding barriers to the reintegration of the two sciences, it is crucial for psychiatrists to reacquaint themselves with a certain level of neurological expertise. Standalone addiction services, traditionally staffed with psychiatrists, should be equipped to manage neurological emergencies due to substance use.

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