



CASE REPORT

Methamphetamine Induced Intracerebral Hemorrhage: A Case Report

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ABSTRACT

Methamphetamine is a psychostimulant agent which induces the release of central and peripheral monoamines. The incidence rate of methamphetamine abuse has been increasing. The negative side effects of methamphetamine abuse which include several disabilities and death, are more common in patients who are younger than 45 years of age. Methamphetamine abuse increases the risk of stroke as well as being in closely associated with the development of ischemic heart disease and atherosclerotic coronary artery disease. In both types of methamphetamine-induced stroke, the possible mechanism is accepted to be cerebral vasculitis. Our study aims to report a rare and lethal case of methamphetamine-induced massive intracerebral hemorrhage with ventricular expansion. As far as we know, our study is the first case report about the lethal methamphetamine consumption reported in Turkey.

Keywords: Methamphetamine, intracerebral hemorrhage, stroke, substance abuse

INTRODUCTION

Methamphetamine is a psychostimulant drug that induces the central and peripheral monoamine release (1). Methamphetamine was first synthesized by Nagai Nagayasi in 1893, 6 years after the discovery of the amphetamine (2). Akira Ogata synthesized crystalized methamphetamine via degrading ephedrine by using red phosphorus and iodine (2). Methamphetamine which has various names street names such as "meth", "ice" and "crystal", has become the most frequently used drug in a short amount of time.

Methamphetamine abuse is an important public health issue. Methamphetamine leads to serious physical

and mental health problems including psychosis and cardiovascular and cerebrovascular diseases (3). According to the drug report of the United Nations (4) there are more than 37 million methamphetamine users worldwide and the annual incidence is 0.77%.

Methamphetamine can be consumed via oral intake, intravenous application, inhalation and in cigarette form. Its effects begin 5 minutes after snorting, 20 minutes after oral consumption and right after the consumption if inhaled or injected (5). The elimination half-life of the methamphetamine is estimated to be more than 12 hours and it is detectable in the urine for 3-5 days after the intake, however this duration may vary according to the consumption method (5).

Here, we aim to report a rare and lethal case of methamphetamine-induced massive intracerebral hemorrhage with ventricular expansion. To the best of our knowledge, our patient is the first case report about the lethal methamphetamine consumption in Turkey.

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CASE PRESENTATION

24 year-old high school graduate, working as sanitation worker was brought to the ER unconscious. He was found unconscious by his relatives. He had no psychiatric or chronic disease history. According to the history which was taken from his relatives, his intelligence level was normal and he was reported to be smoking and no habit of drinking. He was reported to have been occasionally using methamphetamine for the last 3 months. In his physical examinations injection marks were observed on

both of his arms. He had no traumatic findings. He was considered to have injected methamphetamine. The Glasgow Coma Scale was 5 in the initial examination. No abnormal findings were detected in the laboratory tests (full blood count test, serum biochemical profile, prothrombin time, activated partial prothrombin time). In the neurological examination; he was unconscious, his pupils were constricted and his right side was hemiplegic. The initial computed tomography (CT) of the head revealed diffuse hemorrhage in both ventricles and intraparenchymal hematoma accompanied by vasogenic edema in the left frontal lobe (Figure 1-2). In the drug screening tests (the panel included; benzodiazepines, cocaine, methamphetamine, barbiturates, methadone, cannabis, tricyclic antidepressants, opioids, buprenorphine, ecstasy (MDMA), and bonsai) of the urine and the blood samples methamphetamine was positive and ethanol was negative. The patient was referred to a tertiary hospital for cerebral angiography application. No arteriovenous malformations, aneurysms or dilatations were detected in the cerebral angiography (Figure 3). An external drainage catheter was applied and the patient was internalized in the neurosurgical intensive care unit. On the 2nd day of the hospitalization, sudden cardiac arrest occurred and the patient died.



Figure 1: Cranial tomography



Figure 2: Cranial tomography



Figure 3: Cerebral angiography imaging

DISCUSSION

Psychostimulant drugs increase the risk of stroke (6). Cocaine abuse significantly increases the risk of hemorrhagic stroke (6). The pharmacological and the physiological effects of the methamphetamine are similar to that of the cocaine. In addition to these similar effects, methamphetamine's elimination half-life is longer than that of the latter (7). Therefore, methamphetamine causes systemic hypertension for a longer period than cocaine (7). As well as the increased risk of stroke, methamphetamine abuse is closely related to the development of the ischemic heart disease and the atherosclerotic coronary heart disease (7). Methamphetamine abuse has been reported to be associated with aorta dissection (7).

Sympathomimetic effects of methamphetamine include increase in heart rate and blood pressure, increase in alertness, decrease in fatigue and appetite (8). The vascular effects of the methamphetamine can result in stroke and myocardial infarction. The negative side effects of the methamphetamine abuse (which include severe disabilities and death) are more frequent in patients who are younger than 45 years (9). 1 out of every 4 methamphetamine induced stroke show full recovery and one third of all the methamphetamine-induced hemorrhagic strokes are lethal (10). Concomitant smoking, alcohol use, hyperlipidemia, and other related lifestyle factors are considered to play a role in the methamphetamine-induced stroke risk (11).

Methamphetamine-induced stroke may be either ischemic or hemorrhagic and its pathogenesis is unknown (12). Huang et al. reported hemorrhagic stroke in methamphetamine abusers. The widely accepted possible mechanism of methamphetamine-related ischemic and hemorrhagic stroke is vasculitis (12). Methamphetamine has also been reported to damage the vascular wall integrity by fibrinoid necrosis of the

tunica intima and media, therefore causing vessel rupture (14). Neurological symptoms were reported to relieve with steroid and cyclophosphamide treatments (15). In various animal studies lethal dose of amphetamines were reported to cause subarachnoid, diffuse intracerebral and brain stem hemorrhages and significant dilatations in the meningeal, cerebral, cerebellar, and choroid vessels (11). Although brain stem hemorrhages are inducible in animal studies, its prevalence is relatively rare in humans.

Hemorrhagic strokes are either subarachnoid or intracerebral (16). Subarachnoid hemorrhages in young adults frequently occur as a result of an underlying cerebral aneurysm or arteriovenous malformation (AVM) (17). Non-traumatic intracerebral hemorrhage is usually related to hypertension, but also results from AVM, ruptured saccular aneurysm or sympathomimetic drug use in young adults (18). In young adults even in the absence of an underlying cerebrovascular disease, intracranial hemorrhage could occur as a result of the methamphetamine abuse (16). Our patient had no history of any chronic diseases including cerebrovascular disease, yet intracerebral hemorrhage was present.

The initial symptoms of methamphetamine-induced stroke may present as headache, nausea, vomiting, and confusion (10). Symptoms usually occur a few minutes after the consumption (10). Our case was found unconscious and brought to the ER by his relatives.

This present case report aimed to draw attention to the acute lethal outcomes of the methamphetamine use disorder. In young stroke cases, clinicians should consider methamphetamine use disorder as a possible etiology during differential diagnosis and should perform appropriate drug screening tests.

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