European Neurology

Eur Neurol 2000;43:181–182

Combined Amphetamine and Cocaine Abuse Caused Mesencephalic Ischemia in a 16-Year-Old Boy – Due to Vasospasm?

M. Strupp, G.F. Hamann, T. Brandt

Department of Neurology, University of Munich, Klinikum Grosshadern, Munich, Germany

Both the relationship between cocaine or amphetamine abuse and stroke as well as the pathomechanism of cocaine/amphetamine-induced stroke are still unclear. Whereas a recent study showed no association between cocaine abuse and ischemic stroke in young patients [1], others have described cocaine- or amphetamine-related strokes [2–5]. Several pathomechanisms have been discussed in the literature: cocaine-associated cerebral vasculitis [6], cocaine-associated cardiomyopathy leading to embolic stroke [2, 7, 8] or vasospasm [3]. We report on a young patient who used both drugs and subsequently suffered an ischemic lesion, which was most likely caused by vasospasm due to the combined abuse of both drugs.

Case Report

A 16-year-old, previously healthy schoolboy was admitted to our emergency unit complaining of unsteadiness and double vision, which had begun 3 days prior to admission. The patient had no risk factors for atherosclerosis (normal blood pressure, no smoking, no diabetes mellitus), no history of migraine and no family history of stroke. He reported that his symptoms started 5 min after intranasal inhalation of a small amount of 'amphetamine' (see below). He had a 1-year history of regular marihuana abuse. Since the patient still had some of the drug, we were able to analyze it by ligand assay and high-pressure liquid chromatography: it contained amphetamine *and* cocaine, i.e. the amphetamine had been 'cut' with cocaine.

The neurological examination on admission revealed a left-sided internuclear ophthalmoplegia. Further, the patient had incomplete fascicular paresis of the left oculomotor nerve (with moderate ptosis and weakness of the inferior oblique muscle) and saccadic vertical smooth pursuit. His blood pressure was normal. He steadily improved and became symptom free 3 weeks after symptom onset. After discharge he did not use any psychostimulants (follow-up time 6 months).

Cranial MRI was performed on admission. The T_2-weighted images showed a left-sided hyperintense lesion near the midline of the mesencephalon (fig. 1). Follow-up MRI 12 days after symptom onset revealed that the lesion had become much smaller. There were no abnormalities in the T_1-weighted and intra-/extracranial MR angiography images, no contrast enhancement and no evidence of bleeding. Extra- and transcranial Doppler/duplex sonography, transesophageal echocardiography, electrocardiography, chest roentgenography, 24-hour ECG Holter monitoring, lumbar puncture, visually and somatosensory-evoked potentials and the results of laboratory examinations [including blood sedimentation rate, antinuclear and anticytoplasmatic antibodies, coagulation tests (AT-III, protein C and S, factor V Leiden)] were normal, i.e. there was no evidence of other causes of ischemic stroke such as cocaine/amphetamine-induced vasculitis or dissection.

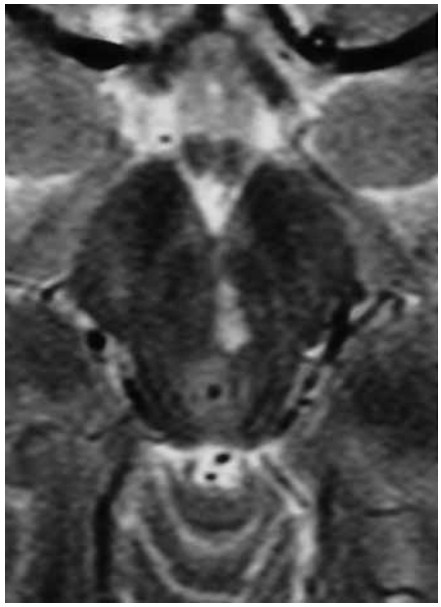

Fig. 1. A left-sided hyperintense lesion near the midline of the mesencephalon close to the oculomotor nucleus was found on admission on T_2-weighted axial images. Follow-up MRI 12 days after symptom onset demonstrated that the lesion had become much smaller and that there was contrast enhancement (not shown).

KARGER

Fax + 41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

Accessible online at:
www.karger.com/journals/ene

Discussion

This case indicates that (1) the ischemic lesion was most likely caused by the acute and combined action of cocaine and amphetamine (this is strongly supported by the close temporal relationship between intake of the drug and onset of the symptoms) and (2) vasospasm due to the synergistic effects of both drugs is the most probable pathomechanism in this case, since we found no evidence of other causes of cocaine/amphetamine-induced ischemia (see above).

While we are aware that in our case vasospasm as the pathomechanism was not proved by angiography, an angiographic study in rabbits clearly showed that cocaine or amphetamine alone caused little or no basilar artery spasm even at high doses, but coadministration of both produced definite basilar artery vasospasm, caused by a synergistic vasoconstrictive effect [9]. This synergism can be explained pharmacologically: amphetamine causes the release of epinephrine and norepinephrine, while cocaine prevents the reuptake of both, thereby sensitizing the arterioles to sympathomimetic neurotransmitters. Thus, the combination of these drugs may be harmful, even in small amounts.

Acknowledgements

We thank Ms. J. Benson for copy-editing the text.

References

1 Qureshi AI, Akbar MS, Czander E, Safdar K, Janssen RS, Frankel MR: Crack cocaine use and stroke in young patients. Neurology 1997;48:341–345.
2 Levine SR, Welch KM: Cocaine and stroke. Stroke 1988;19:779–783.
3 Martinez N, Diez Tejedor E, Frank A: Vasospasm/thrombus in cerebral ischemia related to cocaine abuse. Stroke 1996;27:147–148.
4 Petty GW, Brust JC, Tatemichi TK, Barr ML: Embolic stroke after smoking 'crack' cocaine. Stroke 1990;21:1632–1635.
5 Lambrecht GL, Malbrain ML, Chew SL, Baeck E, Verbraeken H: Intranasal caffeine and amphetamine causing stroke. Acta Neurol Belg 1993;93:146–149.
6 Fredericks RK, Lefkowitz DS, Challa VR, Troost BT: Cerebral vasculitis associated with cocaine abuse. Stroke 1991;22:1437–1439.
7 Sauer CM: Recurrent embolic stroke and cocaine-related cardiomyopathy. Stroke 1991;22:1203–1205.
8 Kaufman MJ, Levin JM, Ross MH, Lange N, Rose SL, Kukes TJ, Mendelson JH, Lukas SE, Cohen BM, Renshaw PF: Cocaine-induced cerebral vasoconstriction detected in humans with magnetic resonance angiography. JAMA 1998;279:376–380.
9 Wang AM, Suojanen JN, Colucci VM, Rumbaugh CL, Hollenberg NK: Cocaine- and methamphetamine-induced acute cerebral vasospasm: An angiographic study in rabbits. AJNR Am J Neuroradiol 1990;11:1141–1146.

Priv.-Doz. Dr. M. Strupp, MD, Department of Neurology
University of Munich, Klinikum Grosshadern, Marchioninistrasse 15
D–81366 Munich (Germany)
Tel. +49 89 7095 2571, Fax +49 89 7095 5584
E-Mail mstrupp@nefo.med.uni-muenchen.de