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The addicted brain: Imaging neurological complications of recreational drug abuse☆

A. Montoya-Filardi*, M. Mazón

Área Clínica de Imagen Médica, Hospital Universitario y Politécnico La Fe, Valencia, Spain

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Abstract Recreational drug abuse represents a serious public health problem. Neuroimaging traditionally played a secondary role in this scenario, where it was limited to detecting acute vascular events. However, thanks to advances in knowledge about disease and in morphological and functional imaging techniques, radiologists have now become very important in the diagnosis of acute and chronic neurological complications of recreational drug abuse.

The main complications are neurovascular disease, infection, toxicometabolic disorders, and brain atrophy. The nonspecific symptoms and denial of abuse make the radiologist's involvement fundamental in the management of these patients. Neuroimaging makes it possible to detect early changes and to suggest an etiological diagnosis in cases with specific patterns of involvement.

We aim to describe the pattern of abuse and the pathophysiological mechanisms of the drugs with the greatest neurological repercussions as well as to illustrate the depiction of the acute and chronic cerebral complications on conventional and functional imaging techniques.

PALABRAS CLAVE
Drogas de abuso;
Mecanismo bioquímico;
Resonancia magnética;
Imagen funcional;
Infarto;

El cerebro adicto: imagen de las complicaciones neurológicas por el consumo de drogas

Resumen Las drogas constituyen un gran problema sociosanitario. Tradicionalmente, la neuroimagen ha tenido un papel secundario limitado a la detección de eventos vasculares agudos. En la actualidad, el radiólogo ha adquirido gran relevancia en el diagnóstico de las complicaciones neurológicas agudas y crónicas, debido al avance en el conocimiento de la enfermedad y al desarrollo de las técnicas de imagen morfológicas y funcionales. Las principales complicaciones son la patología neurovascular, la infección, los trastornos tóxico-metabólicos y

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* Corresponding author.
E-mail address: montoyafilardi@gmail.com (A. Montoya-Filardi).

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la atrofia cerebral. La sintomatología inespecífica y la negación del consumo hacen que la implicación del radiólogo pueda resultar fundamental en la atención de estos pacientes. La neuroimagen permite detectar alteraciones precoces y plantear el diagnóstico etiológico ante patrones de afectación específicos. Nuestro objetivo es describir el patrón de consumo y el mecanismo fisiopatológico de las drogas con mayor repercusión neurológica, así como ilustrar las complicaciones cerebrales agudas y crónicas mediante técnicas de imagen convencional y funcional.

Introduction

Human kind has always wanted to experience new sensations, to reach different states of consciousness and, on occasion, to mask reality. The substances used with this purpose have targeted, whether intentionally or not, the central nervous system (CNS). Consumption patterns have changed and conditions of use have improved, but they all have deleterious effects that we can see and quantify.

Each substance has multiple forms of presentation which are modified all the time, the absorption pathways of the body are limited and they cause complications that overlap one another. Neuro-imaging findings frequently pose the possibility of consumption, and even in view of characteristic patterns, it would be possible to suggest the specific type of drug. The role of the radiologist, therefore, is ever more relevant both finding urgent conditions and visualizing chronic effects.

Our goal is to describe the consumption pattern and the physiopathological mechanisms of the drugs that have the most neurological repercussion (cocaine, amphetamines, heroin, alcohol, cannabis and toluene), as well as to illustrate the acute and chronic cerebral complications through conventional and functional image modalities (positron emission tomography [PET], single photon emission computed tomography [SPECT] and perfusion by magnetic resonance [MRI]).

Cocaine

Cocaine is the main alkaloid obtained from the leaves of a shrub belonging to the family *Erithroxylon coca*, present in the Western regions of South America. There are two forms of presentation: hydrochloride (with the appearance of a fine powder) and alkaloid (also known as *crack*).[1] The most popular route of administration is intranasal, which reaches concentrations in the CNS in 3–5 min. The main action of cocaine is summarized in the sympathomimetic effect due to the blockage of catecholamine reuptake.[1–4] It also blocks the reuptake of serotonin and dopamine transmitters, increasing their extracellular concentrations, especially in the *accumbens* nucleus.[2,5,6]

Addiction caused by cocaine is due to its rapid action mechanism, since its effect is almost immediate after administration.[1] A feeling of euphoria or high is experienced after its consumption mediated mainly by the occupation of dopamine receptors called DA-D2.[1,3–5]

The main cerebral complications derived from cocaine abuse are vascular ones, especially subarachnoid hemorrhages and intraparenchymatous hemorrhages; overall hemorrhagic events are twice as frequent as ischemic infarctions.[1,7,8] Nevertheless, the form of the drug and the route of absorption influence on the type of adverse event. When cocaine is smoked in the form of crack the incidence of hemorrhagic events is greater, while there are no significant differences between ischemic and hemorrhagic events when the drug is sniffed.[9,10]

The physiopathological mechanisms involved in the production of ischemic cerebrovascular accidents are multiple and synergic, but vasoconstriction or vasospasm stand out among them[2,11] (Table 1). This phenomenon can cause endothelial damage consisting of disruption of the tunica media and arteriolar fibrosis, among other alterations.[12] However, the significant vasculitis component stands out, confirmed by the existence of focal stenoses of the vascular lumen and enhancement of vessel walls in angiographic studies.[5,13]

Ischemic infarctions commonly affect the territories of mid and posterior cerebral arteries (Fig. 1), and the border territories, the internal capsule and the hippocampus, without a specific disorder pattern.[1,5,9] Mesencephalic infarction has been associated with the simultaneous use of cocaine and amphetamines.[14] Since it does not show a characteristic distribution, concomitant findings are specially relevant, such as perforation of the nasal septum, accelerated atherosclerosis in young people without cardiovascular risk factors (Fig. 2) and generalized vasospasm.

Table 1 Mechanisms implied in the production of cocaine-induced ischemic cerebrovascular accidents.

Ischemic mechanism	Biochemical phenomenon
Vasoconstriction	Calcium channel blockade[1,4] Increase of serotonin levels[2,5]
Prothrombotic phenomena	Increase of thromboxane levels and platelet aggregation[5,9,10]
Procoagulant diathesis	Reduction of antithrombin III and protein C[7,8]
Atherosclerosis	Nitric oxide inhibition in the endothelium[11]
Vasculitis	Impurity-activated paracrine and immune mechanisms[5,9,13]