Cocaine

Cocaine, also known as coke, is a strong stimulant mostly used as a recreational drug. It is commonly snorted, inhaled as smoke, or dissolved and injected into a vein. Mental effects may include loss of contact with reality, an intense feeling of happiness, or agitation.

Cocaine is extracted from Erythroxylon coca leaves (and other Erythroxylon species) and is thus unrelated to opioids. It blocks the re-uptake of norepinephrine by presynaptic adrenergic nerve terminals. It is available in 2 forms: cocaine hydrochloride (heat-labile and water-soluble, it is usually taken PO, IV, or by nasal insufflation) and the highly purified cocaine alkaloid (free base or crack cocaine, which is heat stable but insoluble in water and is usually smoked). Peak toxicity occurs 60–90minutes after ingestion (except for “body packers”), 30–60 minutes after snorting, and minutes after IV injection or smoking (freebase or crack) [1)].

Acute pharmacologic effects of cocaine

Effects on body systems outside the nervous system include tachycardia, acute myocardial infarction, arrhythmias, rupture of ascending aorta (aortic dissection), abruptio placenta, hyperthermia, intestinal ischemia, and sudden death.

Acute pharmacologic effects pertinent to the nervous system include:

1. mental status: initial CNS stimulation that first manifests as a sense of well-being and euphoria. Sometimes dysphoric agitation results, occasionally with delirium. Stimulation is followed by depression. Paranoia and toxic psychosis may occur with overdosage or chronic use. Addiction may occur

2. pupillary dilatation (mydriasis)

3. hypertension: from adrenergic stimulation.

Non-pharmacologic effects related to the nervous system

1. pituitary degeneration: from chronic intranasal use

Though cocaine intranasal inhalation always determines nasal mucosal damages, extensive septum perforations, and midline destructions-known as cocaine-induced midline destructive lesions (CIMDL)-affect only a limited fraction of patients. CIMDL is viewed as a cocaine-associated autoimmune phenomenon in which the presence of atypical anti-neutrophil cytoplasmic antibody (ANCA) promotes and/or defines the disease phenotype. A 51-year-old man presented with an intracranial tumor-like lesion by its space-occupying effect. CT also revealed the destruction of the nasal septum and skull base. A diagnosis of CIMDL was made in light of the patient's history as well as findings of the physical and endoscopic examinations, imaging studies, and laboratory testing. There was no evidence of other pathologies. Histopathological results from cerebral biopsy led us to consider the intracranial

pathology as an extension of the CIMDL. CIMDL is the result of a necrotizing inflammatory tissue response triggered by cocaine abuse in a subset of predisposed patients. The reported case is the first CIMDL consistent with brain extension mimicking a tumor-like lesion. While the presence of atypical ANCA seems to promote and/or define the disease phenotype, the specific role of these and other circulating autoantibodies needs further investigation [2].

2. cerebral vasculitis: less common than with amphetamines

3. seizures: possibly related to the local anesthetic properties of cocaine

4. stroke [3].

a) intracerebral hemorrhage

b) subarachnoid hemorrhage [4], [5]: possibly as a result of HTN in the presence of aneurysms or AVMs, however, sometimes no lesion is demonstrated on angiography [6]. May possibly be due to cerebral vasculitis

c) ischemic stroke [7] : may result from vasoconstriction

d) thrombotic stroke [8]

e) TIA [9]

5. anterior spinal artery syndrome [10]

6. effects of maternal cocaine use on the fetal nervous system include [11]: microcephaly, disorders of neuronal migration, neuronal differentiation, and myelination, cerebral infarction, subarachnoid and intracerebral hemorrhage, and sudden infant death syndrome (SIDS) in the postnatal period

Treatment of toxicity

Most cocaine toxicity is too short-lived to be treated. Anxiety, agitation or seizures may be treated with IV benzodiazepines, e.g. lorazepam. Refractory HTN may be treated with nicardipine or phentolamine (Regitine®). IV lidocaine used to treat cardiac arrhythmias may cause seizures [12]

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