Case Reports

Cocaine-induced central retinal artery occlusion

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Cocaine abuse in North America has reached epidemic proportions in the last few years.[1,2] There has also been an increase in the number of reports of various medical complications, particularly vascular.[3] These complications most commonly, but not always, develop in people who use large doses of the drug intravenously.

In this report we describe occlusion of the central retinal artery associated with cocaine use. As far as we know this complication has never previously been reported.

Case report

A 38-year-old woman who was a multiple-drug abuser was admitted to hospital after taking an overdose of phenytoin and diazepam. She had a long and varied history of drug abuse. For 6 months before admission she had been taking the barbiturate-containing analgesic Fiorinal (acetylsalicylic acid–caffeine–butalbital) and diazepam; she had also been injecting large doses of cocaine (4 to 5 g/d) intravenously. Immediately after her recovery from the drug overdose she reported loss of vision in her left eye. She had no history of visual impairment and had seen well the day before admission. She gave conflicting reports about her last cocaine injection, but there was reason to believe that her cocaine use had been uninterrupted to the time of admission.

Findings at physicial examination were normal, and there was no obvious source of emboli. The visual acuity was 6/6 in the right eye and only light perception in the left eye. The left pupil was nonreactive to direct light but did react consensually. Indirect ophthalmoscopy of the left eye revealed slightly attenuated retinal arteries, and the retina was diffusely edematous except for the fovea, which showed a cherry-red spot. One week later the macular edema persisted. Intravenous fluorescein angiography showed a patent arterial system. Her visual acuity improved to 6/36 in the left eye after 3 weeks.

Occlusion of the left central retinal artery was diagnosed. We could not detect any of the known predisposing or precipitating causes for this disorder. There was no pharmacologic or clinical reason to implicate the patient's use of phenytoin or diazepam. We speculated that the cocaine was responsible, perhaps through vasospasm rather than thromboembolic occlusion.

Comments

Cocaine inhibits the uptake of norepinephrine by adrenergic nerve endings and can cause intense vasoconstriction of small vessels.[4] An increased number of reports have described serious ischemic complications related to cocaine use; these include thrombotic and nonthrombotic myocardial infarction,[5,6] stroke,[7] abruptio placentae[8] and intestinal ischemia.[9] It has been postulated that people with congenital or acquired deficiency of pseudocholinesterase, the metabolizing enzyme of cocaine, are prone to serious toxic effects of cocaine, including ischemia.[3]

The evidence for the causative role of cocaine in most of the reports, as well as ours, was strong but only circumstantial. None the less, we suggest that spasm of the central retinal artery and the

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attendant visual impairment should be added to the list of potential ischemic complications of cocaine use.

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References

1. Nicholi AM: The nontherapeutic use of psychoactive drugs: a modern epidemic. *N Engl J Med* 1983; 308: 925-933
2. Pollin W: The danger of cocaine [E]. *JAMA* 1985; 254: 98
3. Cregler LL, Mark H: Medical complications of cocaine abuse. *N Engl J Med* 1986; 315: 1495-1499
4. Van Dyke C, Barash PG, Jatlow P et al: Cocaine: plasma concentrations after intranasal application in man. *Science* 1976; 191: 859-861
5. Isner JM, Estes NAM, Thompson PD et al: Acute cardiac events temporally related to cocaine abuse. *N Engl J Med* 1986; 315: 1438-1443
6. Rollingher IM, Belzberg AS, Macdonald IL: Cocaine-induced myocardial infarction. *Can Med Assoc J* 1986; 135: 45-46
7. Caplan LR, Hier DB, Feldman RS: Current concepts of cerebrovascular disease: stroke and drug abuse. *Stroke* 1982; 13: 869-872
8. Chasnoff IJ, Burns WJ, Schnoll SH et al: Cocaine use in pregnancy. *N Engl J Med* 1985; 313: 666-669
9. Nalbandian H, Sheth N, Dietrich R et al: Intestinal ischemia caused by cocaine ingestion: report of two cases. *Surgery* 1985; 97: 374-376

Subacute lead poisoning from retained lead shot

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A few clinical studies have shown that inorganic lead can be absorbed through synovial membranes, although poisoning from retained lead is rare.[1] Psychometric examination can reveal brain dysfunction when blood lead levels exceed 2.41 μmol/L for long periods, and chronic encephalopathy has been described.[2,3]

Case report

A 51-year-old ex-policeman had been under the care of a psychiatrist for 10 years after being extensively wounded in the right arm and chest in 1972 by a shotgun blast. More than 400 pellets remained in his body (Figs. 1 and 2). The psychiatrist was concerned about a steady deterioration in mental function, but the patient's condition was not consistent with endogenous mental illness.

After the injury the patient had continued to work and had held a responsible job in correctional services, but in 1982 he had to resign because of increasing fatigue, impaired intellectual function, irritability, mood swings, uncharacteristic outbursts of temper, poor concentration and insomnia. Between 1983 and 1985 three estimations of the blood lead concentration had shown fluctuating levels, from 0.83 to 3.10 μmol/L. No tests had been made before 1983. During April 1986 seven randomly conducted tests showed a mean blood lead level of 3.45 μmol/L. One month later the mean level from four weekly assays was 2.90 μmol/L; urine collected for 24 hours showed markedly elevated levels of coproporphyrin (1472 [normally 0 to 380] nmol), uroporphyrin (95 [normally 0 to 35] nmol) and porphobilinogen (25.9 [normally 2 to 15] μmol). The hemogram and blood smear appeared normal, and the blood urea nitrogen and serum creatinine levels were within normal limits.

Psychometric assessment involved detailed interviews with the patient, his family and his former colleagues. Intelligence, memory, personality and depression tests revealed a man of normal intelligence (intelligence quotient [IQ] 104) but with a clinically significant difference between his verbal IQ (111) and his performance IQ (96). He had difficulty sustaining effort and concentration, and his functions that relied on current perfor-

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