

COCAINE ASSOCIATED PRIAPISM

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ABSTRACT

Purpose: Cocaine abuse is an ongoing epidemic in the United States. Priapism associated with cocaine use has been reported only twice in the urological literature. To our knowledge we report the first series of priapism associated with cocaine use and the first case associated with the use of this drug in its solid form, known as crack.

Materials and Methods: We retrospectively reviewed the presentation of 3 patients to our emergency department within the last year. Each patient presented with priapism and no identifiable predisposition other than the use of cocaine within 24 hours, as evident on positive urine toxicology.

Results: Each patient delayed seeking treatment, which added to the complexity of therapy. Intracavernosal aspiration and irrigations failed in all 3 cases. Cavernous spongiosal shunting failed in the first 2 cases. Cases 2 and 3 were complicated by the high flow variant of priapism. Case 1 ultimately required partial penectomy for infected, gangrenous, distal penile tissue.

Conclusions: It appears that cocaine can be a cause of refractory priapism and treatment can be challenging. We suggest that urine toxicology screening be considered in such cases. The identification of underlying cocaine abuse is important in preventing priapism recurrence in these patients.

KEY WORDS: cocaine, priapism, penectomy, norepinephrine

Cocaine abuse, particularly in its solid form known as crack, is an ongoing epidemic in the United States. Priapism associated with cocaine use has been reported only twice in the urological literature.^{1,2} To our knowledge we report the first series of priapism associated with cocaine abuse and the first case associated with the use of crack.

CASE HISTORIES

Case 1. R. P., a 49-year-old black man, was a daily crack cocaine abuser who presented to our emergency department on July 8, 1997 with a 96-hour history of priapism following cocaine use. He admitted to at least 5 similar episodes within the previous year, all of which had resolved spontaneously. Medical history was otherwise unremarkable, including no history of sickle cell anemia. Physical examination revealed a disheveled man in obvious distress, with a blood pressure of 195/120. The corpora were fully erect, edematous and tender. Laboratory studies were within normal limits. Manual cavernosal irrigation with normal and heparinized saline yielded dark "crack case" blood. Detumescence did not occur after 3 separate injections of 250 mcg. phenylephrine. An El-Ghorab shunt procedure was then performed.

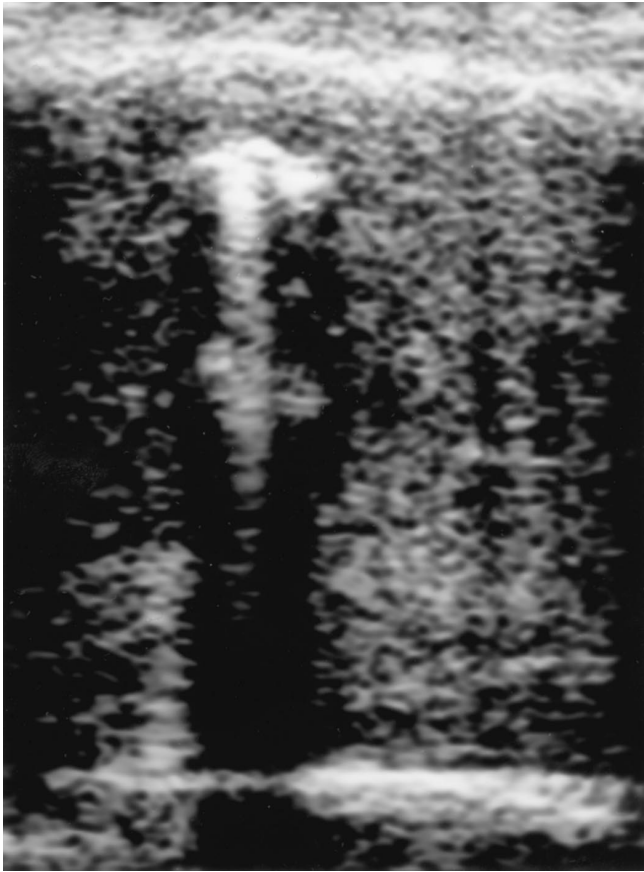
On hospital day 1 tumescence was again noted and duplex ultrasound showed no penile blood flow. A second attempt at aspiration and irrigation was briefly successful but on day 3 priapism persisted and a second El-Ghorab shunt procedure was done, followed by incomplete detumescence. On hospital day 7 the patient was febrile with penile erythema and ultrasound revealed bilateral cavernosal air (see figure). Broad-spectrum antibiotics, including anaerobic coverage, were started. He continued to be intermittently febrile during the next few days, and continuous purulent drainage from the shunt incision was noted. Ultrasound again showed no flow in the penis. Finally débridement, which resulted in partial penectomy, was done. Subsequently defervescence occurred and the patient was discharged from the hospital. No followup was possible, as the patient had an ischemic

stroke due to continued cocaine abuse shortly after hospital discharge, and he is presently ventilator dependent.

Case 2. A. A., a 38-year-old black man, presented to our emergency department on June 26, 1998 with a 36-hour history of a sustained, painful erection following intercourse. He denied any previous episodes, had no remarkable medical history (including no sickle cell anemia and trait) and was taking no medications. Initially he denied cocaine abuse but the urine toxicology screen was strongly positive for cocaine. Physical examination revealed fully engorged and tender corpora. Laboratory studies were otherwise within normal limits. Aspiration and dilute irrigation with 250 mcg. phenylephrine yielded dark, noncoagulated blood, suggestive of a veno-occlusive etiology. Then 500 mcg. phenylephrine were injected 3 times 7 to 10 minutes apart, which resulted in incomplete detumescence. An El-Ghorab shunt procedure was performed.

On hospital day 3 a painful erection was again noted. Penile duplex ultrasound confirmed a low flow state. A second El-Ghorab shunt procedure was performed. On hospital day 6 priapism recurred and penile duplex ultrasound showed a high flow state. Angiographic embolization was performed successfully. No cavernous artery fistula was evident on angiography. Followup is pending.

Case 3. P. P., a 40-year-old white man, presented on September 12, 1998 with a 24-hour history of a sustained, painful erection. He admitted to several less severe episodes within the previous year, all of which had resolved spontaneously. He had no remarkable medical history. Initially he denied cocaine abuse but the urine toxicology screen was strongly positive for cocaine. Physical examination revealed bilateral engorged cavernous bodies and a flaccid glans. Laboratory studies were otherwise within normal limits. Aspiration and normal saline irrigation yielded a dark penile aspirate. Blood gas determination of the aspirate confirmed a venous composition (pH 7.08, carbon dioxide pressure 32, oxygen pressure 32, bicarbonate pressure 10.1). Complete detumescence was finally achieved after 2 instillations of dilute 250 mcg. phenylephrine irrigation.



Case 1. Ultrasound reveals gas in left corpora and characteristic "dirty" acoustic shadow with radiating bands away from gas pocket.¹³

Priapism recurred 8 hours later that day. Duplex ultrasound showed a high flow state with peak cavernosal artery velocities of 80 on the left side and 60 cm. per second on the right side. Angiographic embolization initially was successful. No obvious cavernous artery fistula was noted on angiography. A partial erection returned 8 hours later. The patient refused further intervention. The erection resolved during the subsequent 24 hours of observation. Outpatient followup is pending.

DISCUSSION

Cocaine is an alkaloid ester derived from the leaves of the coca shrub. Cocaine hydrochloride is the crystalline and crack is its solid form after heating with bicarbonate.³ Common routes of administration are intranasal, inhalation and intravenous. Cocaine abuse is a national epidemic. As of 1992, 8.4% of black men and 2.1% of white men reported the use of cocaine.⁴ There are many complications associated with cocaine abuse and they often result from the systemic sympathetic vasoconstriction elicited.⁵⁻⁸

The mechanisms of action of this drug are not entirely clear and continue to be the focus of pharmacological inquiry. It is accepted that cocaine can inhibit the re-uptake of norepinephrine by blocking transport in the presynaptic sympathetic neuron, which results in an abundance of norepinephrine in the synaptic cleft and the consequent propagation of sympathetic discharge.⁵ Although little is known about the possible effects of cocaine on the penile vascular bed, one can postulate that they are similar to those noted elsewhere. It is possible that acute sexual excitement during intoxication could result in an erection with an impaired ability to achieve detumescence, which could be the result of a depletion of presynaptic norepinephrine with time, preventing sinusoidal

contraction and the efflux of penile blood. Fiorelli et al postulated that the cocaine induced erection may be due to the accumulation of serotonin with consequent peripheral vasodilatation and sinusoidal pooling.¹ Cocaine has been shown to inhibit the re-uptake of serotonin⁹ and serotonin can cause vasodilatation.¹⁰

Whatever the mechanism, based on our cases a low flow state is evident on delayed clinical presentation. In addition, manual aspiration and irrigation failed in all 3 of our cases, and surgical shunting also failed in cases 1 and 2. "Conversion" to a high flow state was noted in cases 2 and 3, and this phenomenon has been documented previously.¹¹ Whether high flow represents shunting from an iatrogenic cavernosal artery injury¹² during needle irrigation or the natural history of veno-occlusive priapism is unclear. Also, it is unclear why patient 2 responded to angiographic embolization immediately, while patient 3 initially responded incompletely.

We cannot make firm conclusions about the effect of cocaine abuse on penile physiology based on our data, as our series only included the retrospective examination of 3 cases. In addition, patient followup has been difficult due to non-compliance. Nevertheless, it seems that cocaine abuse can be a cause of priapism and treatment is frustrating, resulting in partial penectomy in 1 patient. We propose that a urine toxicology study be obtained as the identification of underlying cocaine abuse is important in preventing priapism recurrence in these patients.

REFERENCES

1. Fiorelli, R. L., Manfrey, S. J., Belkoff, L. H. and Finkelstein, L. H.: Priapism associated with intranasal cocaine abuse. *J. Urol.*, **143**: 584, 1990.
2. Rodriguez-Blázquez, H. M., Cardona, P. E. and Rivera-Herrera, J. L.: Priapism associated with the use of topical cocaine. *J. Urol.*, **143**: 358, 1990.
3. Gessner, P. K.: Substance Abuse: Essentials of Pharmacology. Philadelphia: W. B. Saunders, pp. 518-519, 1995.
4. Braun, B. L., Murray, D., Hannan, P., Sidney, S. and Le, C.: Cocaine use and characteristics of young adult users from 1987 to 1992: the CARDIA study. *Amer. J. Pub. Health*, **86**: 1736, 1996.
5. Jacobsen, T. N., Grayburn, P. A., Snyder, R. W., 2nd, Hansen, J., Chavoshan, B., Landau, C., Lange, R. A., Hillis, L. D. and Victor, R. G.: Effects of intranasal cocaine on sympathetic nerve discharge in humans. *J. Clin. Invest.*, **99**: 628, 1997.
6. Konzen, J. P., Levine, S. R. and Garcia, J. H.: Vasospasm and thrombus formation as possible mechanisms of stroke related to alkaloidal cocaine. *Stroke*, **26**: 1114, 1995.
7. Pitts, W. R., Lange, R. A., Cigarroa, J. E. and Hillis, L. D.: Cocaine-induced myocardial ischemia and infarction: pathophysiology, recognition, and management. *Prog. Cardiovasc. Dis.*, **40**: 65, 1997.
8. Sudhakar, C. B., Al-Hakeem, M., MacArthur, J. D. and Sumpio, B. E.: Mesenteric ischemia secondary to cocaine abuse: case reports and literature review. *Amer. J. Gastroenterol.*, **92**: 1053, 1997.
9. Lakoski, J. M. and Cunningham, K. A.: The interaction of cocaine with central serotonergic neuronal systems: cellular electrophysiologic approaches. *Natl. Inst. Drug Abuse Res. Monogr.*, **88**: 78, 1988.
10. Lamping, K. G.: Response of native and stimulated collateral vessels to serotonin. *Amer. J. Physiol.*, part 2, **272**: H2409, 1997.
11. Seftel, A. D., Haas, C. A., Brown, S. L., Herbener, T. E., Sands, M. and Lipuma, J.: High flow priapism complicating veno-occlusive priapism: pathophysiology of recurrent idiopathic priapism? *J. Urol.*, **159**: 1300, 1998.
12. Witt, M. A., Goldstein, I., Saenz de Tejada, I., Greenfield, A. and Krane, R. J.: Traumatic laceration of intracavernosal arteries: the pathophysiology of nonischemic, high flow, arterial priapism. *J. Urol.*, **143**: 129, 1990.
13. Rosenfield, A. T., Rigsby, C. M., Burns, P. M. and Romero, R.: Ultrasonography of the urinary tract. In: *Clinical Urography: An Atlas and Textbook of Urological Imaging*. Edited by H. M. Pollack. Philadelphia: W. B. Saunders, pp. 319-386, 1990.