The PHA of granulocytes in our patient resolved after one year as her tuberculosis was treated. This confirms that our patient had the acquired form of PHA and establishes the association of tuberculosis, especially severe disease, with PHA of granulocytes. Although patients with acquired PHA should receive evaluation for underlying malignancies, especially leukemia, infectious etiologies, including tuberculosis, must be considered. Acquired PHA may provide an aid in the diagnosis of patients with tuberculosis, including those with unusual radiographic manifestations or progressive illness, and it appears to indicate an over-all worse prognosis.

REFERENCES


Asystole and Ventricular Fibrillation Associated with Cocaine Intoxication*

Amin A. Nanji, M.B., B.Ch.B., and J. Douglas Filipenko, M.D.

We discuss a patient with cocaine intoxication in whom the initial presentation was that of asystole and ventricular fibrillation. Cocaine has a direct toxic action on the heart and also sensitizes cardiac tissue to the action of catecholamines. Cocaine intoxication should be considered in any patient with unexplained cardiac arrest or ventricular arrhythmias.

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Asystole and Ventricular Fibrillation and Cocaine (Nanji, Filipenko)
Cocaine, an alkaloid of the plant *Erythroxylon coca*, has the peculiar quality of being both a local anesthetic and a sympathomimetic agent with powerful central nervous system effects. In small doses, a dose-related increase in blood pressure and heart rate is seen. Extremely high doses can have a direct toxic action on the heart. Benchmil et al. first reported the occurrence of accelerated ventricular rhythm as a consequence of cocaine abuse. We present a patient who took a large dose of cocaine. She initially had asystole which subsequently converted to ventricular fibrillation. We are aware of only one other sensational case of cardiac arrest occurring with cocaine overdose. The patient, an airline passenger, ingested six cocaine-filled condoms, one of which burst.

**CASE REPORT**

A 23-year-old woman was brought to the emergency room in a comatose state. She was found unconscious at home by a friend. An empty syringe and spoon containing cocaine were found next to her. The paramedical staff could not record her blood pressure and the ECG showed asystole. Sodium bicarbonate, epinephrine and atropine were administered. This produced ventricular fibrillation. On arrival in the emergency room, she was intubated. She was subsequently transferred to the ICU. Her heart rate was 180/min and blood pressure, 90/70 mm Hg. She was still comatose and responded only to painful stimuli. Verapamil (2.5 mg IV) was given; this reduced the heart rate to 100/min. The patient was mechanically ventilated and given intravenous saline solution and sodium bicarbonate. Her blood pressure eventually stabilized at 130/90 mm Hg.

Some relevant laboratory investigations at admission included: arterial blood pH, 6.71; Pco2, 22 mm Hg; lactate, 18 mmol/L; serum sodium, 146 mmol/L; potassium, 5.3 mmol/L; chloride, 102 mmol/L; total carbon dioxide, 3 mmol/L; urea nitrogen, 12 mg/dl; creatinine, 1.8 mg/dl. Enzymes were: creatine kinase, 175 IU/L; aspartate aminotransferase, 105 IU/L; lactic dehydrogenase, 281 IU/L; arterial blood Pao2 after institution of mechanical ventilation, 370 mm Hg. A drug screen for serum for salicylate, alcohol, barbiturates and acetaminophen was negative. A urine drug screen showed the presence of large amounts of cocaine and its metabolites.

About 24 hours after admission to the ICU, the patient's blood pressure rose rapidly to 220/100 mm Hg; this was managed with intravenous sodium nitroprusside. The patient's neurologic state, however, continued to deteriorate. The pupils responded sluggishly to light, corneal reflexes were absent, and papilledema was observed. A CT scan of the head was consistent with the presence of cerebral edema. By 48 hours after admission, the patient's pupils became dilated and fixed. She died immediately afterwards. Of note is the absence of hyperthermia throughout her hospital course. An autopsy was performed and showed the presence of cerebral edema.

**DISCUSSION**

With lethal doses of cocaine, the presenting picture is one of hyperthermia, status epilepticus ventricular arrhythmias and/or respiratory arrest. The reason for the occurrence of asystole and ventricular arrhythmias in the above patient was probably related to a) direct toxic effect of cocaine on the heart, and b) the permissive role of cocaine in accentuating the influence of epinephrine and norepinephrine on cardiac tissue.

While the oral lethal dose of cocaine is given as 1 gram, death occurs with as little as 20 mg given intravenously. We do not know how much cocaine with which our patient injected herself.

This case demonstrates an unusual feature of cocaine overdose and should alert physicians to consider cocaine intoxication in a patient with cardiac arrest and/or ventricular arrhythmias.

**REFERENCES**

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**Amiodarone-induced Hypersensitivity Pneumonitis* Evidence of an Immunological Cell-Mediated Mechanism**


Interstitial pneumonitis developed in a patient who had received a cumulative dose of 985 g of amiodarone in nine years. No other cause for pneumonitis was found. The following findings favor an immunologic mechanism of hypersensitivity due to amiodarone: positive skin and basophil degranulation tests with amiodarone; lymphocytosis and inverted ratio of helper suppressor T lymphocytes in bronchoalveolar lavage fluid; secretion of leukocyte inhibitory factor, as shown by the inhibition of migration of peripheral blood leukocytes; and positive lymphoblastic transformation in the presence of amiodarone.

Amiodarone, a derivative of benzo-furan, has been in wide use since 1967 as an antiarrhythmic drug, and its multiple side effects are well known. However, and unexpectedly, it is only since 1980 that several dozen cases of lung disease associated with use of this drug have been reported.

We present here a case of pneumonitis which developed after nine years of treatment with amiodarone. Several biologic findings favor an immunologic origin of the disease (hypersensitivity due to amiodarone).