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Cardiac Dysrhythmias Associated with Chemical Peeling with Phenol

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Chemical peeling of various skin lesions and wrinkles with phenol (carbolic acid) is a common practice in plastic surgery, otolaryngology, and dermatology. For small lesions, chemical peeling produces only mild discomfort and usually is performed with analgesia and sedation in an office. However, for larger lesions, regional or general anesthesia may be required to provide sufficient analgesia to facilitate an immobile field. Morbidity, including dysrhythmias,[1] laryngeal edema,[2] and toxic shock syndrome,[3] and mortality[4] can occur with phenol application to large lesions. We present the case of a 10-year-old boy who had a life-threatening dysrhythmia develop during phenol application to a giant hairy nevus.

REPORT OF A CASE

A healthy 36-kg, 10-year-old boy with a giant 12 × 17 cm hairy nevus, constituting 1.9% of his body surface area and covering his left scapula area and nape, was admitted for phenol exfoliation of the entire lesion under general anesthesia. Routine preoperative chest roentgenogram, hemoglobin determination, and medical examination were unremarkable. This procedure was to be the sixth on the lesion, including three previous chemical peels with phenol within the past 2 years. All three of these peels were performed with the patient under general anesthesia and were without incident.

Preinduction monitoring included blood pressure by sphygmomanometer, cardiac rhythm by continuous ECG, and breath sounds and heart tones by precordial stethoscope. The patient requested an inhalation induction. Anesthesia was induced by inhalation of nitrous oxide 60% and halothane up to 3%. Following peripheral venous cannulation, endotracheal intubation was accomplished easily without using muscle relaxants. Bilateral breath sounds were noted, and an esophageal stethoscope and nasal temperature probe were inserted. The patient was turned prone and bilateral breath sounds again verified. Anesthesia was maintained with a concentration of halothane less than 1% in 50% nitrous oxide. Ventilation was controlled. Intravenous fluids during the hour of phenol application totalled 200 ml of lactated Ringer's solution. Phenol application consisted of swabbing the phenol solution onto the nevus with cotton-tipped applicators. The solution consisted of 40% phenol and 0.8% croton oil in hexachlorophine soap and water. A total volume of less than 6 ml (2.4 g phenol) was used.

The patient was hemodynamically stable until 55 min into the phenol application, at which time multifocal and coupled premature ventricular complexes (PVCs) developed on ECG. Blood pressure remained stable, and manual chest compressions were not initiated. Halothane and nitrous oxide were discontinued. Two iv boluses of lidocaine 50 mg failed to resolve the dysrhythmias. Analysis of arterial blood gases revealed Pa_{O_2} 412 mmHg, Pa_{CO_2} 39 mmHg, *p*H 7.36, base excess −3 mEq/l, HCO_3 21 mEq/l, and Sa_{O_2} 98%. Plasma potassium (3.7 mEq/dl) and sodium (138 mEq/dl) were normal. The nasopharyngeal temperature did not increase.

Bretylium sulfate 250 mg was given by iv infusion following the failure of lidocaine to suppress the multifocal PVCs. This promptly resulted in a decrease in frequency of multifocal and coupled PVCs. The patient awakened and remained hemodynamically stable. The trachea was extubated upon return of spontaneous respirations. The dysrhythmia converted to sinus tachycardia following completion of the bretylium infusion. During the next 2 h he was observed closely and monitored in the recovery area, and he remained in sinus rhythm with stable vital signs. He was dismissed to an intensive care unit for cardiac monitoring and had an uneventful recovery, with discharge 36 h later.

DISCUSSION

We did not measure phenol blood levels and cannot state that these cardiac dysrhythmias definitely were caused by phenol toxicity. However, such dysrhythmias are the primary morbidity reported with chemical peeling with phenol. Truppman and Ellenby[1] reported dysrhythmias, including ventricular tachycardia and bigeminy, in 10 of 43 consecutive patients undergoing chemical face peels. They were unable to correlate these dysrhythmias with age or the presence of existing cardiac disease. There was a high degree of correlation with the size of the area to which phenol was applied and the duration of application. This correlation suggests cutaneous phenol absorption.

Skin absorption does occur; Litton[5] reported blood phenol levels of 6.8 μg/ml 1 h following facial application of phenol 1.5 g in a 50% solution, decreasing to 1 μg/ml at 4 h. Unfortunately, little is known about the pharmacokinetics or pharmacodynamics of phenol. Toxic blood levels and rate of cutaneous absorption are not delineated clearly. Fatal oral doses in adults have ranged from 1 to 15 g, with death within 24 h associated with intractable dysrhythmias and myocardial and respiratory depression.[6]

Phenol is used by anesthesiologists for neurolytic blocks in 5–10% solutions in glycerol without systemic sequelae. Solutions of 40–80% phenol are common for chemical peel. At these high concentrations, proteins of the superficial epidermis immediately are denatured,

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keratin is coagulated, and the underlying dermis becomes hyperemic.[4] This breakdown of the skin barrier and the increase in blood flow in the underlying dermis presumably facilitate systemic uptake of phenol. Soaps or propylene glycol often are added to phenol solutions to emulsify phenol and water and lower surface tensions. Croton oil is used as an epidermolytic agent to increase the cutaneous penetration of phenol. It is extremely toxic to gastrointestinal mucosa when taken orally,[7] but its systemic toxicity is unknown. The relative contribution of phenol and croton oil to dysrhythmia production during chemical peel is unclear. If dysrhythmias or signs of systemic toxicity occur during phenol application, therapy entails hemodynamic support, dysrhythmia suppression, and immediate removal of any solution remaining on the skin surface with soap and water.

In summary, we have presented this case to emphasize the apparent association of cutaneous application of phenol solutions with systemic toxicity, usually marked by dysrhythmias. Anesthesiologists involved in the care of patients undergoing such procedures should be aware of this association.

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