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Sudden Cardiac Arrest during Percutaneous Ultrasonic Nephrostolithotomy

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Ultrasonic percutaneous nephrostolithotomy is a relatively new procedure for the removal of renal and upper ureteral stones. Two groups have reported on the use of the procedure in a total of 32 patients.[1,2] There have been few major complications of this technique reported. The procedure uses ultrasonic energy delivered by a metal probe through a percutaneous track into the renal pelvis to fracture stones. The fragments then are flushed out with large quantities of irrigating fluid. We wish to report a cardiac arrest as a major complication of this new procedure.

REPORT OF A CASE

The patient was a healthy 41-year-old, 60-kg woman with a staghorn calculus in her right renal pelvis. She had previously had a percutaneous nephrostomy tube inserted under local anesthesia without difficulty. Laboratory values included a serum sodium of 138 mEq·l^{-1}, serum potassium of 4.5 mEq·l^{-1}, hematocrit of 39%, and total bilirubin of 0.1 mg/dl. Meperidine, 50 mg, promethazine, 25 mg, and atropine, 0.4 mg, were given im 1 h prior to surgery. Anesthesia was induced with 300 mg thiopental iv in divided doses and maintained with inspired concentrations of 60% N_2O and 0.7 to 1.0% halothane using controlled ventilation. Nondepolarizing neuromuscular blocking drugs were not used.

The case proceeded uneventfully for approximately 90 min. By this time, sterile water irrigation fluid had been in use for about 45 min and a large portion of the stone had been fractured and removed. The returning irrigating fluid had been clear. The vital signs had been stable with a systolic blood pressure of 90 mmHg, and a regular heart rate of 80 beats/min with a sinus rhythm. Blood then began returning with the irrigation fluid, and immediately a 30-s period of bradycardia unresponsive to atropine 0.8 mg iv occurred. This was followed by asystole. Closed chest cardiac massage was initiated immediately and ventilation was controlled with a $F_{I_{O_2}}$ of 1.0. Resuscitation was successful with the addition of epinephrine, 1.0 mg iv, sodium bicarbonate, 50 mEq iv, and calcium chloride, 500 mg iv, followed by electrical cardioversion. A systolic blood pressure of 120 mmHg was reestablished with a sinus tachycardia at a rate of 130 beats/min approximately 5 min after the arrest. Small amounts of frothy blood-tinged secretions were suctioned from the endotracheal tube after the arrest. A blood sample drawn during resuscitation showed a serum sodium of 121 mEq·l^{-1}, serum potassium of 4.5 mEq·l^{-1}, and a spun hematocrit of 28% (capillary tube). The serum was not examined for gross color changes. Surgery was terminated. The patient awoke in 1 h without signs of any cardiovascular or neurologic sequelae. Furosemide, 10 mg iv, was administered and the pulmonary edema resolved over the next few hours. Pertinent laboratory data 5 h postoperatively included a serum sodium of 126 mEq·l^{-1}, serum potassium of 4.4 mEq·l^{-1}, and hematocrit 26%. The following day serum sodium was 131 mEq·l^{-1}, serum potassium was 4.0 mEq·l^{-1}, total serum bilirubin 3.4 mg/dl, serum hemoglobin 600 mg/dl, and the hematocrit 32%. The patient was discharged on the third postoperative day with no evidence of impairment of renal, cardiac, or neurologic function.

DISCUSSION

There are several possible causes for the cardiac arrest. One is simple intravascular volume overload with acute heart failure. This would be unlikely to cause a sudden cardiac arrest without other premonitory signs of volume overload and so we suspect this did not occur. Another possibility would be acute hyponatremia from free water intake. Under general anesthesia this could present as a sudden cardiac arrest without earlier signs of hypervolemia. A third possibility is the sudden entrance of a bolus of hypotonic fluid with acute hemolysis and hyperkalemic cardiac arrest. This was not excluded by the normal serum potassium values 5–10 min following the arrest, since the

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Key words: Surgery: ultrasonic nephrostolithotomy. Fluid balance: water intoxication. Complications: arrest, cardiac.

total amount of potassium release would not need to be very large. The elevated postoperative bilirubin and hemoglobin levels indicate that appreciable hemolysis occurred and probably contributed to the clinical problems described in this case. A fourth possibility is air embolism, which can present as a sudden cardiac arrest. The patient was in the right lateral position with extreme flexion and the kidney rest was at the upper limit of its travel. Thus, there was at least a 10-cm gradient from the surgical site to the right atrium. Sudden opening of a large vessel conceivably could have allowed sufficient air to be entrained to cause cardiac arrest. However, the gradient in this case seemed small, and no murmurs were heard with the esophageal stethoscope. The appearance of blood in previously clear irrigation fluid suggests that there was the sudden opening of a vessel, presumably secondary to manipulation of the probe.

Alken[1] reported on a series of 17 patients in whom he had performed percutaneous stone removal using the ultrasonic lithotrite. Complications included three residual stones, retroperitoneal extravasation of fluid in one case, bleeding successfully managed conservatively in another case, and ureteral stenosis requiring operation in one case. Segura *et al.*[2] described 15 patients managed with this technique and reported minimal postoperative morbidity, with no serious sequelae. Anesthetic techniques in these reported series involved local, epidural, and general anesthesia. The operative technique has become a standard one and is widely believed to be associated with no serious morbidity.

Regardless of the exact cause of our patient's cardiac arrest, percutaneous ultrasonic nephrostolithotomy is clearly not without major risks including possible water intoxication, venous air embolism, and, as in our case, cardiac arrest. In this case, the sudden appearance of blood in previously clear irrigation fluid was the earliest sign of impending problems but was followed within seconds by a complete cardiac arrest. Since we believe the electrolyte disturbances from free water intake and hemolysis to be the major factors, we have modified our protocol and now use normal saline irrigation fluid for these procedures.

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Entrapment of a Mitral Valve Prosthesis with a Left Atrial Catheter

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The use of invasive monitoring devices have greatly enhanced our abilities to care for patients undergoing cardiac surgery. In particular, the use of a left atrial catheter provides an accurate method of quantitating filling pressures and appropriately adjusting left ventricular preload. We report an unusual situation in which a left atrial catheter resulted in temporary, life-threatening malfunction of a prosthetic valve disc.

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Report of a Case

A 39-year-old woman had a 4-year history of progressive dyspnea. There was no history of rheumatic heart disease or primary pulmonary disease. On physical examination, she had a murmur and an audible click consistent with a stenotic mitral valve.

During cardiac catheterization, her cardiac index was 2.29 $l \cdot m^2$. The pulmonary capillary wedge pressure was 19 mmHg and increased to 34 mmHg following exercise. The mitral valve area was 0.9 cm^2. Left ventricular function was normal with an ejection fraction of 0.6. The ECG was normal, and the chest roentgenogram showed left atrial prominence with some increase in pulmonary vascularity. Because of a diagnosis of mitral stenosis, a mitral valve replacement was scheduled.

Prior to induction of anesthesia, direct arterial pressure monitoring was established, and a flow-directed pulmonary artery catheter was inserted via the right internal jugular vein. Anesthesia was induced by iv narcotics. Vital signs remained very stable following induction of anesthesia and during cardiopulmonary bypass. The duration of cardiopulmonary bypass was approximately 2 h. The stenotic mitral