SELECTED REPORTS

Recurrent Pulmonary Embolus after Insertion of Intracaval Prosthesis*

Robert J. DiBenedetto, M.D. and William A. Miller, M.D.

A patient is described with recurrent pulmonary embolus following combined therapy of placement of a caval umbrella device for caval interruption and anticoagulation. The source of this embolus was felt to be from a thrombus located on the proximal portion of the umbrella. This is the first reported instance of this complication.

The vena cava umbrella device for prevention of pulmonary emboli has found wide acceptance since Mobin-Uddin et al1 first described it in 1967. Recurrent pulmonary emboli, after placement of the intracaval device, have been infrequent,2-5 occurring in less than 2 percent of insertions. This report concerns a patient who developed a pulmonary embolus after placement of the caval umbrella. A thrombus located on the proximal surface of the device was felt to be the source of embolization.

CASE REPORT

A 67-year-old white woman was hospitalized in January, 1972 for removal of an impacted gallstone in the remnant of her cystic duct (cholecystectomy had been performed 20 years previously). History revealed chronic intermittent edema of the lower left leg resulting from a previous episode of phlebitis. The postoperative period following the second operation was characterized by congestive heart failure and bronchopneumonia. On the day of discharge, the patient developed left upper quadrant abdominal pain of a pleuritic nature. This subsequently cleared only to recur three days later. She was re-hospitalized and found to have normal vital signs except for pulse rate of 120. Auscultation of the lungs revealed the presence of bilateral basilar rales. Moderate left upper quadrant abdominal tenderness was noted. Swelling of the left lower extremity was present, but there was no calf tenderness or Homan's sign. Chest x-ray examination and electrocardiogram were unremarkable. The hematocrit was 37 volumes percent, the WBC count 9,800 cells per cubic mm with a normal differential count. Arterial blood gases revealed Po2 58 mm Hg, PCO2 of 37.6 mm Hg, and pH 7.4. The LDH was 400 units and the SGOT, CPK and SGPT were normal. Lung scan revealed multiple bilateral areas of decreased perfusion felt to represent multiple pulmonary emboli (Fig 1). Subcutaneous heparin (75 mg) was given every six hours. The Lee White clotting time was maintained at two to three times the control value of seven minutes. Coumadin was instituted simultaneously in therapeutic doses and the prothrombin time was prolonged to twice the control value of 12 seconds.

Two days after admission, the patient developed right pleuritic chest pain associated with increasing rales over the right lower lung. It was felt she had sustained another embolus despite adequate anticoagulation. A vena cava umbrella was placed at the level of the fourth lumbar vertebrae. The heparin was withdrawn four days later, but coumadin was continued.

The patient did well until 14 days later when she developed confusion and dyspnea. Lung scan demonstrated adequate perfusion of the previously underperfused areas, but with a new extensive flow deficit to the entire right upper lobe (Fig 2). The LDH was 350 units. In an attempt to determine the source of this new embolus, a bilateral femoral iliac venogram was performed. This revealed nearly complete occlusion of the left iliac vein with extensive collateral development presumably secondary to the chronic phlebitis. There was a 2 cm in diameter thrombus located on the proximal surface of the umbrella (Fig 3). The vena cava was patent above the device and there was flow of contrast material through and around the device via collaterals. It was felt that this thrombus was the source of embolization. Intravenous heparin therapy (50 mg every four hours) was instituted. The patient gradually improved and her clinical findings cleared.

Seven days later, repeat venacavagram disclosed persistent patency of the cava with no change in the thrombus. Repeat lung scan showed return of normal perfusion to the right upper lobe. She was discharged 14 days after the recurrent embolus on 5 mg coumadin per day.

*From the Departments of Medicine and Radiology, Memorial Medical Center, Savannah, Georgia.

CHEST, VOL. 63, NO. 6, JUNE, 1973

FIGURE 1. Lung scan on admission showing areas of decreased perfusion in both upper lung fields and the right lower lobe.

FIGURE 2. Lung scan 14 days after insertion of intracaval umbrella demonstrating massive flow defect in right upper lobe.
sources of further embolization. The proximity of the renal vein usually prevents propagation. Since the vena cava remained patent in this case and the thrombus was demonstrated on the proximal surface of the umbrella, it was felt that this was the source of the embolus. Perhaps the low insertion at the level of the L4 vertebral body is responsible for propagation of clot in this instance. The recurrent embolus took place two weeks after placement of the device. This period of time is within the usual six to eight weeks required for endothelialization and fibrosis of the proximal thrombus. Although a medium sized collateral vessel was demonstrated in the region of the umbrella, this was not felt to be the route of the recurrent embolus. We believe that the diameter of this collateral vessel was not sufficient to allow passage of an embolus of this size unless numerous small emboli followed precisely the same pathway to the pulmonary vasculature of the right upper lobe. This seems to be a very remote possibility. An ovarian vein route for this embolus seems doubtful in view of the more obvious source. We believe that this report demonstrates that a thrombus located on the proximal portion of the caval umbrella in association with free flow of blood through the prosthesis may give rise to repeated pulmonary emboli.

Mobin-Uddin et al suggest continuation of heparin therapy for five to seven days after insertion of the prosthesis. This patient was maintained on heparin therapy for four days after placement. Coumadin, which had been instituted at the time of admission in full therapeutic doses, was continued following withdrawal of heparin. Perhaps prolonged administration of heparin would have prevented this complication.

This report clearly demonstrates the need for continued vigilance regarding recurrent pulmonary emboli during the initial several weeks following insertion of the vena cava device. An increased awareness of this possibility may reveal this sequence of events to be more frequent than formerly presumed. This case also reveals the importance of proper placement of the caval umbrella at the L3 level so that the renal veins will prevent propagation of thrombus from the proximal portion of the umbrella and also the need for continued heparin administration during the critical period following prosthesis insertion.

REFERENCES

5. Mobin-Uddin Vena Cava Umbrella Filter: For interruption
of the inferior vena cava to prevent pulmonary embolism. Edwards Laboratories, 103534 7/70 rev. 2/72 5M 8-1-223

Hyperkalemic Cardiac Arrhythmia Secondary to Spironolactone*

Chalat Pongpaew, M.D., Reoadee Na Songkhla, M.D., and Robert L. Kozam, M.D.

Although multiple adverse reactions can occur during the use of spironolactone, the most serious complication is related to its potassium-sparing effect which may induce dangerous and even fatal hyperkalemia. One patient is presented who developed severe hyperkalemia after spironolactone resulting in ventricular standstill and survival following treatment with sodium bicarbonate.

*From the Division of Cardiology, Methodist Hospital of Brooklyn, Brooklyn.
Reprint requests: Dr. Kozam, 215 Hudson, Tenafl, New Jersey 07670

Skin rash, gastrointestinal irritation, hirsutism, gynecomastia, and muscular weakness are adverse reactions related to the use of the potassium-sparing diuretic, spironolactone.1 The side effects are usually infrequent and mild. The most serious complication is related to its potassium-sparing effect which may induce dangerous and even fatal hyperkalemia.1 The following case report presents an occurrence of severe hyperkalemia after the excessive use of spironolactone, resulting in ventricular standstill, and survival after treatment with sodium bicarbonate.

CASE REPORT
A 69-year-old man was brought to the emergency room of Methodist Hospital of Brooklyn in a state of cardiovascular collapse, intensely cyanotic, without pain, and with a level of consciousness inconsistent with his clinical state. A brief physical examination revealed blood pressure 60 mm Hg by palpation, and an irregular heart rate of 20-25 beats per minute. Because of the fear of impending cardiac arrest, resuscitative measures were instituted. Following initial electrocardiogram (Fig 1A) which revealed the pattern of a dying heart with a ventricular rate of 13 beats per minute, atropine sulfate, 0.4 mg, was given intravenously and an isoproterenol drip was started. Almost simultaneously, sodium bicarbonate, 44.6 mEq was given intravenously to correct probable metabolic acidosis in the setting of clinical shock. A similar dose was repeated after a five minute period. After the second dose of bicarbonate, the electrocardiogram revealed bizarre QRS complexes occurring with such regularity as to suggest the presence of an AV junctional tachycardia.

Figure 1A. Emergency room electrocardiographic strip revealing the pattern of a dying heart with a ventricular rate of 13 beats/min. B. AV junctional rhythm approximately five minutes after 44.6 mEq of intravenous sodium bicarbonate. C. AV junctional tachycardia ten minutes later—note the decreased widening of the QRS complex.