Pulmonary Thromboembolism Associated With Radiofrequency Catheter Ablation

To the Editor:

An 8-year-old boy diagnosed as having Wolff-Parkinson-White syndrome, with no structural heart defects, was referred for radiofrequency catheter ablation. The procedure was performed with the patient under deep sedation with intravenous midazolam and intravenous fentanyl. Two quadripolar electrode 5 Fr catheters (Bard®) were inserted percutaneously via the femoral vein and positioned consecutively in the upper right atrium, His bundle and apex of the right ventricle. A decapolar electrode 5 Fr catheter (Cordis®) was inserted via the left brachial vein and positioned in the coronary sinus. Electrophysiological studies revealed a right posteroseptal accessory pathway. Mapping was done by anterograde venography with a deflectable quadripolar electrode 6 Fr catheter and distal thermocouple (Cordis®). After the ablation catheter was positioned, heparin was given with an initial bolus injection of 100 UI/kg, followed by 1000 UI/h. Applications of 60°C were programmed for 60 seconds. The first two applications were stopped after 10 seconds because they were ineffective. A third application eliminated pre-excitation impulses in the first second, and this result persisted for 60 seconds with stable impedance. Total duration of the procedure was 3.6 hours, and duration of x-ray examination was 4.75 min.

An echocardiogram taken immediately after ablation showed a longitudinal thrombus along the right atrium and ventricle to the outflow tract, apparently adhered to the tricuspid valve. A repeat echocardiogram taken a few minutes later failed to show evidence of thrombus formation. No recurrences were observed on hemodynamics or respiratory function. A chest x-ray showed no indications of consolidation, and computed tomography with contrast medium showed nonocclusive filling defects in both left inferior lobar arteries and in at least one of the right inferior lobar arteries (Figure 1). After the ablation procedure the child was kept on dicoumarin anticoagulants for 6 months, and no recurrence of thromboembolic phenomena was seen.

Although radiofrequency ablation is considered a safe technique, the incidence of thromboembolic complications is non-negligible (between 0.8% and 1.3% depending on the series). In recent years several groups have investigated the factors that influence the creation of a prothrombotic state. The biochemical markers that best reflect this condition are thrombin-antithrombin complex and D-dimer, a marker of fibrinolysis. Different studies have concluded that the concentration of both markers is significantly increased after insertion of the sheaths. However, the greatest increase occurs during electrophysiological study before radiofrequency ablation is begun, and this increase is directly proportional to the duration of the study and the number of catheters used. The radiofrequency ablation procedure itself is followed by a further nonsignificant increase in markers, and their concentrations remain elevated for at least 48 hours.

Anticoagulation protocols vary widely between centers. Some groups use no anticoagulation during ablation procedures in the right side of the heart and do not use platelet antiaggregants for these patients. Intravenous heparin is usually not given until the electrophysiological study is completed, before the ablation procedure is begun. It now seems evident that all patients should receive anticoagulation treatment immediately after the guidewire is inserted. Heparin can in fact be given subcutaneously, with or without platelet antiaggregants, before the procedure is started. We emphasize the importance of obtaining an immediate post-procedure echocardiogram, as an intracardiac thrombus may be rapidly released to the pulmonary or systemic circulation and go undetected until complications have developed.

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Fig. 1. Helicoidal computed tomography with intravenous contrast. This axial section shows filling defects in a left inferior lobar artery (A) and right inferior lobar artery (B), with partial obstruction of the lumen.

REFERENCES


