

Case Report

Pathological Findings of Cavotricuspid Isthmus Tissue Eighteen Days after Radiofrequency Catheter Ablation for Typical Atrial Flutter

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A 75-year-old man with a prior myocardial infarction, who underwent a coronary artery bypass graft, suffered from typical atrial flutter. He underwent a cavotricuspid linear catheter ablation. Eighteen days after the ablation, he suddenly died. A transmural ablation line was created between the inferior vena cava and tricuspid annulus. Transmural loss of the cardiomyocytes and small clusters of coagulative necrosis were observed. Evidence of edema and a patchy hemorrhage remained in the extracellular space.

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Key words: Atrial flutter, Pathology, Cavotricuspid isthmus

Linear ablation of the cavotricuspid isthmus is a generalized radical therapy for treating typical atrial flutter.¹⁾ However, there are few pathological reports after the linear ablation of the cavotricuspid isthmus.²⁾ This report describes a case with pathological findings of the cavotricuspid isthmus 18 days after a linear ablation for typical atrial flutter.

Case Report

A 75-year-old man, who had a prior myocardial infarction resulting in a poor left ventricular ejection fraction (29%), underwent coronary artery bypass grafting. Ten days after the operation, he developed atrial flutter (**Figure 1**). Twenty-three days after the operation, he underwent an electrophysiological study and was diagnosed with typical atrial flutter. He underwent an ablation procedure in which radiofrequency current was delivered with an 8-mm-tip

ablation catheter with a temperature setting of 60 °C in the temperature control mode and the power output limited to 40 W. Bidirectional isthmus conduction block was achieved by applying ablation in a single line across the cavotricuspid isthmus. Unfortunately, 18 days after the ablation, he died suddenly.

Six hours after his death an autopsy was performed. The macroscopic observation of the cavotricuspid isthmus is shown in **Figure 2**. **Figure 2A** shows the endocardial site. **Figure 2B** shows the epicardial site and his right coronary artery, which was not damaged, but was removed. A linear lesion was clearly observed to have been created in a straight line transmurally between the tricuspid annulus and inferior vena cava, and its color had changed to a dark brown. The right atrial segments from the inferior vena cava region to the tricuspid annulus were dissected along that line. The tissue was transversely sectioned serially at 3 mm intervals.

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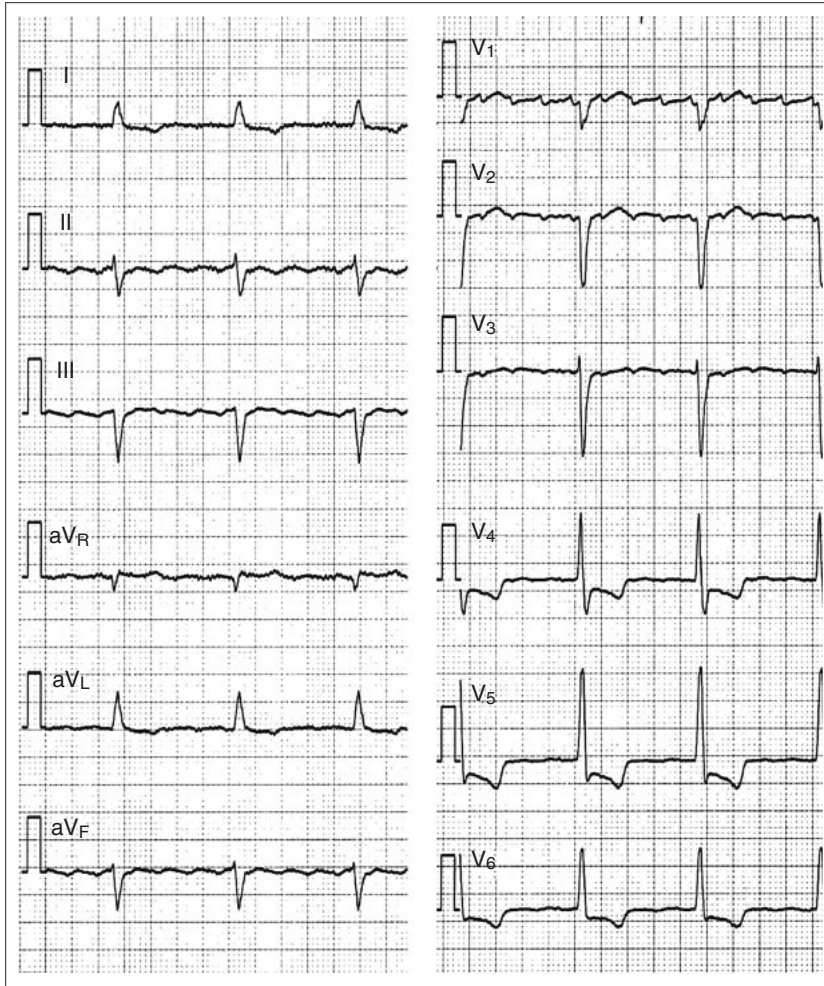


Figure 1 Twelve-lead ECG showing atrial flutter.

All 3-mm slabs showed that the ablation line along the cavotricuspid isthmus was created transmurally (**Figure 2C**). Each 3-mm slab was routinely processed and stained with hematoxyline (**Figure 3A, 3C,**) and Masson's trichrome (**Figure 3B,**). Histologically, a transmural loss of the cardiomyocytes was observed (within the dashed rectangle and **Figure 3C**). In that region, small clusters of coagulative necrosis were observed at the epicardial site, and evidence of edema and a patchy hemorrhage remained in the extracellular space. Inflammatory cells were rarely visible. In the area unaffected by the ablation, mild fatty replacement and interstitial fibrosis were consistently observed. Myocardial tissues was visible (**Figure 3A and 3B,** arrow).

Discussion

This report describes pathological changes occurring less than 1 month after a radiofrequency

catheter ablation procedure.

The cavotricuspid isthmus ablation is an established therapy for the treatment of typical atrial flutter.¹⁾ Successful ablation has been defined as an interruption in the atrial flutter, bidirectional isthmus conduction block, and no induction of persistent typical atrial flutter.²⁾ In this case, a transmural linear lesion was observed in a straight line between the tricuspid annulus and inferior vena cava, which coincided with the electrophysiological findings after the ablation.

Matsui et al.²⁾ reported that, 38 days after ablation there was complete replacement of the fibrotic lesion along the ablation line. In porcine³⁾ and canine⁴⁾ studies 1 month after the ablation, the histologic images of the ablation lesions revealed that the lesions had been replaced with fibrotic tissue. In the case reported here, coagulative necrosis and a hemorrhage remained, but there was no fibrotic tissue observed. In the canine study, after more than

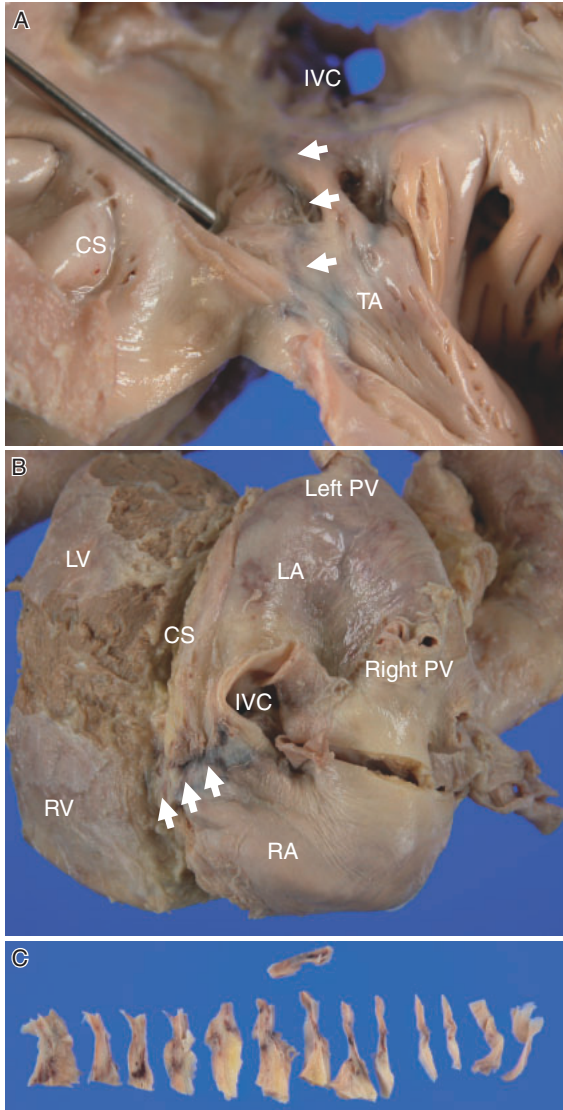


Figure 2 A: Endocardial view of the ablation site. B: The epicardial view of the ablation site and the right coronary artery, which was not damaged, but was removed. C: The right atrial segments from the inferior vena cava region to the tricuspid annulus were dissected along that line. The tissue was transversely sectioned serially at 3 mm intervals. A linear lesion was clearly observed to have been created in a straight line transmurally between the tricuspid annulus and inferior vena cava, and its color had changed to a dark brown. IVC, inferior vena cava; TA, tricuspid annulus; CS, coronary sinus; LV, left ventricle; RV, right ventricle; RA, right atrium; LA, left atrium; PV, pulmonary vein.

2 weeks had passed, the peripheral reparative phenomena had begun and the necrotic areas were smaller. The inflammatory cells progressively disappeared as the density of the fibroblasts increased. The reparative phenomena cited were similar to what we found in our case.

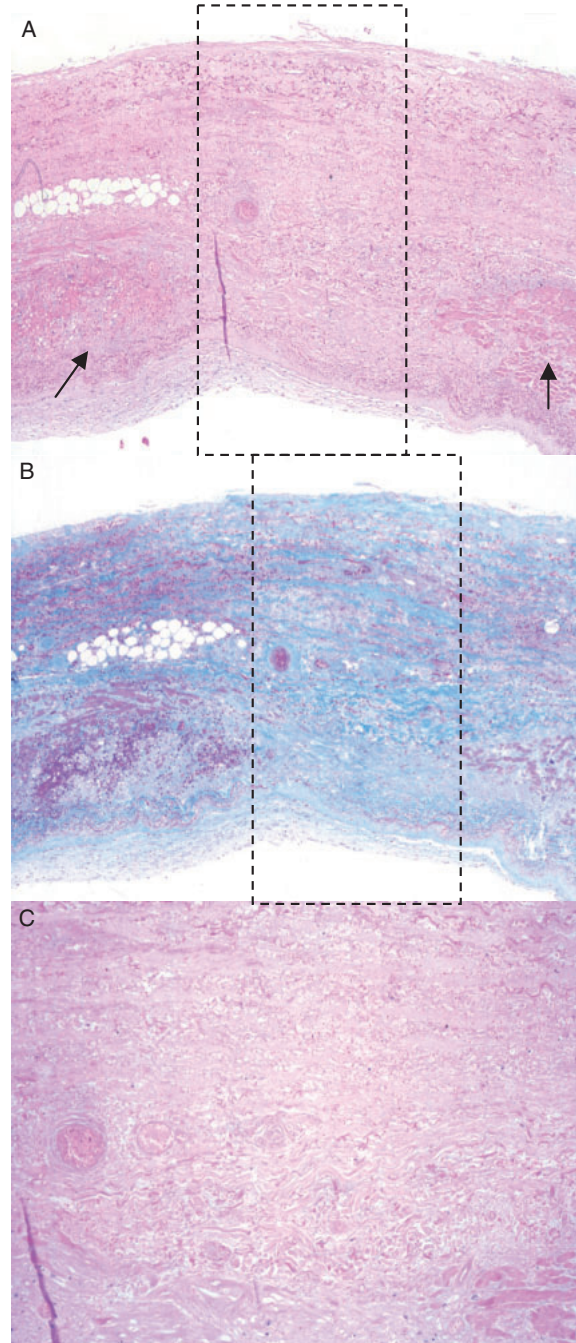


Figure 3 A: Transverse serial sections along the cavotricuspid isthmus linear ablation line. Each 3-mm section was stained with hematoxyline. Within the dashed rectangle, a transmural loss of the cardiomyocytes is observed with small clusters of coagulative necrosis. B: Each 3-mm section was stained with Masson's trichrome. Within the dashed rectangle edema can also be observed. C: Each 3-mm section was stained with hematoxyline. A patchy hemorrhage is observed in the extracellular space, and very few inflammatory cells are visible.

In our case, some slight cardiac edema remained. Schwartzman et al.³⁾ using echocardiography, revealed that the cardiac edema resulting from linear ablation resolved within 4 weeks. Matsui et al.²⁾ reported that no edema was observed 38 days after the ablation. The findings in our case suggested that cardiac edema would resolve 3 to 4 weeks after the ablation.

The patient had a prior myocardial infarction resulting in a poor left ventricular ejection fraction (29%) and underwent coronary artery bypass grafting. The cause of death in our case is suspected to be the ventricular arrhythmia. As for the clinical course of this case, it was similar to that following normal cardiac operations. Therefore, we considered the condition of the heart to be that of a normal 18-day postoperative heart.

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