

Development of delayed cardiac tamponade 55 days after catheter ablation for atrial fibrillation with a new oral anticoagulant

Takeshi Kitamura · Seiji Fukamizu ·
Harumizu Sakurada · Masayasu Hiraoka

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1 Case

A 66-year-old man underwent catheter ablation for paroxysmal atrial fibrillation (AF) using an irrigation catheter, for which the 15-mg rivaroxaban (standard dose in Japan) he had been taking was discontinued. Pericardial effusion (PE) was not evident on the post-procedure intracardiac echocardiography, and rivaroxaban was re-administered. He was discharged symptom-free from the hospital 2 days later (blood pressure [BP], 118/56 mmHg; heart rate [HR], 80 bpm). He remained symptom-free at follow-up 30 days post-procedure (BP, 124/80 mmHg; HR, 70 bpm). Serum hemoglobin was unchanged at 16.6 mg/dL. He developed chest discomfort with hypotension 55 days after the procedure (BP, 104/80 mmHg; HR, 90 bpm). Transthoracic echocardiography revealed a large amount of PE, prompting a diagnosis of delayed cardiac tamponade (DCT). Emergency pericardiocentesis was performed to aspirate hemorrhagic effusion (850 mL), followed by rapid symptom improvement. Serum hemoglobin decreased to 11.9 mg/dL, necessitating a blood transfusion. Rivaroxaban was discontinued. He experienced no further signs of PE.

2 Discussion

To the best of our knowledge, this case presents the most delayed DCT occurrence in the literature [1–3]. Further, this is the first DCT case reported with the use of a new oral anticoagulant (NOAC). The global incidence of AF ablation-related cardiac tamponade is 1.31 % [2], while the global DCT incidence is 0.2 % with a 5 % mortality rate [1]. In a previous report, the independent predictors of a DCT event include excessive volume infusion, irrigation catheter use, and a procedure for paroxysmal AF, following which DCT has developed a median of 12 days (range 0.2–45 days) later [1]. The potential mechanisms of DCT include a rupture of the sealed ablation-induced left atrial wall or small pericardial hemorrhages due to the intense post-procedural anticoagulation [3]. DCT could also occur in the setting of Dressler's syndrome where non-hemorrhagic PE accumulation develops suddenly [3]. In our case, abrupt symptom development and hemorrhagic PE are consistent with an acute process; however, the mechanism is unknown.

As NOACs have been widely used, attention must be paid to DCT signs/symptoms after AF ablation not only during the perioperative period but also during clinical follow-up.

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T. Kitamura (✉) · S. Fukamizu
Department of Cardiology, Tokyo Metropolitan Hiroo Hospital,
2-34-10 Ebisu, Shibuya-ku, Tokyo, Japan
e-mail: take1124@hotmail.co.jp

H. Sakurada
Tokyo Metropolitan Health and Medical Treatment Corporation
Ohkubo Hospital, 2-44-1 Kabuki-cho, Shinjuku-ku, Tokyo, Japan

M. Hiraoka
Toride Kitasohma Medical Center Hospital, 1926 Nonoi, Toride City,
Ibaraki, Japan