

Long Extending Thrombus Formation Around a Transseptal Puncture Site After Transcatheter Edge-to-Edge Mitral Valve Repair

Ai Kagase, MD; Masanori Yamamoto, MD; Mikan Kojima; Takahiro Tokuda, MD

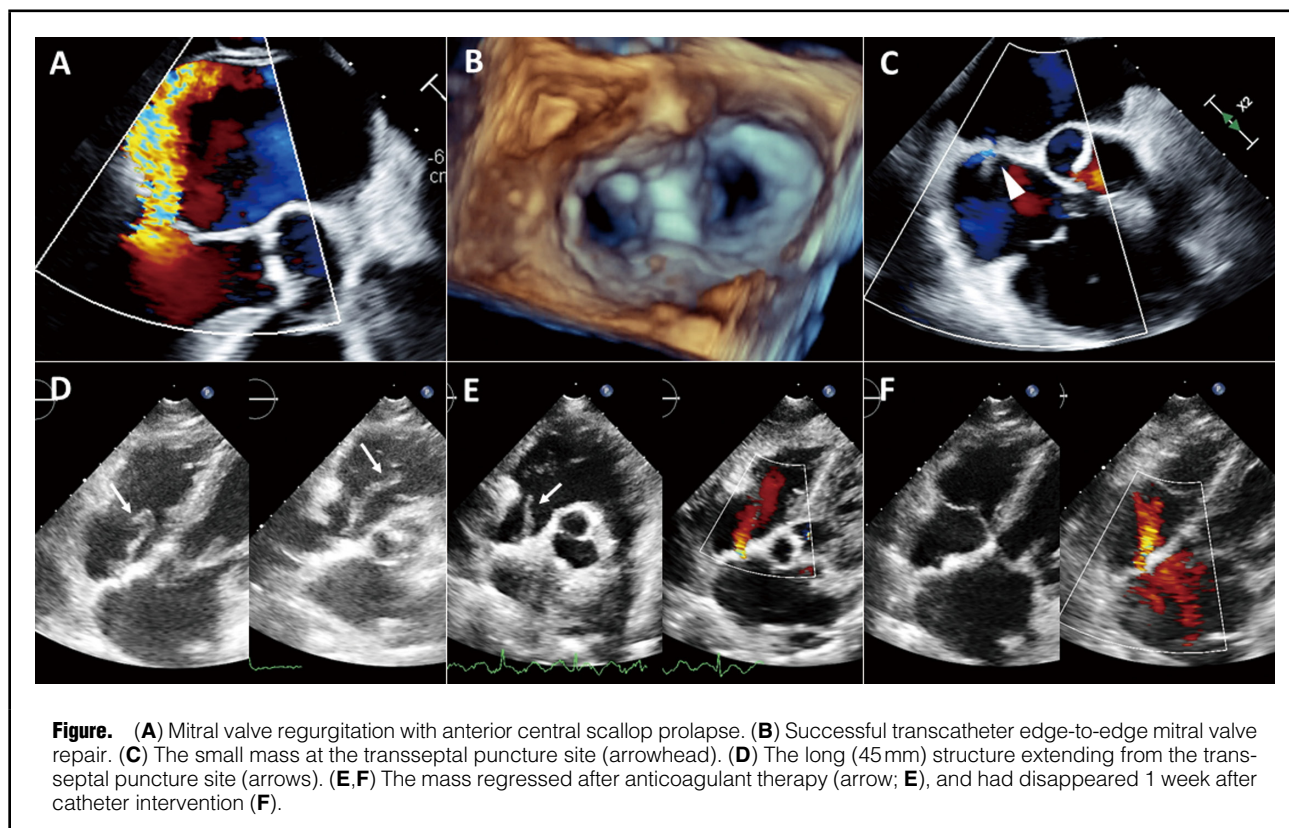


Figure. (A) Mitral valve regurgitation with anterior central scallop prolapse. (B) Successful transcatheter edge-to-edge mitral valve repair. (C) The small mass at the transseptal puncture site (arrowhead). (D) The long (45 mm) structure extending from the transseptal puncture site (arrows). (E,F) The mass regressed after anticoagulant therapy (arrow; E), and had disappeared 1 week after catheter intervention (F).

A 90-year-old woman was transferred to Nagoya Heart Center because of cardiogenic shock with severe mitral valve regurgitation. The mitral valve regurgitation was caused by chordal elongation, which led to anterior central scallop prolapse (Figure A). Transcatheter edge-to-edge mitral valve repair (TEER) was successfully performed without complications (Figure B). After TEER, transesophageal echocardiography showed a newly developed small mass not found previously in the

right atrium around the transseptal puncture (TSP) site (Figure C). The patient had no history of coagulation disorders and oral anticoagulation was not prescribed; however, dual antiplatelet therapy was administered because of recent coronary stenting. The active clotting time was maintained over 200s during TEER. The following day, transthoracic echocardiography (TTE) revealed a wriggling, long structure extending from the TSP site to the right ventricle (Figure D; Supplementary Movie). We suspected

Received May 16, 2022; revised manuscript received September 14, 2022; accepted September 17, 2022; J-STAGE Advance Publication released online October 29, 2022 Time for primary review: 37 days

Department of Cardiology, Nagoya Heart Center, Nagoya (A.K., M.Y., M.K., T.T.); Department of Cardiology, Toyohashi Heart Center, Toyohashi (M.Y.), Japan

Mailing address: Masanori Yamamoto, MD, Department of Cardiology, Nagoya Heart Center, 1-1-14 Sunadabashi, Higashi-ku, Nagoya 461-0045, Japan. E-mail: masa-nori@nms.ac.jp

All rights are reserved to the Japanese Circulation Society. For permissions, please e-mail: cr@j-circ.or.jp

ISSN-2434-0790



this structure to be a thrombus and started anticoagulant therapy using heparin. No clinical symptoms relating to thrombotic events or infective endocarditis were observed during anticoagulation therapy. TTE findings confirmed that the thrombus gradually regressed; it disappeared 1 week after starting anticoagulation therapy (**Figure E,F**). Thus, it is conceivable that TEER caused the formation of the new thrombus at the TSP site, which could have contributed to the potential risk for thromboembolism because thrombi can grow if left untreated. The thrombus resulting from TEER may be small and disregarded as an artifact. However, the imaging findings here suggested that the thrombus had extended, thus increasing the risk of systemic embolism.

This case is reported in accordance with the Declaration of Helsinki and was approved by the Ethics Committee in Nagoya Heart Center (Reference no. 1012-08).

Disclosures

A.K. and M.Y. are members of the transcatheter training faculties at Abbott. The other authors have nothing to disclose.

Supplementary Files

Supplementary Movie

Please find supplementary file(s);
<https://doi.org/10.1253/circrep.CR-22-0051>