Coronary Plaque Erosion after Abemaciclib Treatment Onset: An Unknown Side Effect?

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Abstract

The current article describes a 72-year-old woman who suffered an acute myocardial infarction due to plaque erosion (PE) 2 weeks after abemaciclib treatment onset due to advanced breast cancer. Abemaciclib is a cyclin-dependent kinase 4 and 6 inhibitor that has recently demonstrated efficacy and safety in advanced breast cancer. Of major concern, however, reported thromboembolic rates in randomized clinical trials testing this drug range from 0.6 to 5%. To the best of our knowledge this is the first thrombotic coronary side effect ever reported. We suggest that a treatment that increases thromboembolic risk, such abemaciclib, may have triggered PE in our patient, 15 days after abemaciclib initiation. New molecules are promising in cancer treatment; however, care must be paid to their potential cardiotoxic effects.

Keywords
- thrombosis
- acute myocardial infarction
- cardiology

A 72-year-old woman without cardiovascular risk factors was seen in the emergency department for chest pain. Due to advanced breast cancer she had initiated abemaciclib treatment 2 weeks prior to her presentation. The electrocardiogram showed ST-segment elevation in V2–V5. Emergent coronary angiography revealed a mild stenosis in the proximal left anterior descending (LAD) coronary artery with normal coronary flow (►**Fig. 1A**). Optical coherence tomography of the proximal LAD stenosis revealed a large mixed (red and white) thrombus with some posterior shadowing, but with no signs of plaque rupture, and a good residual lumen, suggestive of plaque erosion (PE) (►**Fig. 1B,C**). Immediately proximal to this segment, a large plaque with lipid content and infiltrated (bright speckling) neointima, was also detected (►**Fig. 1D**). Conservative management with aspirin, ticagrelor, and enoxaparin was decided. Troponin T peak was 512 ng/L (normal < 28 ng/L). Clinical course was favorable. The patient was discharged on dual antiplatelet therapy.

PE has been described as the underlying pathological substrate in up to one-third of patients with acute myocardial infarction. Advances in intracoronary imaging have improved anatomical characterization of PE in the clinical setting.[1,2] In contrast with plaque rupture, the pathophysiological mechanisms leading to PE remain poorly understood.[1] Although female gender and smoking have been linked as risk factors of PE,[1] other predisposing factors and triggers remain unknown. Abemaciclib is a cyclin-dependent kinase (CDK) 4 and 6 inhibitor that has recently demonstrated efficacy and safety in advanced breast cancer. Of major concern, however, reported thromboembolic rates in randomized clinical trials testing this drug range from 0.6 to 5%. These may significantly underestimate actual thromboembolic complication rates observed in real-world patients.[3] Furthermore, out of the reported cases of adverse reaction to CDK4/6 inhibitors between years 2018 and 2019, 2.9% were cardiotoxicities.[4] The pathophysiological basis for abemaciclib embolic risk and cardiotoxicity is not clearly understood. We suggest that a treatment that increases thromboembolic risk, such abemaciclib, may have triggered PE in our patient, 15 days after abemaciclib initiation. Alternatively, this drug may enhance the thrombotic component occurring during the acute phase of PE explaining the clinical presentation as an ST-segment elevation acute myocardial infarction.[5] This case, despite its isolated nature, strikingly illustrates that although new

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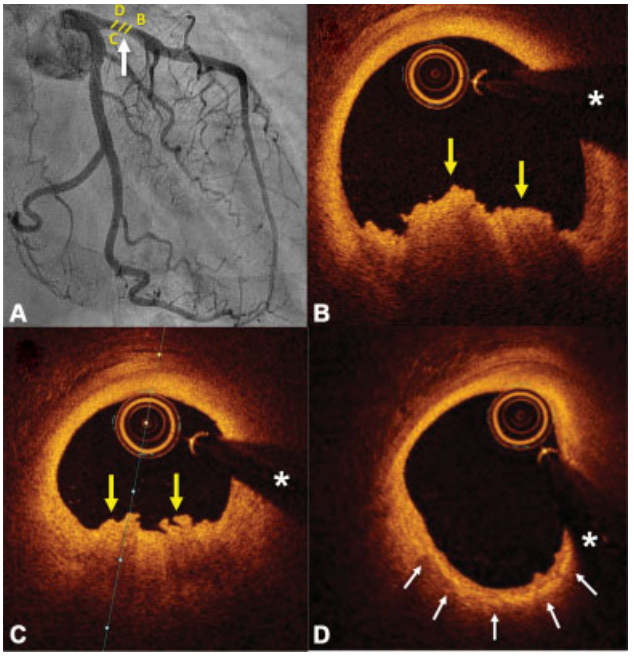

Fig. 1 (**A**) Coronary angiogram showing a mild stenosis (white arrow) on the proximal left anterior descending coronary artery (LAD). (**B–C**) Characteristic images suggestive of plaque erosion with some overlying thrombus (yellow arrows). (**D**) Immediately adjacent proximal plaque with a large lipid content and an infiltrated (bright speckling) neointima (white small arrows). *Denotes wire artifact.

molecules are promising in cancer treatment, care must be paid to clarify and monitor their potential cardiotoxic and prothrombotic effects.

Conflict of Interest

None declared.

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