Coronary Kinking in a Marathon Runner: A “Dangerous Liaison”?

Tortuosidad arterial coronaria en un corredor de maratón: ¿una «relación peligrosa»?

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We report the case of a 48-year-old male marathon runner, without any cardiovascular risk factor, referred to our hospital for an anterior ST-segment elevation myocardial infarction that occurred during endurance training. Coronary angiography showed thrombus in the mid segment of the left descending artery, localized just before a sharp angle of the vessel that produced a peculiar “kinking” of the coronary artery (Figure A), with preserved TIMI 3 flow. Successful manual thromboaspiration (Eliminate™, TERUMO, Japan), together with abciximab bolus followed by 12-hour infusion, were performed with almost complete resolution of thrombus in the acute phase (Figure B). After 3 days, a new coronary angiography confirmed persistence of artery patency with complete resolution of thrombus (Figure C). This is an emblematic case allowing us to unravel a pathophysiological cause of acute coronary syndrome. Previous in vitro studies demonstrated that high-intensity exercise is able to induce platelet hyperreactivity and a hypercoagulable state mediated by the shear stress mechanism. In addition, systolic kinking of coronary artery, often associated with myocardial bridging, might cause ischemia through direct damage to the underlying endothelial cells. Furthermore, anatomic properties of myocardial bridging may predispose to myocardial infarction through the convergence of increased atherosclerosis at the entry site of the tunneled coronary segment. In our case, exercise-induced high heart rate, shortened diastolic perfusion time, increased contractility, compression of artery and increased flow velocity might have caused myocardial ischemia and locally impaired shear stress with subsequent acute thrombus formation. This case could represent a paradigm of ST-segment elevation myocardial infarction in which long-term antiplatelet therapy may be preferred over stent implantation.