No reflow phenomenon decelerating cardiac tamponade from a guidewire-induced distal coronary perforation: a paradoxal ‘protective’ effect

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ABSTRACT
Guidewire-induced coronary artery perforation during percutaneous coronary intervention is a potentially dramatic complication that can cause acute pericardial tamponade. We report a case of a male patient who had no reflow and a missed guidewire-induced distal coronary perforation after a primary coronary intervention for ST segment elevation myocardial infarction at a district hospital. It seems that the poor coronary flow post-procedure decelerated the accumulation of blood in the pericardial cavity and prevented the development of full-blown tamponade immediately post-procedure. This is the first description of this paradoxal ‘protective’ effect of no reflow in a patient with this unusual combination of two adverse outcomes after a primary coronary intervention.

INTRODUCTION
Guidewire-induced coronary artery perforation during percutaneous coronary intervention (PCI) is a potentially dramatic complication, which can cause pericardial tamponade and cardiogenic shock and may be associated with high morbidity and mortality.1,2 Studies report that the incidence of coronary perforation during PCI is 0.3–0.6%, with distal guidewire-related perforation accounting for 31–51% of these cases.3

We report a case of a 70-year-old male patient who had initially presented with crushing central chest pain due to anterior ST segment elevation myocardial infarction in a district hospital with PCI ability. He was a relatively late presenter, as he had experienced several episodes of intermittent chest discomfort for a few days prior to his admission, while on the day of admission the chest pain had been continuous for more than 10 h. After being loaded with 500 mg aspirin and 600 mg clopidogrel, he underwent emergency coronary angiography which demonstrated a subtotal occlusion in the left anterior descending artery (LAD), followed by primary PCI with implantation of three drug eluting stents at the proximal and mid segment of LAD.

The chest pain and the ST elevations did not resolve following the primary PCI. The central chest pain persisted for several hours, and a pleuritic component developed slowly in the next few hours post-PCI. An echocardiogram in the coronary care 4 h after PCI showed a small circumferential pericardial effusion (up to 1.0 cm in diastole adjacent to the right ventricle) with no evidence of pericardial tamponade and no haemodynamic compromise (Fig. 1). These findings were considered as consistent with post-infarction pericarditis, and the patient was treated with high-dose aspirin. The pleuritic chest pain resolved completely during the second day of hospitalization. However, serial echocardiograms demonstrated a slow and progressive increase of his pericardial effusion (up to 2.0 cm in diastole adjacent to the right ventricle 4 days later).

His haemodynamic status deteriorated on the 5th day post-infarction, and he was then transferred to our tertiary cardiology department. On arrival, the patient had clinical signs of impending cardiac tamponade (sinus tachycardia at 105 beats per minute and hypotension with a systolic blood pressure of 100 mmHg). A new echocardiogram on arrival to our department demonstrated a large circumferential pericardial effusion up to 2.5 cm in diastole adjacent to the right ventricle (Fig. 2A), and severe systolic impairment of the left ventricle with an ejection fraction of approximately 25%, due to akinesia of the anteroapical...
segments and the apex. The inferior vena cava was dilated (2.58 cm at end-expiration) (Fig. 2B) with a blunted respiratory response of ~25% at deep inspiration.

A review of the images of the coronary procedure, which had been performed at the district hospital, showed that the distal end of the coronary guidewire had an exaggerated curvature and was located outside the lumen of the distal LAD (likely in the pericardial cavity) (panels A and B – white arrows). Panel A: Right anterior oblique view with cranial angulation. Panel B: Left anterior oblique view with caudal angulation.

Due to further deterioration of the haemodynamic status and development of pulsus paradoxus, the patient went to the theatre and had a successful emergency pericardial window. Six hundred milliliters of blood were
removed from the pericardial cavity. There was no active extravasation of blood from the distal LAD into the pericardial cavity at the time of surgery. The patient’s condition improved slowly and he was discharged a week later. He was stable and clinically well when he was reviewed in the clinic 6 weeks post-discharge.

Distal coronary perforation induced by a guidewire is a potentially devastating complication of coronary angioplasty that may lead to acute cardiac tamponade and rapid haemodynamic deterioration. This latter condition is usually fatal unless it is treated with emergency decompression of the pericardial cavity with pericardiocentesis or surgery. Early recognition of distal guidewire-induced coronary perforation and prompt treatment is essential to prevent life-threatening cardiac tamponade. Strategies such as coil embolization with autologous fat particles through a microcatheter may successfully seal the perforation before cardiac tamponade develops. In the patient we describe, it seems that the – usually detrimental – no reflow and poor coronary flow after PCI decelerated the accumulation of blood in the pericardial cavity and prevented the development of full-blown tamponade immediately post-procedure. To the best of our knowledge, this is the first description of this paradoxal ‘protective’ effect of no reflow. Periprocedural development of pericardial effusion in the setting of PCI should raise suspicion of iatrogenic injury of the treated coronary artery.

Authors’ contributions
Konstantinos Konstantinou contributed towards data collection, reference search and article writing; Konstantinos Aznaouridis conceived the idea of this case report and contributed towards data collection, article writing and proof reading; Constantina Masoura contributed towards critically reviewing the manuscript and proof reading; George Lazaros contributed towards critically reviewing the manuscript and proof reading; Dimitrios Tousoulis contributed towards critically reviewing the manuscript and proof reading.

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