Conservative management of broken guidewire: Case reports

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Abstract
Fractures of coronary guidewires during percutaneous coronary intervention within a coronary vessel lumen are a rare but serious complication. There have been several cases reported in the literature, some managed with surgical intervention, others with medical therapy. We present two prospective cases from our center. Both cases were managed successfully with medical therapy.

Keywords
Coronary angiography, fractured guidewire, retained guidewire, entrapped guidewire, incarcerated guidewire, conservative management

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Introduction
Percutaneous transluminal coronary angioplasty (PTCA) is a useful management strategy in the treatment of ischemic heart disease. Entrapment and fracture of an angioplasty guidewire is a very rare complication (0.22%) which may lead to significant morbidity and mortality.\(^1\) In our institution, of the 1308 cardiac catheterization procedures performed within the last year, there was only one case complicated by the fracture of a guidewire (0.07%). The management of fractured guidewires ranges from conservative management to interventional/surgical strategies depending on the location, chronicity, and clinical situation of the patient. Verbal consent to report the cases was obtained from the patients.

Case reports
The first case describes a 60-year-old female presenting with typical chest pain and found to have ST-elevation myocardial infarction (STEMI). Her medical comorbidities are significant for arterial hypertension (HTN) and end-stage renal disease requiring hemodialysis. Her physical exam was normal and electrocardiography (ECG) demonstrated ST elevations in the anterior lead territory. The patient was referred for immediate coronary angiography. During the diagnostic portion of the angiogram, a 95%, “Type C” lesion\(^1\) was noted in the proximal left anterior descending (LAD) coronary artery (Figure 1). Thrombolysis in Myocardial Infarction (TIMI) 2 coronary flow was seen on the diagnostic portion of the cardiac catheterization, and a 6F XBLAD 3.5 guide catheter was used to cannulate the vessel. Initial attempt to cross the lesion utilizing a Balanced Middle Weight (BMW) wire was unsuccessful due to a tortuous proximal segment of the LAD. The BMW wire was then removed without incident. A Whisper 0.014 × 190 cm wire was then inserted with successful crossing of the LAD lesion. Angioplasty was performed with a Maverick 2.5 × 12 mm balloon. An attempt was made at passing a 2.5 × 24 mm Resolute stent over the Whisper wire. The attempt was unsuccessful due to proximal tortuosity. The case was further complicated by the creation of a non-flow-limiting dissection of the LAD just distal to the first diagonal branch. A Runthrough NS 0.014 × 180 cm wire was placed distally in the LAD as a buddy wire,
adjacent to the Whisper wire, and a 2.5 × 12 mm Xience stent was delivered over the Runthrough wire to the LAD lesion. Prior to the deployment of the stent, the Whisper wire was removed, and at this point, it became obvious that the Whisper wire had fractured. The distal segment of the Whisper wire remained within the distal LAD, which was noted to have a small vessel lumen. Attempts to remove the retained segment of the Whisper wire were employed after the successful stent deployment. Additional wires were utilized to twist around the retained segment but were unsuccessful. Various snares and GuideLiner employment were also unsuccessful. Further options, inclusive of continued percutaneous techniques and surgical techniques were discussed in detail with other team members. The patient was noted to be hemodynamically stable, with TIMI 1 flow in the distal LAD (Figure 2). The decision was made to leave the segment of broken guidewire within the small caliber distal LAD, as risks of complication from further manipulation would have exceeded the potential benefits (Figure 3). Following completion of the procedure, there was no improvement in angiographic appearance of the 95% stenosis and the flow remained at TIMI 1. The residual lesion was diffuse and had a hazy appearance.

Echocardiography in the catheterization laboratory showed severe hypokinesis of the distal anteroseptal, apical, and inferoapical segments with an estimated left ventricular ejection fraction (LVEF) of 40% with no evidence of pericardial effusion. The patient was observed in the Coronary Care Unit (CCU) for 3 days. The patient was treated with aspirin and clopidogrel. A repeat echocardiogram on the third day of admission demonstrated similar findings as the previous study. The patient was discharged without incident. After discharge, the patient was followed in the outpatient continuity clinic with the Cardiology. For the 12 months since the initial STEMI and the retained wire, the patient was noted to be chest pain free, in stable condition with no further events.

The second case describes a 62-year-old female, referred from another institution, presenting with chest and epigastric pain. Her medical comorbidities include a known retained guidewire from a failed PTCA, a 6-cm segment of a Whisper 0.014 × 190 cm Hi-Torque wire, located in the left circumflex artery (LCX), in addition to HTN, diabetes mellitus, and...
peripheral vascular disease (Figures 4–7). The PTCA attempt was done 12 months prior to this admission, for a similar clinical scenario. Physical exam was remarkable for tachycardia, hypoxia, and hyperglycemia. ECG showed sinus tachycardia with an old left bundle branch block (LBBB). Computed tomography (CT) angiography of the chest was performed to rule out pulmonary embolism (PE); however, the results were interpreted as equivocal. As a precaution, the patient was started on therapeutic doses of Lovenox. Troponin I levels were monitored and noted to elevate from 0.08 to 0.25 ng/mL upon which the patient was transferred to CCU. Ventilation/Perfusion scan was performed. Pulmonary team was consulted for hypoxemia and questionable PE. Ventilation/Perfusion scan showed homogeneous radiotracer distribution on perfusion images in both lungs. Lower extremity Doppler study was negative for deep vein thrombosis (DVT). The hypoxemia resolved after 24 h, and the etiology was felt to be an exacerbation of her underlying chronic obstructive pulmonary disease (COPD). Therapeutic dosing of Lovenox was decreased to prophylactic dose for DVT. Troponin I peaked at 0.85 ng/mL. As a part of her non-ST-elevation myocardial infarction (NSTEMI) workup, the patient underwent diagnostic cardiac angiogram. Diagnostic angiography demonstrated non-obstructive lesions of the mid-LAD and the ostial first diagonal branch. The distal portion of the LCX containing the retained guidewire was noted to be occluded (Figures 8 and 9). No PTCA was performed. The patient was treated with aspirin and clopidogrel. Echocardiography showed an estimated LVEF of 45% with severe hypokinesis of the inferoseptal and inferior segments, as well as mild hypokinesis of inferolateral segments with no significant pericardial effusion. After recovery, the patient was discharged in stable condition with dual antiplatelet therapy including aspirin and Plavix as well as regular follow-up within the cardiology clinic. During the 6-month follow-up since the NSTEMI, the patient remained stable, pain, and event free.

Discussion

While PTCA remains as a mainstay therapy for coronary ischemia, the potential complication of retained angioplasty hardware may have significant consequences.

Guidewire manipulation with tortuous segments of a coronary artery may create a situation in which there is entrapment and overcoiling of the guidewire. This then can lead to excessive bending with consequent fracture of the guidewire.\(^5\)\(^7\) The Whisper wire, which was used in both cases described above, is noted to have the characteristics of being a full polymer wire with a hydrophilic coating. This decreases friction and allows for easier navigation through tortuous vessels at the expense of decreased tactile feedback and increased risk of perforation.

The characteristics of the lesion may additionally have a role in guidewire fracture. Arterial segments which are tortuous and/or heavily calcified lead to increased shearing forces with hardware manipulation/extraction and therefore increase the risk of wire detachment.\(^8\) The guidewire remnants may then serve as a nidus for vascular endothelial injury and platelet deposition, leading to acute thrombus formation\(^9\) or chronic total occlusion of the involved vessel, as seen in our second case report. The retained guidewire fragments in patent coronary arteries may cause arterial narrowing despite systemic anticoagulation.\(^10\)

The management of patients with retained fractured guidewire fragments during PTCA remains particularly challenging.
due to the lack of data regarding clinical outcomes. Multiple interventional modalities have been developed for the retrieval of broken guidewire segments, such as snares, filter wires, additional guidewire manipulation, and retrieval forceps. Currently, there are no guidelines in regard to the optimal management or choice of antiplatelet/anticoagulation regimen for these patients. Conservative management may be appropriate after attempts at retrieval of the broken fragment of guidewire have been unsuccessful and the wire is retained within a small coronary branch with a small area at risk and an acceptable post-occlusion blood flow where the risk of intervention will likely exceed the benefits of more aggressive measures. However, in cases where the area at risk is large and blood flow has been compromised after the failure of endovascular retrieval of broken guidewire fragments, more aggressive measures such as emergent cardiac surgery should be considered.\textsuperscript{11} “The University Hospital IRB permits the publication of case reports for educational purposes.”
Conclusion

In all cases of fractured guidewires, the risks and benefits of aggressive measures must be weighed against one another. Further investigation with large case series/randomized controlled trials would be needed to allow for improved decision making regarding wire type, length, location, and determination of antiplatelet/anticoagulation therapy.

Declaration of conflict of interests

The authors declare there is no conflict of interest.

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