Coronary stent fracture in an octogenarian patient: from bad to worse

Andreas S. Triantafyllis1,2, Petros N. Fountoulakis1, Georgios Charalampidis1, Konstantinos Kotinas1, Pavlos Tsinivizov1, Dimitrios Varvarousis1, Andreas Giannakopoulos1, Sofia Apollonatou2, Areti Stefanidou2, Stamatios Chatzopoulos3, Dimitrios Tsipitsios4, Konstantinos Tsamakis5, Konstantinos Kyfnidis1, Leonidas E. Poulimenos1

1. Department of Cardiology, Asklepieion General Hospital, Athens, Greece; 2. Department of Critical Care Medicine, Asklepieion General Hospital, Athens, Greece; 3. 6th Department of Orthopedics, Asklepieion General Hospital, Athens, Greece; 4. Department of Neurology, Democritus University of Thrace, Alexandroupolis, Greece; 5. Department of Psychiatry, Attikon University Hospital, Athens, Greece

Correspondence to: andtridoc@yahoo.gr
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Stent fracture represents a rare cause of stent failure especially in the era of new generation coronary drug-eluting stents (DES). It is described as the interruption of stent continuity resulting in increased risk of in-stent restenosis and stent thrombosis and predisposes to repeat revascularization.[1] Although a growing number of octogenarians benefit today from an invasive treatment of coronary artery disease (CAD), this benefit could be lost in frail patients with multiple comorbidities.[2] Octogenarians constitute a high-risk population in whom frailty and multiple comorbidities mandate for careful treatment planning.

An 84-year-old woman was transferred to the Emergency Department (ED) due to a fall following an episode of acute chest pain with diaphoresis. She reported angina in minimal exertion for the last couple of months, which was relieved with the use of sublingual nitrates. Her medical history included CAD treated with stent implantation in the proximal left anterior descending (LAD) artery seven years ago in the context of a positive stress test, hypertension and dyslipidemia under treatment with aspirin, valsartan, hydrochlorothiazide, metoprolol and atorvastatin.

At presentation, blood pressure was 180/90 mmHg, with a heart rate of 75 beats/min in sinus rhythm, and an oxygen saturation of 90% on room air. Physical examination revealed bilateral crackles and a third heart sound (S₃) while a painful, shortened and externally rotated right femoral was noted. Electrocardiography (ECG) recorded left bundle branch block (LBBB) with ST-segment depression in leads V2–V6 (Figure 1A). Bedside transthoracic echocardiography (TTE) showed severe hypokinesia of the anterior wall and impaired left ventricular ejection fraction (EF) around 40%. Radiography of the pelvis illustrated a right femoral intertrochanteric fracture (Figure 1B, arrows). High-sensitive troponin-I was elevated (525 pg/mL, normal < 15.6 pg/mL), whereas other routine blood tests, including inflammatory markers, were normal.

During her stay in the ED, the patient rapidly deteriorated and developed acute pulmonary edema not responding to standard therapy with intravenous diuretics, nitrates and non-invasive ventilation, demanding invasive respiratory support with intubation. Emergency computed tomography pulmonary angiography (CTPA) excluded acute pulmonary embolism. Therefore, the diagnosis of acute heart failure due to a non ST-elevation myocardial infarction (NSTEMI) was set and the patient was transferred to the intensive care unit (ICU) for further treatment. After multidisciplinary (MDT) consultation between cardiologists, ICU specialists and orthopedic surgeons, the patient was considered too frail at that point to undergo an urgent coronary angiography (CA). Hence, beyond mechanical ventilation, she was treated conservatively with intravenous furosemide and nitrates, subcutaneous...
enoxaparin, and per nasogastric tube dual antiplatelet therapy with aspirin and clopidogrel. The patient gradually improved and was extubated, however, high-sensitive troponin remained elevated reaching a peak after seven days (7.351 pg/mL, normal < 15.6 pg/mL). With the patient stabilized, we decided to perform CA prior to any orthopedic intervention of the femoral fracture given the very high surgical risk.

CA was performed through the left ulnar artery approach since the radial arteries were not palpable. CA revealed fracture in the middle part of the previously implanted stent of the LAD (Figure 2A, arrow) with severe in-stent restenosis (Figure 2B, arrowheads, Video 1) while the rest of the coronaries showed non-obstructive lesions. After vigorous lesion predilatation with escalating non-compliant (NC) balloons at high pressures, a new 3.0 × 28 mm drug-eluting stent (DES) was implanted with excellent angiographic result (Figure 2C, arrowheads, video 2).

The patient improved impressively after revascularization and gradual mobilization was initiated under close guidance from the revalidation and physio team. The MDT decided conservative treatment of the femoral fracture at that point given the patients’ relatively satisfactory mobility status. Pre-discharge TTE illustrated recuperation of the systolic function of the left ventricle with only mild hypokinesia of the anterior wall, and an ejection fraction (EF) = 50%. She was discharged in very good condition under dual antiplatelet treatment with aspirin and clopidogrel for one year and prophylactic dose of subcutaneous enoxaparin for six weeks. The possibility of surgical intervention of the femoral fracture was to be re-evaluated during monthly scheduled follow-up depending on patients’ clinical status and after weighing the risks of potential cessation of antiplatelet therapy.

At 3-months follow-up, the patient reported no angina or dyspnea and her physical activity and walking speed were evaluated as satisfactory, thus allowing for a conservative treatment of the femoral fracture.

Figure 1  Electrocardiogram showing left bundle branch block with ST-segment depression in leads V2->V6 (A) and radiography of the pelvis illustrating a right femur intertrochanteric fracture (B, arrows).

Figure 2  Fluoroscopy showing fracture in the middle part of the previously implanted stent of the LAD (A, arrow) with severe in-stent restenosis as depicted in angiography (B, arrowheads). Excellent angiographic result after new stent implantation in the LAD (C, arrowheads). LAD: left anterior descending artery.
Current guidelines recommend an invasive strategy in the very elderly population (≥ 80 years) with NSTEMI. However, these recommendations are based on data in which this population was underrepresented. Although studies suggested a clinical benefit from an invasive strategy in octogenarians, this benefit could be lost in frail patients with multiple comorbidities. Additionally, technical difficulties such as heavy calcification can lead to stent failure with dismal outcomes. Decision making on whether to perform percutaneous coronary intervention (PCI) or not in the very elderly population should be very meticulous.

CAD represents a leading cause of global morbidity and mortality with increasing prevalence as life expectancy rises. Almost a fifth of octogenarians with cardiovascular disease are treated with PCI with very good results. The retrospective study of Barywani, et al. depicted the beneficial effects of PCI on the prognosis and survival of octogenarians with ACS. More specifically, PCI improved quality of life for elders with symptomatic CAD and relieved angina when compared to younger patients. Of course, it is noteworthy that, the growing use of the transradial (wrist) approach in the very elderly population, as performed in our case, has reduced significantly the vascular complications of PCI.

The recently published RINCAL randomized trial did not show a survival benefit of PCI versus medical therapy alone in very elderly patients presenting with NSTEMI, despite the relief of angina symptoms, and the decrease in reinfarctions and unplanned revascularizations. However, this trial had just a one-year follow-up which might be too short to show any survival benefit of PCI. Retrospective analysis of the ACOS, ACACIA, LONGEVOSCA and GRACE registries and post hoc analysis of the TACTICS-TIMI 18 trial demonstrated net clinical benefit from an intervention-guided strategy in those aged ≥ 75 years. These studies also underpinned that elderly patients despite being at greatest risk (yet more likely to benefit from intervention), were less likely to be prescribed guideline-mandated pharmacotherapy, to be offered early invasive imaging or receive revascularization. The Norwegian After Eighty study (mean age of 85) demonstrated that an invasive strategy was superior to OMT alone driven primarily by a reduction in non-fatal MI and need for urgent revascularization. Alongside, Sui, et al. recently reported that an invasive strategy was superior to a conservative strategy in reducing mortality in a Chinese cohort of patients aged 80 years or older with NSTEMI. The latest European Society of Cardiology guidelines on NSTEMI (2020) recommend the application of the same interventional strategies for older patients as used for younger patients, despite lack of recent randomized studies or observational data.

Frailty has become an emerging health concern as the aging of the general population progresses. The prevalence of very frail patients undergoing PCI has recently reached almost 12% leading to greater risk of in-hospital mortality, bleeding and vascular complications. Comorbidities and frailty are associated with higher rates of complications and consumption of healthcare resources while a significant association between frailty and a worse prognosis in patients with ACS has been described. Various risk scores, such as the Canadian Study of Health and Ageing Clinical Frailty scale and FRAGILTY index, have been proposed for risk stratification. Frailty Index is considered a well-established screening tool for patients older than 70 years. Frailty assessment represents an overall marker of various indices including impairment of functional (grip strength, walking speed), cognitive and nutritional status (unintentional weight loss). In addition, the implementation of -widely used in cardiology-risk scores, such as the GRACE ACS mortality and CRUSADE bleeding risk scores can be very helpful on risk stratification and decision making. Therefore, octogenarians presenting with ACS may benefit from a successful revascularization strategy after carefully assessing frailty, comorbidities, the risks of the intervention and the suitability to tolerate relevant medication. The combination of a high frailty index (femoral fracture) alongside a high ischemic (stent fracture in the LAD) and bleeding risk (triple antithrombotic therapy) in our patient mandated for a meticulous multidisciplinary approach to plan the optimal therapeutic strategy.

However, clinical characteristics present in octogenarians such as coronary lesions with high calcium content (LHCC), arterial access difficulties, tortuosity, by-pass grafts, diabetes, chronic renal failure and poor adherence to antiplatelet regimens due to bleeding are likely to impact adversely on acute and long-term results of PCI and cause stent failure. Stent fracture represents an important cause of stent failure which may modify the mech-
nctional integrity of the stent resulting in in-stent restenosis and stent thrombosis. The prevalence of coronary stent fracture approaches almost 4%, thus predisposing the patient to repeated revascularization procedures. The pathophysiologic mechanisms of stent fracture are quite complex. According to Ino, et al., stent fracture may originate from alteration of the vascular anatomy after stent implantation accompanied by local reactive endothelial proliferation and coagulation disorders of the affected coronary artery. This pathology is enhanced by mechanical factors such as LHCC, defects of the stent material and design, excessive stent post-dilatation with NC balloons, the location of the responsible lesion as well as procedural aspects. Furthermore, biological factors may contribute to stent fracture and include polymer hypersensitivity, impaired endothelial function and vascular remodeling. Moreover, the amount of hinge motion of the vessel, defined as the difference of the angle in the target lesion between systole and diastole (delta angle), and total stent length may increase the risk of stent fracture. The more increment the delta angle (> 16°) the more intense the hinge motion, leading to higher rates of stent failure. Intense hinge motion causes strut stretching at the time of deployment, repetitive kinking of the stent during the cardiac cycle and increased axial stiffness in areas of overlapping stents. In our case, after meticulous evaluation of the angiographic films of the previous PCI, we hypothesized that excessive post-dilatation with oversized NC balloons caused disruption of the mechanical integrity of the implanted stent resulting in its fracture. The diagnosis of stent fracture can be established with multi-modality imaging. Computed tomography angiography (CTA) is a non-invasive method for the estimation of the stent integrity. Invasive CA remains the first line modality to suspect and reveal stent fracture during fluoroscopy, as in our case. Optical coherence tomography (OCT) has recently become a prominent method for depicting stent morphology, especially in cases of partial stent fracture thanks to its higher resolution (10-15 μm) when compared to intravascular ultrasound (IVUS). In our case, due to inability of the patient to cooperate and the urgency to conclude the procedure in a safe and timely manner, intracoronary imaging with IVUS or OCT could not be performed. In addition to intracoronary imaging, radiological stent enhancement with stent boost, as in our case, can be very helpful to diagnose stent fracture. Treatment of symptomatic stent fracture causing in-stent restenosis is based on careful predilatation with high-pressure or NC balloons followed by implantation of a new DES and dual antiplatelet treatment for at least 12 months after the intervention.

In conclusion, the net clinical benefit from an invasive strategy in octogenarians could vanish in frail patients with multiple comorbidities. Consideration of technical difficulties during the intervention, frailty degree, comorbidities, the adherence to medication and bleeding risks should be carefully weighted in order to avoid stent failure. Decision making on whether to perform PCI or not in the very elderly population should be very meticulous.

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