ABSTRACT: Background. Longitudinal deformation of coronary stents has been recently described and seems to be more frequent with certain contemporary stent platforms. Indeed, in order to increase flexibility and deliverability, stent manufacturers have reduced strut thickness and the number of connectors within cells; this could negatively affect other mechanical properties of the device, such as the resistance to longitudinal stress. Moreover, longitudinal deformation has been associated to adverse events, such as stent thrombosis. Methods. We report 3 cases of longitudinal stent deformation observed at our institution. Results. The first case was a consequence of postdilatation of the stent with a non-compliant balloon, whereas the other 2 cases involved the treatment of bifurcation lesions. One case was complicated by acute, inadvertent stent thrombosis; such a dreadful complication, to the best of our knowledge, has not been previously reported. Conclusions. Although longitudinal stent deformation is an infrequent finding, usually not associated with adverse events, at least in the short term, it can sometimes turn into a catastrophic, life-threatening complication. The growing number of reports about this issue in recent years should prompt the operators to carefully select coronary stents, especially when dealing with certain lesion subsets, such as ostial lesions, bifurcations, and long lesions.

We report 3 cases of LD observed at our catheterization laboratory with the Boston Scientific platinum-chromium (PtCr) stent platform (Promus Element everolimus-eluting stent and Omega bare-metal stent).

Case 1. A 75-year-old woman underwent coronary angiography because of a history of effort angina. She was found to have 2-vessel coronary artery disease, with 2 significant stenoses involving, respectively, the mid-segment of the right coronary artery (RCA) and the mid-segment of the left anterior descending (LAD) coronary artery (Figure 1A). Direct stenting of the RCA was performed with an 4.0 x 20 mm Omega stent at 16 atm; then, direct stenting of the LAD was performed with a 3.5 x 16 mm Promus Element stent deployed at 14 atm (Figure 1B). Despite a good angiographic appearance, we decided to postdilate the stent with a Sprinter NC 3.5 x 9 mm balloon (Medtronic, Inc). Unexpected resistance was felt during advancement of the balloon into the deployed stent; after postdilatation, deformation of the proximal edge of the stent with the appearance of nested struts was evident (Figure 1C). Nevertheless, final angiographic result was good (Figure 1D), the clinical course of the patient was unremarkable, and she was free from adverse events at 30-day follow-up exam.

Case 2. A 65-year-old man underwent staged percutaneous coronary intervention (PCI) on a LAD/first diagonal bifurcation lesion (Medina 0,1,0) (Figure 2A). One week prior, he was admitted with inferior ST-elevation myocardial infarction and received two coronary stents on the RCA (2.5 x 12 mm Promus Element and 4.0 x 20 mm Omega). Before PCI, a pressure wire on the LAD after intracoronary adenosine yielded a fractional flow reserve (FFR) of 0.79. The bifurcation was approached with a provisional, single-stent strategy; after wiring both branches, we directly implanted a Promus Element 3.0 x 20 mm at 12 atm (Figure 2B). Then, according to the proximal optimization technique,5 we performed proximal postdilatation with a 3.5 x 12 mm Sprinter Legend balloon (Medtronic, Inc) inflated at nominal pressure. The angiographic result was suboptimal at the diagonal branch ostium; we then decided to perform kissing balloon. Although guidewires were easily exchanged, difficulties were encountered in advancing a 2.5 x 12 mm Sprinter Legend balloon in the diagonal branch through the stent struts; after this, LD of the proximal stent edge was evident (Figure 2C). Notably, stent struts encroached the vessel lumen, preventing further attempts of stent postdilatation; finally, we had to rewire the vessel and dilate the stent with an undersized balloon (3.0 x 10 mm Sprinter Legend) before terminating the pro-

Key words: longitudinal stent deformation, complications.

The tendency of some coronary stent platforms to undergo longitudinal deformation (LD) has been recently reported both in case series1,2 and in bench tests.3,4 LD is the result of compression applied to the stent struts along the longitudinal axis by other devices, such as guide catheters, postdilatation balloons, or imaging tools. This results in shortening of the stent (the “concertina” effect), with nesting and crushing of the stent struts into and over each other. The clinical implications of this mechanical behavior are not trivial, since it has been associated with a potentially catastrophic event such as stent thrombosis. Moreover, LD often needs to be corrected, usually by postdilatation and, if necessary, implantation of additional stents, making the procedure longer and more complex.

From the Interventional Cardiology, Sandro Pertini Hospital, Rome, Italy. Disclosure: The authors have completed and returned the ICMJE Form for Disclosure of Potential Conflicts of Interest. The authors report no conflicts of interest regarding the content herein.


Address for correspondence: Dr Stefano Rigatti, UO Emodinamica, Ospedale Sandro Pertini, Via dei Monti Tiburtini 385, 00157 Roma, Italy. Email: stefanorigatti@yahoo.it
procedure with a kissing-balloon inflation, achieving good angiographic result both on the main and on the side branch (Figure 2D). The patient was discharged without adverse events; 2 weeks later, he was readmitted because of atypical chest pain and underwent a stress test which was negative.

Case 3. An 81-year-old gentleman with a history of permanent atrial fibrillation and a pacemaker was admitted to our Intensive Coronary Care Unit following syncope with head trauma. In the emergency room, frequent runs of unsustained ventricular tachycardia were discovered; computed tomography scan discovered a subdural hematoma; blood test showed an increase in cardiac markers.

Since arrhythmias were well controlled by medical therapy (amiodarone), a conservative strategy was initially adopted; in addition, according to the consulting neurosurgeon, antiplatelet therapy was contraindicated. Nevertheless, although asymptomatic for angina, the patient experienced several episodes of acute dyspnea, which were considered to be ischemic equivalents since his ventricular systolic function was preserved. Therefore, the patient was referred for coronary angiography; his antithrombotic therapy consisted only of aspirin (100 mg/day) and enoxaparin (4000 UI/day).

Angiography showed significant disease of the LAD (Figure 3A), with a hazy lesion at the bifurcation with the first diagonal branch (Medina 1,1,1). After giving a loading dose of clopidogrel (300 mg), we decided to treat this lesion with a provisional, single-stent approach. After wiring both branches, we directly implanted a 3.5 x 20 mm Omega on the LAD with good angiographic result. Guidewires were easily exchanged, but resistance was felt during advancement of a balloon in the diagonal branch to perform kissing-balloon inflation. After a while, the patient developed sudden chest pain with marked ST changes; angiography revealed acute stent thrombosis with TIMI 2 flow (Figure 3B); bail-out abcix-
imab was immediately given. At this point, we tried to reopen the main vessel, but as in the previous case, stent struts encroached the lumen so that it was impossible to advance even small balloons (1.5 mm diameter) (Figure 3C). A second guidewire was then advanced in the LAD and allowed us to dilate the stent with growing diameter balloons (up to 3.5 mm). TIMI 3 flow was obtained, thrombus aspiration with Export catheter (Medtronic, Inc) was performed, and a second stent (3.5 x 18 mm Vision; Abbott Vascular) was implanted proximally at 14 atm. Final angiography showed a persistent thrombotic burden in the proximal LAD and at the ostium of the diagonal (Figure 3D). Following the procedure, troponin I peaked at 3.79 ng/mL; however, predischarge echocardiography showed preserved left ventricular ejection fraction without new regional contraction disturbances.

**Discussion.** Mechanical properties of coronary stent platforms can significantly affect both periprocedural and postprocedural issues in coronary stenting. For example, high flexibility and low crossing profile allow the operator to reach more easily distal lesions in tortuous vessels, as well as to perform direct stenting; open-cell geometry and wider cell area can facilitate the treatment of bifurcation lesions, making access to side branches easier; stent strut thickness and metal alloy composition can influence vascular response to stenting in terms of inflammation and endothelial injury, ultimately affecting clinical endpoints such as restenosis. Therefore, in recent years, new stent platforms with thinner struts, but preserved strength and radioopacity, were created using alloys such as cobalt chromium and PtCr instead of stainless steel. Nevertheless, efforts to maximize deliverability and conformability of the stents, reducing both strut thickness and the number of fixed connectors between cells, could negatively affect other mechanical properties, such as the resistance of the stent to longitudinal forces.

Since the first report by Hanratty et al, who described 3 cases of longitudinal compression of ostial stents by guide catheter deep engagement, there have been several reports about this issue, involving the Boston Scientific PtCr stent platform in the majority of cases.

The prevalence of this platform is probably due, in part, to its higher radioopacity as compared to other stents, making strut deformation angiographically more evident; nevertheless, Prabhu et al showed that this platform has a markedly lower resistance to longitudinal compression mainly because of its particular design. The importance of the number of connectors as a main determinant of resistance to longitudinal forces was also underlined by Ormiston et al.
LD is a rare phenomenon; in the report by Williams et al., its global incidence was 0.2%, although it was higher in the PtCr platform (0.86%). Notably, LD was not observed in the PERSEUS and PLATINUM randomized controlled trials; this probably reflects the low prevalence of complex lesions (only 21% were American Heart Association/American College of Cardiology type C lesions) as compared to real-world series. Indeed, LD is more likely to happen in complex procedures, such as ostial lesions, bifurcations, and long lesions. In our catheterization laboratory, we only observed LD with the PtCr platform, with an incidence of 1.02% (3 cases out of 240 Promus Element and 54 Omega stent implanted).

The etiology of LD is heterogeneous. It can be induced by secondary devices passed into the stent, such as undeployed postdilatation balloon, intravascular ultrasound catheters, filterwire devices, and even by withdrawal of a buddy wire. In the case of proximal stent edge involvement, causative factors include deep engagement of the guide catheter or guide catheter extensions.

Bifurcation lesions, especially when approached with a provisional, single-stent technique, represent a particular anatomic subset in which LD may happen; in our experience, the critical step is represented by crossing the stent struts with a balloon to dilate the side branch. This may lead to compression of the proximal stent edge, especially if stent struts are malapposed.

In our series, this mechanism was responsible for LD in both case 2 and case 3; furthermore, it was not prevented by proximal stent optimization in case 2.

The clinical impact of LD is not well understood. Theoretically, especially in drug-eluting stents, one could expect an increased risk of thrombosis, possibly due to stent strut malapposition and nesting, as well as increased restenosis rate, since, as a consequence of LD, the stent gets shortened and leaves the proximal part of the target lesion uncovered. Although Leibungdut et al did not report any major adverse cardiac events during 30-day follow-up, serious adverse events were observed in other series, including emergent cardiac surgery to remove trapped devices, acute stent thrombosis a few hours after the procedure, subacute and late stent thrombosis. In case 3, we observed a dreadful complication represented by acute, intraprocedural stent thrombosis, which was also facilitated by suboptimal periprocedural antithrombotic therapy. To the best of our knowledge, this is the first report of an intraprocedural stent thrombosis caused by LD.

Treatment of LD is represented by postdilatation of the stent, in order to correct strut malapposition. However, in some cases this can be extremely difficult, requiring the use of a very small-diameter balloon in order to cross stent struts encroaching on the lumen. Although keeping the guidewire within the deformed stent is considered to be very important in order to
maximize the chances of postdilatation, in cases 2 and 3 in our series, we could not cross the stents even with 1.25 mm balloons and we succeeded only after rewiring the stent with another guidewire.

**Conclusion.** LD, although infrequent, is a potentially serious complication that needs to be prevented rather than treated. In view of this, several technical tricks could be helpful, such as prolonged balloon inflation during stent delivery, in order to achieve better stent expansion, careful predilatation, and proximal optimization technique when treating coronary bifurcation with a provisional approach. In the case of aorto-ostial lesions, crushing the proximal struts with the tip of the guide catheter may be prevented by re-engagement of the guide coaxially over the deflated stent balloon before withdrawal.

Finally, some stent platforms and designs seem to be more vulnerable to LD; therefore, careful device selection should be considered when particular subset of lesions, such as ostial, bifurcation, and long lesions, are being approached.

**References**