

## Diagnostic de fracture du stent : présentations cliniques et modalités d'imagerie.

## Diagnosis of stent fractures: Clinical presentation and imaging modalities

Aymen Noamen<sup>1</sup>, Nadhem Hajlaoui<sup>1,2</sup>, Hassen Ibn Hadj Amor<sup>1,2</sup>, Khalil Houissa<sup>1,2</sup>, Dakher Laahideb<sup>1,2</sup>, Wafa Fehri<sup>1,2</sup>, Habib Haouala<sup>1,2</sup>

1 Cardiology Department, Military Hospital of Tunis

2 Faculty of medicine of Tunis, University of Tunis El Manar

### Résumé

La fracture du stent est une entité sous estimée. Elle regagne d'importance récemment grâce au recours fréquent à l'imagerie endocoronaire. Des préoccupations croissantes concernant cette complication comme une cause potentielle de resténose et de thrombose du stent entraînant des mauvais résultats cliniques. Cela nous incite à établir un algorithme diagnostique clair avec des aspects d'imagerie bien définis et un consensus thérapeutique bien codifié. Nous rapportons trois cas de fracture de stent avec des présentations cliniques variées avec différents outils d'imagerie pour confirmer le diagnostic.

### Mots-clés

Angioplastie complexe, complication, fracture du stent.

### Summary

Stent fracture is an underestimated entity emphasized by the frequent recourse to intravascular imaging tools, rising concerns about it as a potential cause of stent restenosis and thrombosis which can lead to adverse clinical outcomes and underlying the importance to have a clear diagnosis algorithm with a gold standard imaging patterns and consensual therapy. We reported three illustrative cases of stent fracture with various clinical presentation and different imaging tools to confirm the diagnosis.

### Keywords

Complex angioplasty; complication; stent fracture.

### Correspondance

Aymen Noamen

Cardiology Department, Military Hospital of Tunis

E-mail: noaymen@gmail.com

## INTRODUCTION

Stent fracture (SF) is an underestimated entity emphasized by the frequent recourse to intravascular imaging tools. It raises concerns as a potential cause of stent restenosis and thrombosis which can lead to adverse clinical outcomes, underlying the importance to have a clear diagnosis algorithm with a gold standard imaging patterns and consensual therapy.

We report three illustrative cases of SF with various clinical presentation and different imaging tools to confirm the diagnosis

### Case 1 :

A 66 year-old man with a history of hypertension and was a smoker. He presented with recurrent angina in May 2007; 15 months after a percutaneous coronary intervention (PCI) with 2.5\*23 mm Sirolimus drug eluting stent DES (Cypher) in the mid portion of the right coronary artery (RCA) for a new onset angina. The coronary angiography showed in stent entry restenosis. The lesion was predilated and we deployed an overlapping 2.5\*28mm Sirolimus DES (Cypher). Additional high-pressure postdilatation at 20 atm was performed. During close clinical observation in the intensive care unit twenty four hour after the procedure, the patient had a syncope and seizure due to ventricular fibrillation stopped with an electrical shock. EKG revealed an ST segment elevation in the inferior leads. Blood test revealed elevated levels of Creatine phosphokinase (CPK) (850UI/l, normal level <176UI/l). Intravenous bolus of Abciximab (Reopro) bolus was administered. EKG abnormalities returned to baseline.

The angiographic control didn't show any thrombosis but a complete linear transverse fracture of the previously inserted stent (Figure 1).



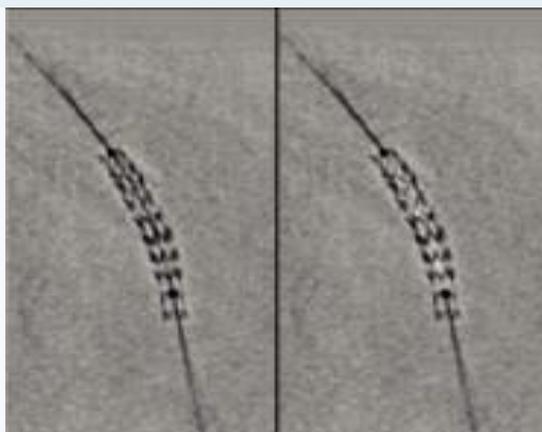
**Figure 1:** a complete linear transverse fracture of the stent in the proximal right coronary artery.

The fracture site was close to the stent overlap, at the distal edge of the first proximal stent; hinge motion was noted during the cardiac cycle. We decided to cover the stent fracture with a 2.5\*13mm Paclitaxel DES (Taxus). The patient suffered no adverse sequelae and remains well at follow-up.

### Case 2

A 78-year-old man was admitted with a history of class 3 Canadian cardiovascular society CCS grading of angina seven year after an angioplasty of mid left anterior descending artery with a 2,5x18mm Sirolimus drug eluting stent (Cypher) for unstable angina. Coronary angiography showed focal in-stent restenosis. Balloon angioplasty was planned as initial strategy motivated by the focal pattern of restenosis in a small diameter stent with thick struts.

During this procedure, we performed a Digital image enhancement revealing a stent fracture with two fractured segments still maintained contact without definite displacement (Figure 2). This stent fracture was not evident on angiography. So, we opted to deploy a 2.75\*20mm DES Zotarolimus DES in the fracture site with a satisfying final result after a postdilatation on angiography and digital image enhancement.



**Figure 2:** stent fracture, multiple struts fracture with acquired transection and gap in the stent body

### Case 3

A 50 year old male smoker, dyslipidemic with a past long history of coronary artery disease:an acute anterior myocardial infarction in November 2004 for which he had undergone an angioplasty of the mid portion of the left descending artery with bare metal stent Microdriver 2.5\*12 inflated at 12 atm after a predilatation. After 6 months restenosis occurred. It was a complex bifurcation lesion that required the deployment of drug eluting stent Cypher 2.5\*23 with a kissing balloon using two 2.25\*15

mm balloons. He was admitted with recurrent angina with an angiographic aspect of irregularity in the stent entrance and a doubt about a stent fracture near the bifurcation which was not easy to distinguish from the stent distortion induced by the kissing balloon during the last procedure. We performed an intravascular ultrasound imaging which showed image slices with no visible stent struts a complete stent separation in two pieces. We measured external elastic membrane (EEM) and lumen cross-sectional area (CSA) at the reference segments, at the minimum lumen site, and at stent fracture sites. We ended the procedure by the deployment of two overlapping stents Promus 2.5\*38 and onyx 3\*15 covering the entrance of the Cypher. Patient was discharged after 24 hours.

## DISCUSSION

In the era of drug-eluting stents (DES), Stent fracture (SF) has been implicated as a cause of stent failure. A SF is defined as the complete or incomplete separation of the stent strut by a fluoroscopic image and/or the absence of a stent strut on at least one slice of IVUS image. We classified SF into the following four types: type I, a single strut fracture only; type II, multiple strut fractures at different sites; type III, complete transverse SF without displacement of fractured fragments more than 1mm during the cardiac cycle; type IV, complete transverse linear type III fracture with stent displacement (1,2).

Coronary SF was first reported in 2002 after a BMS implantation in a venous bypass graft (3).

The first case of coronary DES fracture appeared in 2004 (4).

The reported incidence of SF varies widely between different studies. It ranges between 1% and 8% (5) and is most likely underestimated due to different reasons.

Patients with SF might be asymptomatic, particularly in case of minor fractures, and therefore, if the angiographic or any other imaging modality follow-up were clinically driven, then many cases of SF were not reported.

SFs can be hard to diagnose by angiography alone. Patients might be treated as stent thrombosis or stenosis without the detection of SF, especially if other more sensitive diagnostic imaging modalities were not used. Several patients might present with sudden cardiac death before diagnosis. Stent fracture is found in 29% of the stented lesions at autopsy, which is higher than clinically reported (6).

Despite stent fracture is commonly associated with recurrent angina related to in stent restenosis, it is characterized by a clinical polymorphism. It could be behind different scenarios as it described in our 3 cases. In the first case, the patient presented an acute

myocardial infarction and serious cardiac arrhythmia. Sudden death would occur unless electrical shock was delivered. In the second and the third case, patients were admitted with non ST elevation myocardial infarction or unstable angina.

In stent restenosis associated with SF is due the lower amount of drug dispensed in the fracture area and to greater mechanical aggression from the fractured struts, as both of these factors cause smooth muscle cell proliferation and abnormal endothelialization (7,8). On the other hand, MI was related to the exposure of a free metal strut protruding into the vessel lumen which could trigger platelet activation and resultant stent thrombosis. SF could involve partial or complete breakage of the stent; the latter event may cause immediate flow obstruction, thrombosis, and myocardial infarction (9).

Some studies have shown that certain Sirolimus-eluting DES (Cypher) cause a greater number of SF their closed-cell design and the use of stainless steel, a material of lower flexibility and conformability than new cobalt, chromium, or platinum alloys.

Also procedural factors should be considered as the implantation of long stent with a larger balloon at high pressure or overlapping site of stents, especially when it was placed in tortuous vessels or calcified lesions (10) and anatomical factors, RCA is more dynamic than other epicardial vessels, meaning that more flexion points exist in certain segments during the cardiac cycle. A stent in this location can be subjected to repetitive distorting forces, which can cause a fracture resulting from mechanical fatigue.

The coronary imaging progress has transformed the approach regarding SF and permit to push forward investigation to confirm or revise some findings and to understand the mechanisms. With conventional fluoroscopy, SF can go unnoticed if not radiologically obvious. We were limited by the stent visibility which underestimates the incidence of SF.

In several studies the SF was limited to type III/IV. This fact shows that the identification of SF was relatively difficult in the situation of type I/II (1, 2).

Stent boost improves stent visualization with the balloon left in place for motion compensation of the acquired images. As in our second case, SF was recognized after the Digital image enhancement.

In the third we were not able to confirm SF even after Stent boost. We performed an intravascular ultrasound Imaging IVUS. At times, it may be difficult to differentiate between incomplete SF and stent deformation (pronounced curves, postdilation of lateral branches, calcified lesions), which causes strut separation and asymmetry, but it is not difficult to detect the absence of struts. If the fracture is complete, total disappearance of struts is observed in various consecutive images

IVUS is helpful to identify stent fracture as a cause of stent failure and to understand possible mechanisms of stent fracture such as aneurysm formation.

The most common intravascular findings are the absence of struts in a broad area of the vessel circumference and the presence of abundant hyperplasia in the same segment (11).

Intravascular ultrasound (IVUS) may be more likely to identify mechanisms of stent failure—including stent fracture—compared with angiography by providing information regarding neointima formation, vessel remodeling, perivascular tissue, stent expansion, stent strut distribution, and malapposition.

Based on the IVUS findings, 2 patterns of stent fracture according to possible mechanisms were suggested. Type I stent fracture does not occur in association with a coronary aneurysm, but presents early, can be partial or complete, appears to be related to stent length and direct mechanical stress due to vessel hinge movement during cardiac contraction, and is frequency adjacent to calcium. Type II stent fracture presents late, is usually complete, and occurs in association with aneurysm formation and late-acquired stent malapposition (12). Although IVUS remains the current gold standard for detailed assessment of results of stent implantation, the resolution of IVUS is only approximately 150  $\mu$ m, and the echoes frequently cause artifacts; intravascular optical coherence tomography (with 10 times the resolution of IVUS and fewer artifacts) may be a superior technique to assess stent struts (13).

Regarding the major cardiovascular events related to stent fracture treatment regain a crucial importance

especially with the discrepancy between clinical presentation and SF type and in the absence of a clear consensus. Not all SF are associated with clinical sequelae. Management of the fracture should thus be individualized, according to the presence of ischemia induced by restenosis, the type of stent fracture, and the presence of factors that predict possible recurrence. In some cases, the degree of fracture is low enough to warrant a more conservative approach of follow-up without further treatment.

In others as in our 3 cases, the stent fracture is identified and successfully treated by repeat stenting with a different type of stent with a higher flexibility.

We underline the importance of the dual antiplatelet therapy. Except for patients with poor medication compliance, there was no patient with stent thrombosis. So we could carefully suggest that if SF patients were continued on medication with dual or triple antiplatelet agents, there would be a very low rate of major adverse cardiac events (post-detection of stent fracture) including stent thrombosis associated with SF. But continued medication with combination antiplatelet agents is very hard during whole life in the real-world. The appropriate duration of combination antiplatelet therapy is needed following SF.

## CONCLUSION

We recommend careful angiography review and/or IVUS at the ISR of DES.

Physicians should consider all risk factors for stent fracture during angioplasty to minimize the risk of further complications.

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