Case Report

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Intrastent Coronary Pseudoaneurysm in the Setting of Primary Angioplasty: An Early Presentation

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Abstract

Pseudoaneurysm formation is an uncommon but dreaded complication of percutaneous coronary intervention (PCI). Herein, we describe the case of a patient who developed symptomatic pseudoaneurysm within a month of primary PCI using a zotarolimus eluting stent (ZES). Despite a review of the literature and an examination of the procedural data, we were not able to explain the definite mechanism of pseudoaneurysm formation in our case. We assume that there was stent strut malapposition leading to non-angiographically visible dissection that provoked this phenomenon.

Keywords: Coronary pseudoaneurysm; Primary angioplasty; Stent malapposition; Zotarolimus eluting stent

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Introduction

The prevalence of coronary artery pseudoaneurysms is 0.2-10% in patients undergoing angiography [1]. Besides atherosclerosis, there are various etiologies for coronary artery pseudoaneurysms such as coronary artery dissection [2], trauma, infection and congenital malformations [3]. Pseudoaneurysms may represent a risk for serious adverse outcomes since they can lead to complications such as thrombosis with distal embolization, rupture, cardiac tamponade and myocardial infarction. DES can pose an inherent danger for the development of pseudoaneurysm in that it delays or prevents vascular healing by causing inhibition of neointimal hyperplasia and inducing a hypersensitivity reaction at the site of contact with the coronary endothelium. However, drug hypersensitivity to zotarolimus leading to formation of coronary pseudoaneurysm has not yet been reported.

Case History

We present the case of a 70 year old diabetic, hypertensive lady presenting with complaints of recurrent rest angina since past 1 week. Clinical examination was unremarkable except for bilateral minimal basal crepitations. ECG revealed sinus bradycardia, heart rate of 56 bpm, left ventricular hypertrophy and ST-T changes suggestive of evolved inferior wall MI. 2D echocardiography showed nondilated left ventricle, mild posterobasal and inferoseptal hypokinesia with preserved wall thickness and ejection fraction of 45%. Patient was taken up for coronary angiography and adhoc PCI. Left coronary angiogram revealed significant distal left main and ostial LAD disease along with long segment mid LAD and proximal non-dominant left circumflex artery stenosis (**Figures 1A-1C**). Right coronary angiogram showed proximal total thrombotic occlusion (**Figure 1D**). In view of ongoing ischemia and intra-procedural unstable junctional rhythm, PCI of culprit RCA lesion was done after thrombus aspiration and intravenous abciximab administration with DES 3 × 38 mm (RESOLUTE INTEGRITY™, Medtronic, Minneapolis, MN, USA) (**Figure 1E and 1F**) without any stent post-dilatation using non-compliant balloon. Post procedure TIMI III flow was achieved (**Figure 2A and D2**), sinus rhythm was restored, angina resolved and patient was subsequently discharged after 48 hrs of hospitalization on optimal medical therapy including dual antiplatelet drugs (aspirin and ticagrelor). After 2 weeks patient again sought medical help for resurgence of chest pain. ECG and ECHO did not show any evidence of new ST-T changes or regional wall motion abnormality respectively. Check angiogram was planned as symptoms persisted despite stepping up of anti-anginal medications which revealed similar left coronary

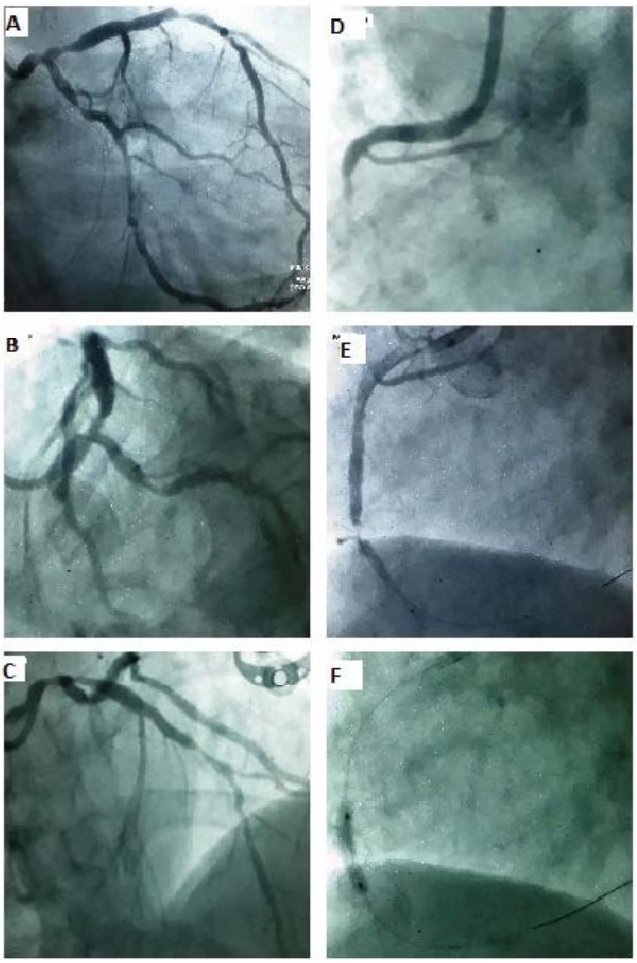

Figure 1 Left coronary angiogram showing significant stenosis in distal left main, proximal and mid LAD and proximal LCX in AP caudal (A), LAO caudal (B) and AP cranial (C) views respectively. Panel D shows proximal RCA total occlusion followed by subsequent wiring (E), thrombus aspiration and ballooning (F) of mid RCA critical lesion abutting a small RV branch during primary angioplasty.

anatomy as previous but right coronary angiogram showed a large ovoid coronary outpouching in mid RCA through stent struts at the site of origin of a small RV branch suggestive of intrastent coronary pseudoaneurysm (**Figure 2B and 2E**). Magnified view of fluoroscopic images revealed a focal area of stent under expansion just proximal to the site of outpouching (**Figure 2C and 2F**). On reviewing the index procedure data, a focal site of stent under-expansion was found that coincided exactly with the origin of pseudoaneurysm (**Figure 3**). Surprisingly, though distal coronary flow remained TIMI III grade. A provisional diagnosis of intrastent coronary pseudoaneurysm secondary to stent malapposition due to mechanical periprocedural factors was kept and patient underwent CABG. Intraoperative findings revealed 10 × 4 mm thin walled pseudoaneurysm arising within stent struts with surrounding friable myocardium and inflamed pericardium devoid of gross pus collection. Post CABG patient developed septicemic shock and succumbed 48 hours late.

Discussion

Coronary artery aneurysm (CAA) is defined as a dilation of the

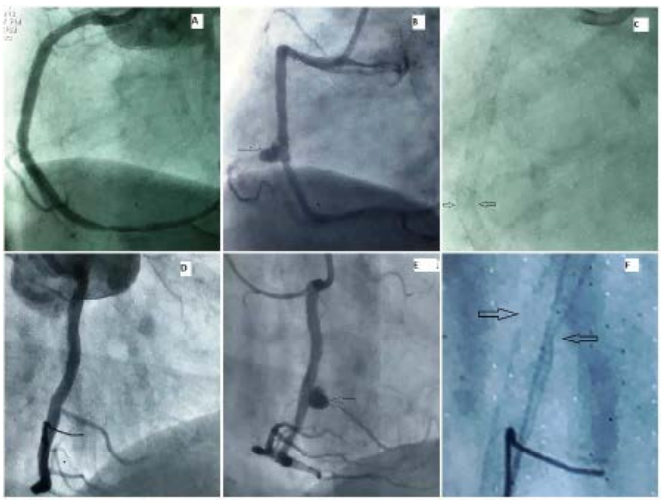

Figure 2 LAO views showing post angioplasty RCA with TIMI III flow (A), a pseudoaneurysm (hollow arrows) in mid RCA at the origin of RV branch with TIMI III flow 2 weeks after the index procedure (B) and sites of possible stent under-expansion (hollow arrows) resulting in formation of pseudoaneurysm (C). Similar findings are shown in RAO views (D, E, F).

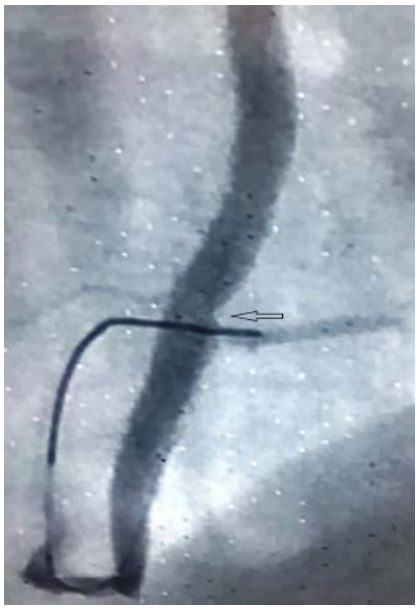

Figure 3 Enlarged RAO view showing a significant 'indentation' (hollow arrows) resulting from stent malapposition in mid RCA after primary angioplasty. This malapposition might have been the triggering factor for the development of pseudoaneurysm later on.

coronary artery that exceeds 1.5 times the reference diameter of the adjacent coronary segments that are angiographically normal. Post PCI (including coronary angioplasty, directed coronary atherectomy, and laser angioplasty), the incidence of pseudoaneurysm is 1.5-5%1. Occurrence of pseudoaneurysms in the right coronary artery, left anterior descending branch, left circumflex branch, and left main trunk is 40.4%, 32.3%, 23.4%, and 3.5%, respectively [1]. Pseudoaneurysm formation is more commonly a result of traumatic rupture or deep-vessel injury

of an artery. Post PCI pseudoaneurysm formation is known to occur as early as 2 months of procedure [4]. Causes of vascular injury include anatomical anomalies in the coronary artery, atherosclerosis, rapidly injected contrast agent, catheter type (it is significantly more frequent with left Amplatz guiding catheters), improper catheter placement and deep engagement during balloon drawing. Positive regional vascular remodeling, plaque regression, atheroma burden, stent malapposition, stent edge dissection, late dissolution of the thrombotic material captured by the stent struts, cell necrosis, apoptosis and spontaneous dissection that is not visible through the angiography are various other mechanisms responsible for formation of pseudoaneurysm. In the STRESS [5] study, Palmaz-Schatz stent was associated with presence of a coronary artery pseudoaneurysm in 3.9% patients that increased up to 32% if corticoid drugs and colchicine were concomitantly administered after the implant, probably due to incomplete stent strut apposition resulting from use of anti-inflammatory agents in late post-implantation phase. Role of DES in the formation of pseudoaneurysms remains controversial. Incidence of pseudoaneurysm formation after DES implantation is low [6] (0.2-2.3%) within first 9 months of implantation- a rate similar to that reported after BMS (0.3-3.9%). Studies suggest patient-specific sensitivity to rapamycin (sirolimus [7,8] as a cause of aneurysmal dilation and incomplete apposition. Paclitaxel-eluting stents [9] contribute similarly to the late positive remodeling (re-endothelization) of aneurysmal vascular walls by inhibiting cell proliferation. The polymer of drug eluting stent in which drug is embedded may cause hypersensitivity reaction, vasculitis and incur the risk of eosinophilic or heterophilic infiltrates that can erode the vessel wall and weaken the wall resulting in subsequent dilatation of the segment of coronary artery. The risk of rupture increases in patients more than 40 years of age, presence of angina pectoris, congestive heart failure, infective endocarditis or distal embolization. Treatments for coronary-artery pseudoaneurysm in¬clude drug therapy (e.g., antiplatelet therapy and anticoagulant therapy), PCI (covered stenting [10] or coil embolization [4]) or surgical treatment (coronary artery bypass surgery [11] and ligation). Drug therapy (beta-blockers and anticoagulation to prevent progressive dissection and superimposed thrombus formation) is usually reserved for patients which are asymptomatic or without ST-T changes, troponin elevation, angiographic stenosis, and have TIMI [3] grade distal coronary flow. In our patient, although the use of intravascular imaging (OCT/IVUS) would have helped in delineating the exact etiology of pseudoaneurysm, yet probably suboptimal stent deployment (malapposition/ stent strut fracture) with angiographically invisible coronary dissection can be attributed to the formation of pseudoaneurysm, in addition to differences in the kinetics of drug elution and inflammation.

Conclusion

In our patient, we hypothesize that a non-angiographically visible dissection along with stent malapposition/ stent strut fracture could have provoked this phenomenon. Intravascular imaging should be employed frequently to identify the underlying mechanisms and guide probable treatment options for postinterventional coronary pseudoaneurysms. This uncommon but life-threatening coronary pathology must be addressed promptly and judiciously with appropriate treatment modality, depending on the underlying etiology to avoid complications and reduce mortality.

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