Open Journal of
Clinical & Medical
Case Reports

Volume 2 (2016)
Issue 6

ISSN 2379-1039

Acute Coronary Spasm with Ventricular Fibrillation following Partial Reabsorption of a Bioresorbable Vascular Scaffold

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Abstract

The introduction of Bioresorbable Vascular Scaffolds (BVS) represents the most important novelty in coronary intervention. The relation between BVS and coronary spasm, a potential fatal condition in the subset of endothelial dysfunction, is poorly understood yet. Nowadays no correlation with bioresorbable scaffolds is demonstrated yet. We describe a case of a young man who experimented an ECG documented acute coronary spasm leading to ventricular fibrillation after 2 years from the BVS' implantation.

Keywords

Bioresorbable vascular scaffold; Coronary spasm; Ventricular fibrillation; Intravascular ultrasound

Introduction

Bioresorbable Vascular Scaffolds (BVS) with their potential role of "vascular reparative therapy" represent the last innovation in coronary intervention [1]. We present a case of acute coronary spasm leading to ventricular fibrillation occurred in a vessel previously treated with BVS, which partially reabsorbed after two years from the implantation.

Case Presentation

A 41 year-old man was referred to our hospital complaining the sudden onset in the first morning, at rest, of acute chest pain, lasting 10 minutes and regressing spontaneously. Cardiovascular risk factors included hypertension and smoking status. His past medical history was significant for previous (2 years ago) unstable angina due to severe stenosis of proximal LAD [Fig.1A], the extent of which was not modified by intracoronary nitroglycerin. Therefore he was treated with percutaneous coronary intervention (PCI) and implantation of a 3.5 x 18 mm Bioresorbable Vascular Scaffold (BVS) [ABSORB RX - Abbott Vascular Santa Clara, CA, USA] [Fig.1B]. ECG in stable condition, after BVS implantation, is showed in Fig. 2A. Patient's chest pain was reported the same as that previously experienced and was responsive to sublingual nitroglycerin. ECG did not show any ventricular repolarization abnormalities, and cardiac enzymes were normal. He was on therapy with: acetylsalicylic acid (100 mg), Irbesartan (150 mg) and atorvastatin (40 mg). The multi-projection angiogram, performed the following day, demonstrated a moderate stenosis of proximal LAD, in the same site of previous BVS implantation [Fig. 1C]. Due to the patient's stable condition no immediate invasive investigation was carried out, postponing a further evaluation with functional test or intravascular ultrasound (IVUS) to the common

judgment of the heart team meeting. During the following day the patient complained another episode of chest pain at rest, resolved after administration of sublingual nitroglycerin; so heparin continuous i.v. infusion was started and the patient was transferred to the intensive care unit. At 05:08 a.m. there was new onset of resting angina episode, with ST anterior elevation in the superficial ECG [Fig.2B] and following ventricular fibrillation [Fig.2C] treated with external defibrillation. The successive coronary evaluation with IVUS revealed a partial reabsorption of BVS in the first tract of LAD with a minimal lumen area (MLA) of 3.1 mm^2 [Fig.3]. PCI was then performed with implantation of a 3.5 x 25 mm DES [Create (CID, Saluggia, Italy)] with optimal final result [Fig. 1D] and concomitant therapy with calcium channel blockers was initiated. No further episodes of angina occurred in the following three days and the patient was discharged in good clinical conditions with the following medical therapy: acetylsalicylic acid (100 mg), Ticagrelor (90 mg) bid, Amlodipine (10 mg), Atorvastatin (80 mg), Pantoprazole (20 mg). An ambulatory re-evaluation, performed eight months later, revealed no further angina episodes, and good clinical status (NYHA I, CCS 0).

Discussion

Coronary spasm is caused by local hyper-activity, which could be triggered by spontaneous or provocative stimulation, including hyperventilation, vasovagal reaction, drugs and mechanical stimulus [1, 2]. Focal coronary artery spasm due to coronary artery smooth muscle hyperactivity has been well described following delivery of a drug-eluting stent [3]. Endothelium dysfunction which can be caused or aggravated by DES implantation [4, 5] is involved in this process, since the vessel has been permanently caged by a metallic prosthesis [6]. To overcome this phenomenon, bioresorbable vascular scaffolds have been developed with the aim to provide a provisional vessel scaffolding, which is subsequently bioresorbed, allowing the treated coronary segment to recover a response to shear stress and other pharmacological and physiological stimuli [6, 7]. Contrary to that which happens with DES, coronary segments treated with bioresorbable scaffolds are only transiently scaffolded and able to restore vasomotion [8] as demonstrated at 12 and 24 months in ABSORB Cohort B and ABSORB Cohort A trials, respectively [6, 9]. As the mechanical framework of the ABSORB BVS is aimed *a priori* to dissolve within 2 years, the scaffold's resistance to vasomotion induced by vasoactive drugs decreases over time, thereby permitting the potential recovery of normal vasomotor tone. Provocation testing using intracoronary acetylcholine is safe and effective in demonstrating coronary artery vasospasm [10]. Long-acting calcium channel blockers have been shown to decrease the number of anginal attacks and are considered the mainstay of treatment [11]. In this case we used a BVS in a young man to treat a focal atherosclerotic plaque of proximal LAD with the intent to avoid a permanent scaffolding of the vessel, reducing the risk of late and very late stent thrombosis. Ergonovine test was not performed at the time of first PCI, since the severity of the atherosclerotic stenosis was considered significant both at baseline and after tests with vasodilator. The angiographic and IVUS control performed after 2 years from the index procedure, demonstrated a partial BVS reabsorption, sufficient to permit a complete restoring of vessel vasomotion, lacking the caging effect of the scaffold. Due to the clinical evidence of variant angina (young smoker patient, presenting typical resting angina in the first morning associated with ECG evidence of ST elevation in the absence of significant coronary obstruction) and subsequent ventricular fibrillation, we did not perform any provocation intracoronary testing. Nowadays there is a poor experience in stenting for coronary spasm and little is known about long term prognosis. We don't know "*a posteriori*" if stenting

with DES could have been avoided by initial medical treatment with calcium channel blockers. Up to date, literature's findings, coming from previous studies and reports, highlighted the role of coronary stenting as an adjuvant and/or alternative therapy to drugs in patients with coronary spasm and severe electrical and/or hemodynamic instability [12, 13]. Therefore, due to the acute and severe presentation of the cardiac event in our patient with a life-threatening arrhythmia, we decided to implant immediately a DES inside the BVS' partial residual even in the absence of significant atherosclerotic stenosis, postponing the usual medical treatment with calcium channel blockers. We submit that a combination of the restored vasomotion with the endothelial dysfunction (typical of the injured vessel), could have been the common thread leading to life-threatening coronary vasospasm experienced by the patient. Further studies are necessary to investigate the relationship between BVS absorption and coronary spasm, aiming to a safe use of this revolutionary device.

Figures

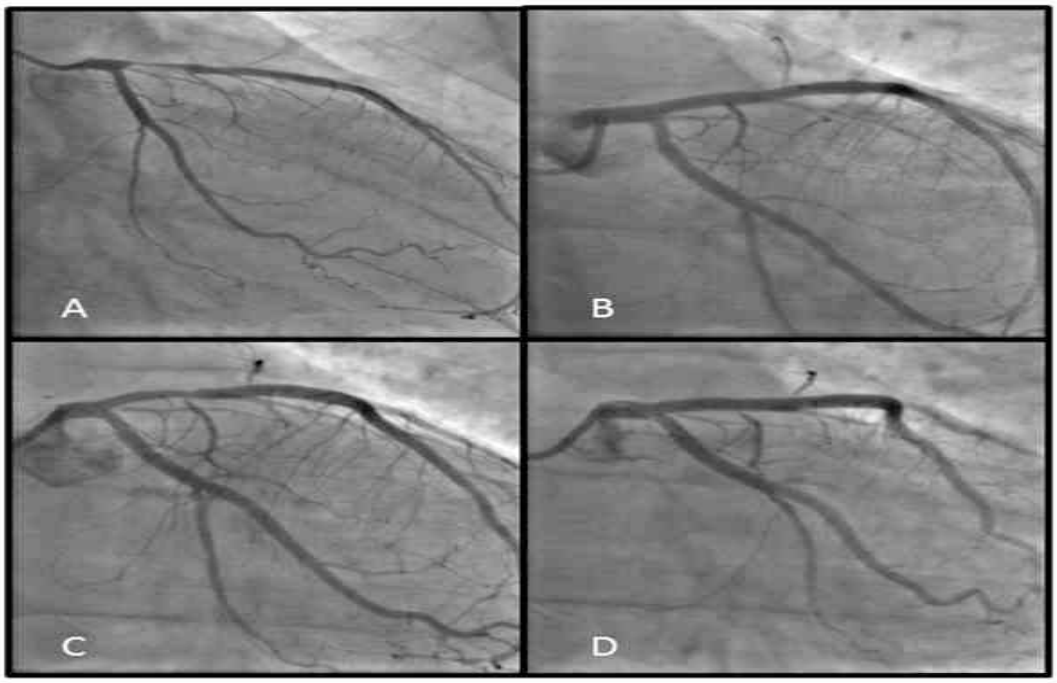

Figure 1A. (RAO 30°, CAU 21°): severe stenosis of proximal LAD
Figure 1B. (RAO 30°, CAU 21°): result after BVS implantation
Figure 1C. (RAO 30°, CAU 21°): moderate stenosis of proximal LAD, in the same site of previous BVS implantation
Figure 1D. (RAO 30°, CAU 21°): Final result after DES implantation

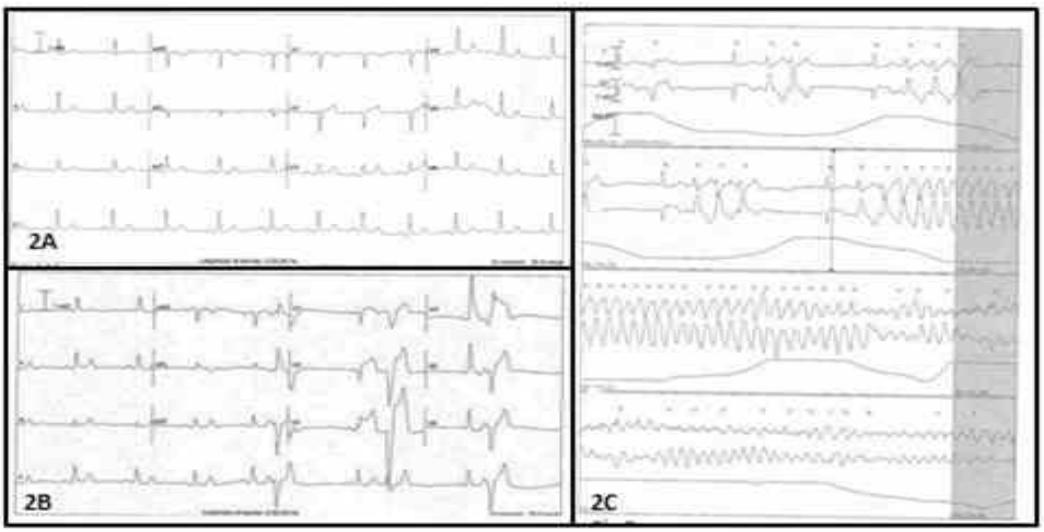

Figure 2A. ECG in stable condition, after BVS implantation
Figure 2B. Superficial ECG showing ST anterior elevation
Figure 2C. Continuous ECG monitoring showing ventricular fibrillation

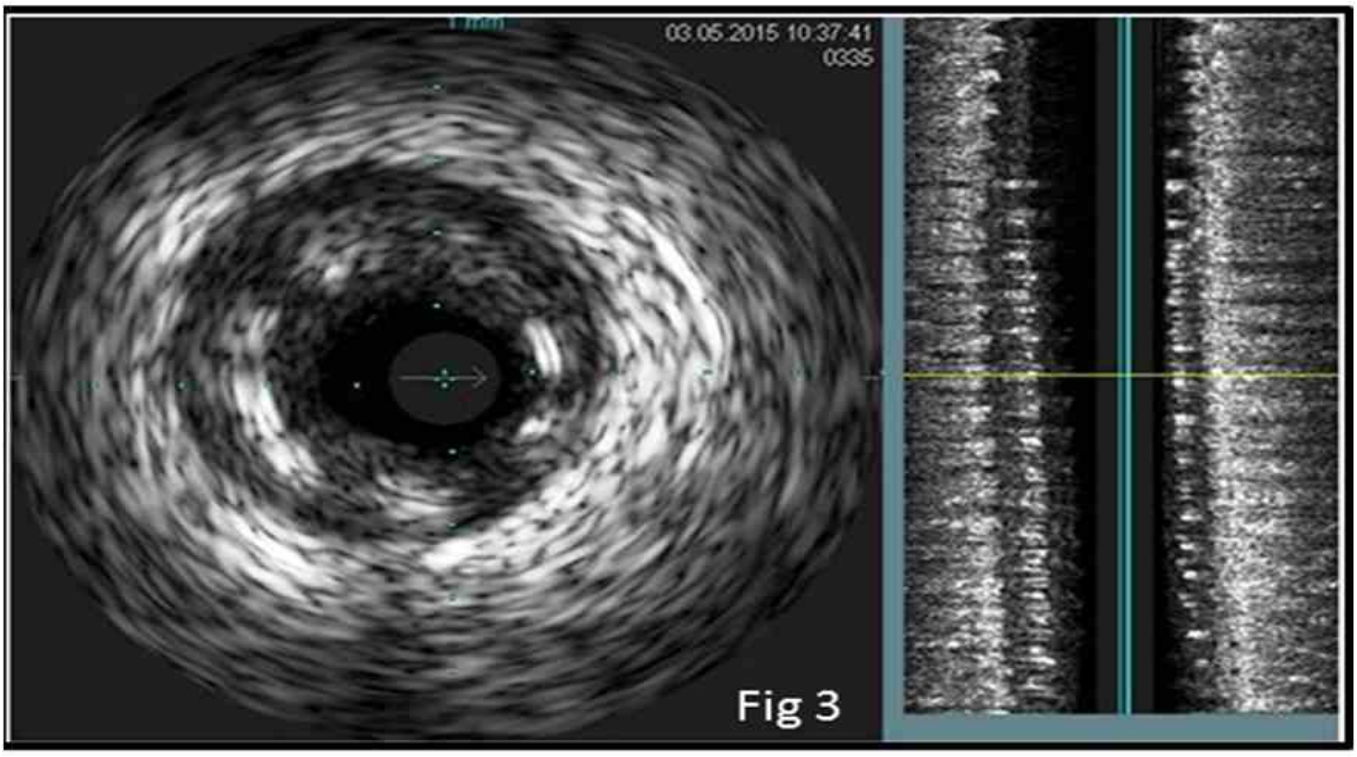

Figure 3: IVUS imaging of proximal LAD showing partial BVS reabsorption

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Manuscript Information: Received: December 15, 2015; Accepted: March 17, 2016; Published: March 21, 2016

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Citation: Arrivi A, Lazzari L, Bazzucchi M, Bock C, Milici C, Placanica A et al. Acute coronary spasm with ventricular fibrillation following partial reabsorption of a bioresorbable vascular scaffold. Open J Clin Med Case Rep. 2016; 1091

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