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Case Report

A Case of Giant Right Coronary Artery Aneurysm after DES Implantation

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Key Words :
Coronary artery aneurysm, PCI.

Abstract:

Coronary artery aneurysms (CAAs) are not very uncommon but giant coronary artery aneurysms are rare with a reported prevalence of 0.02% to 0.2%. Recently, with the advent of implantation of drug-eluting stents, there are increasing reports suggesting stents causing coronary aneurysms, months or years after the procedure. Though most of the CAAs are asymptomatic, they may present with various clinical presentations like angina pectoris, fistula formation, pericardial tamponade, compression of surrounding structures, congestive cardiac failure or sudden death. The natural history and prognosis remain unclear. Despite the important anatomical abnormality of the coronary artery, the treatment options of CAAs are still poorly defined and present a therapeutic challenge. We report a case of iatrogenic giant CAA following percutaneous transluminal coronary angioplasty (PTCA) with its relevant management strategy.

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Introduction:

Introduction of Drug-Eluting Stent (DES) proved to be an important step forward in reducing rates of restenosis and target lesion revascularization after percutaneous coronary intervention, but at the same time potentially causing serious complications. One of the rare complications of DES is coronary artery aneurysm with a reported incidence of 0.3% to 6.0%, and most "aneurysms" are in fact pseudo aneurysms rather than true aneurysms.[1,2] Residual dissection and deep arterial wall injury (rupture or resection of the vessel media) caused by oversized balloons or stents, high-pressure balloon inflations, atherectomy, and laser angioplasty have all been associated with coronary artery aneurysms after coronary intervention.[3] Drug-eluting stents (DES), which locally elute anti-proliferative drugs, can dramatically inhibit neointimal growth, thereby suppressing restenosis,[4,5] but at the same time potentially causing coronary aneurysms due to other mechanisms, such as delayed re-endothelialization, inflammatory changes of the medial wall, and hypersensitivity reactions.[6,7] These findings may be due to delayed healing secondary to the anti-proliferative action of the eluted drug, cell necrosis and/or apoptosis from the antimetabolite effect of the drug, and hypersensitivity reactions to the drug/polymer mixture on the DES.[6,8] However, the true incidence, clinical course, and treatment of coronary artery aneurysms after DES implantation remain largely unknown. Treatment of CAA is somewhat controversial, and there is no consensus on the modality of treatment in a given clinical situation. We report a case of giant CAA after DES implantation presenting with fever, which was treated by surgery.

Case report:

A 35 year old diabetic, hypertensive male was admitted in SSMC & Mitford hospital with high grade fever associated with chill and rigor. Patient underwent coronary angiogram and angioplasty of RCA following acute inferior wall Myocardial infarction (AMI) four months back. Documents related to angiogram and angioplasty was not available. On query he gave history of admission for a febrile episode of 10 days after PCI and was hospitalized and treated with parenteral

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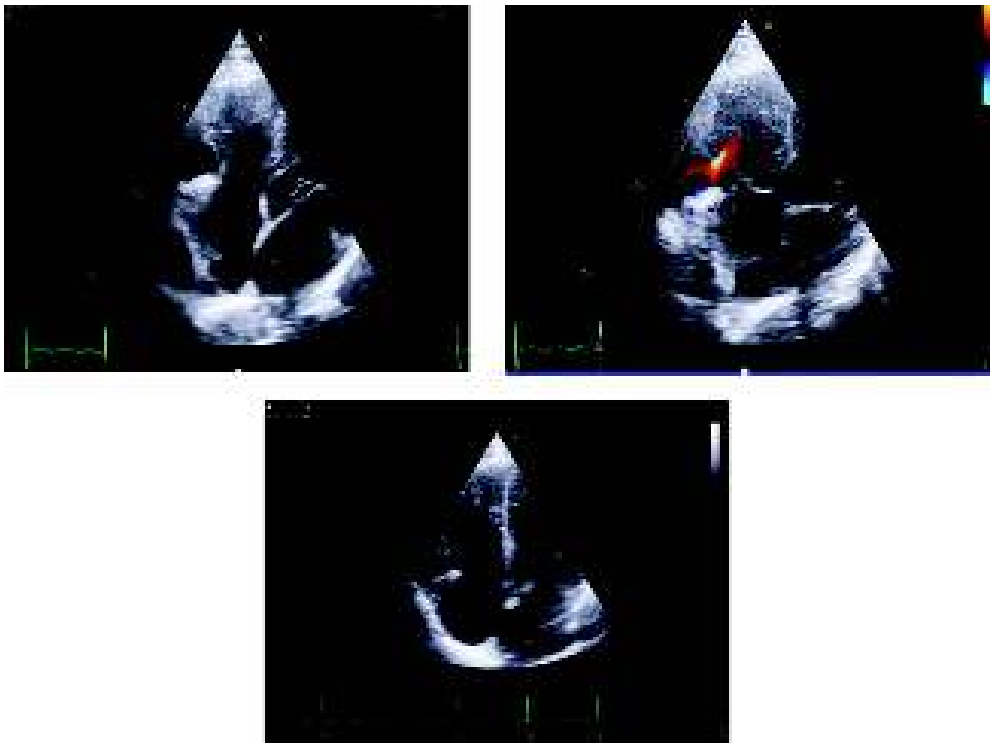
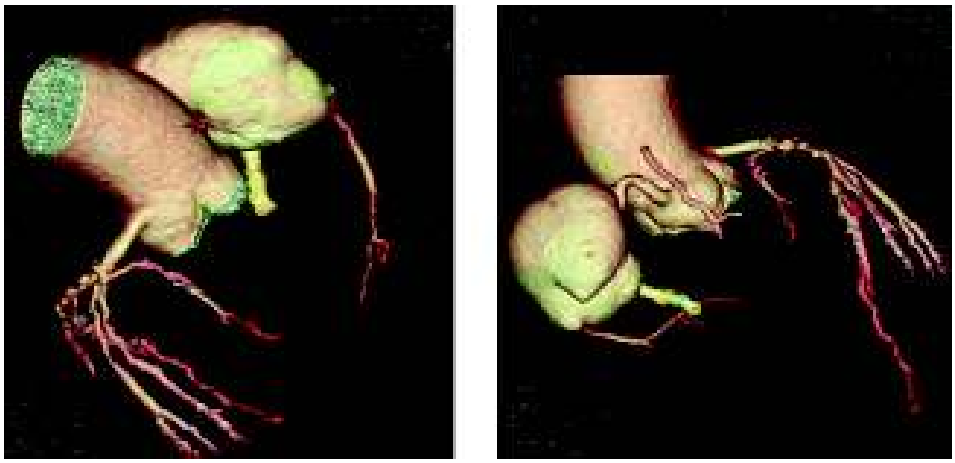
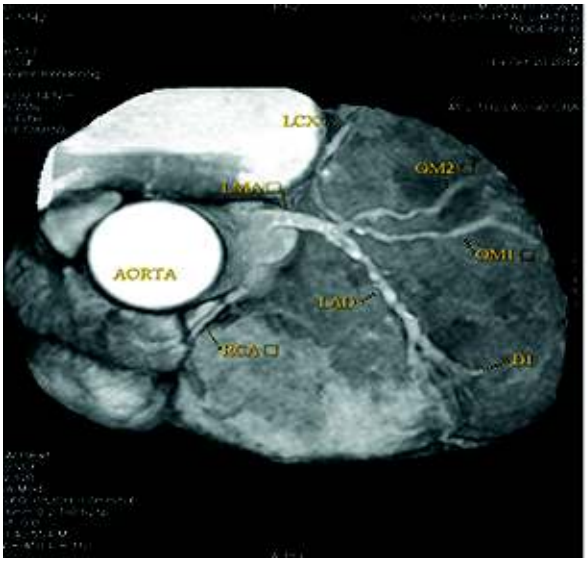
antibiotics. At that time he was diagnosed as pneumonia. According to statement of patient he was discharged home with afebrile status.

One month after that episode again he developed high grade fever and admitted in our cardiology department without any past document.

Investigations revealed neutrophilic leukocytosis with high ESR and CRP. Chest X-ray was normal. Echo reveals there is an aneurysmal dilatation of RCA with a metallic shadow within the aneurysm and it communicates with RV. Inferior wall hypokinesia. All the valves are normal in morphology without any vegetation. Fair LV systolic function. Blood culture revealed Pseudomonas aeruginosa growth and he was treated with sensitive parenteral antibiotics. Patient respond well and fever subsided by 5 days. Parenteral antibiotics was continued for 4 weeks. In the meantime CT angiogram was done. CT angiogram reveals large aneurysm (4cm×3.5cm) in RCA, stent in the mid RCA seen through the aneurysm with 40-50% stenosis in RCA proximal to aneurysm. After controlling infection CAG was done, that reveals large aneurysm in the stented segment of RCA.RCA occluded distal to aneurysm.



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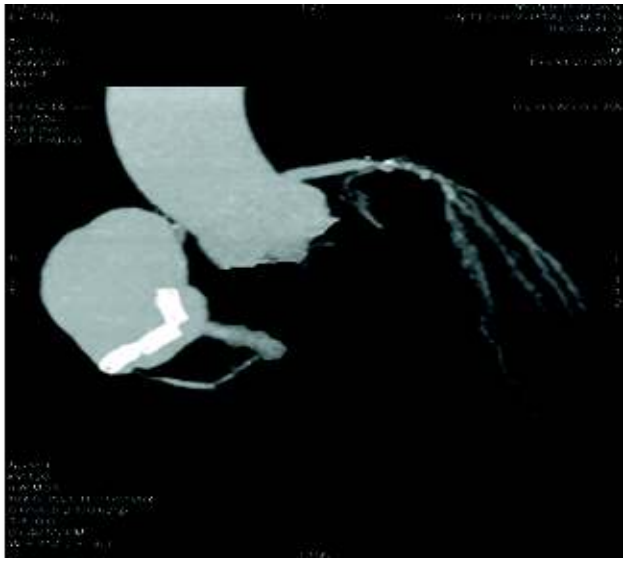

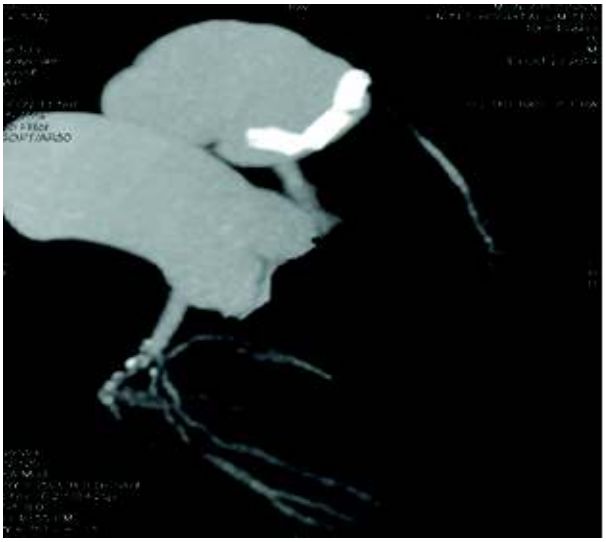

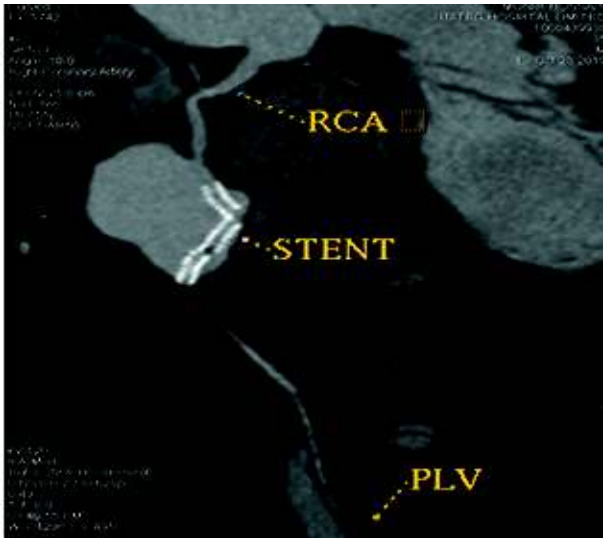
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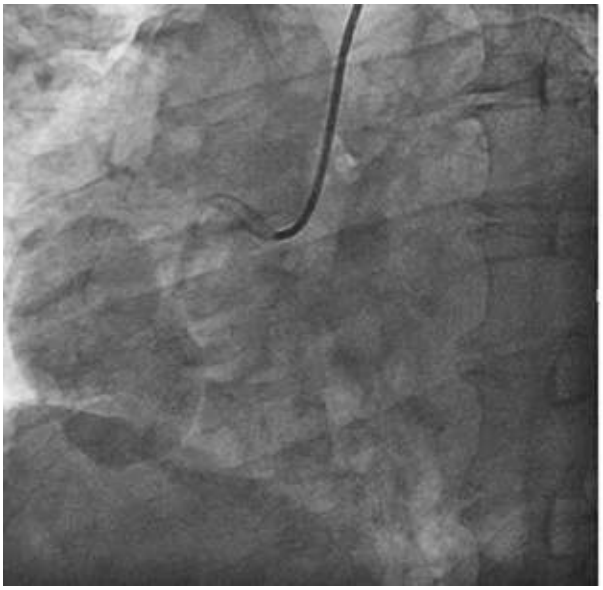

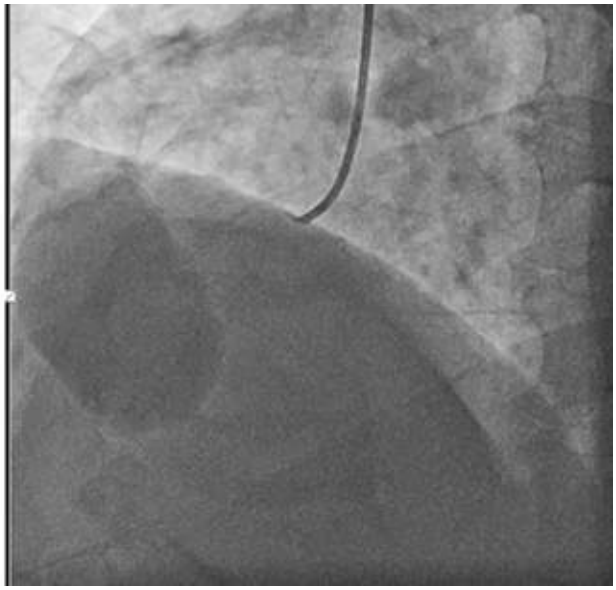

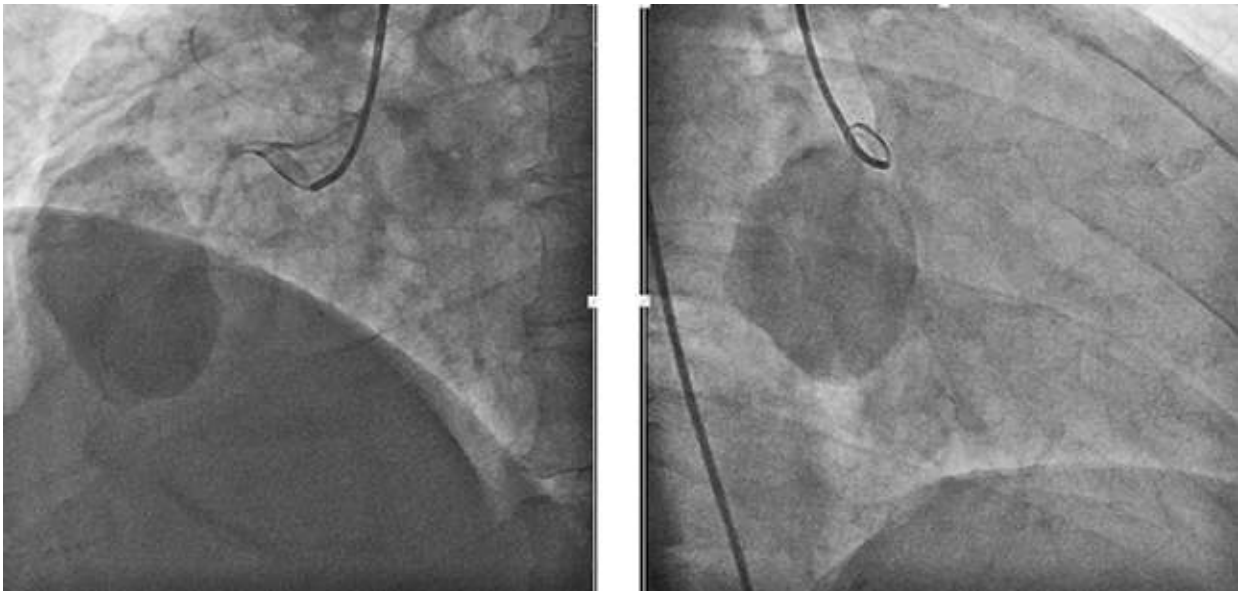

Rest of coronary arteries are normal with minor plaques in LAD. Management was discussed with surgeon and decided to do CABG and resection of aneurysm. The patient performed better after complete revascularization

Discussion:

Coronary artery aneurysm (CAA) is an uncommon condition and is defined as dilatation of the coronary artery exceeding 50% of the reference vessel diameter.[9] CAAs are termed giant if their diameter transcends the reference vessel diameter by greater than four times or if they are >8 mm in diameter.[10] It occurs in about 0.3%–4.9% of patients undergoing coronary angiography [9] and are reportedly present in 1.4% of postmortem examinations.[9]

CAAs are found in the epicardial coronary arteries, most frequently in the proximal and middle segments of the right coronary artery (RCA) (68%) adjacent to the right atrium, indicating that the adjacent area of atrial tissue is weak that helps in the formation and enlargement of aneurysm, followed by the proximal left anterior descending (LAD) (60%) and left circumflex (LCx) (50%). CAA of the left main stem (LMS) is exceedingly rare and occurs in only 0.1% of the population.[9,11]

The difference between ectasia and aneurysm are often subtle and mainly semantic. If the length of the dilated segment is more than 50% of artery, it is called ectasia. Dilatation may be either focal or diffuse, and aneurysms are classified as either fusiform (longitudinal dimension > transverse dimension) or saccular (transverse dimension > longitudinal dimension) in morphology. It has been noted that about one-third of CAAs are associated with obstructive coronary artery disease and have been associated with myocardial infarction, arrhythmias, or sudden cardiac death.[12]

Commonest cause of CAA in adult is atherosclerosis; however, other causes include Kawasaki's disease, autoimmune disease (polyarteritisnodosa, systemic lupus erythematosus, and scleroderma), rheumatic, mycotic coronary emboli, syphilis, trauma, coronary artery dissection, congenital cause, and angioplasty.[13]

More recently, with the increasing use of drug-eluting stents (DESs), there are growing reports signifying stents causing coronary aneurysms, months or even years after the intervention.[14] CAAs after coronary intervention are infrequent, with a reported incidence of 0.3%–6.0% and most aneurysms are in actual fact pseudoaneurysms rather than true aneurysms.[15]

Pathophysiology of Aneurysm Formation after DES Implantation

The proposed mechanism of stent-related aneurysm formation is multi-factorial. Mechanical factors considered in the formation of CAA are oversized balloons or high-pressure balloon inflation resulting in intimal and medial tear with weakening and

stretching of the artery. These mechanical consequences may finally lead to the perforation of the media without penetration of blood through the adventitia resulting in aneurysm formation. In addition to the mechanical risk factors for aneurysm formation that are observed with both BMS and DES, there are other potential mechanisms that may be specific to DES. Inflammatory reactions in response to implanted DES have been postulated as 1 such factor. Although inflammatory and allergic reactions to nickel and molybdenum have been reported after BMS implantation,[16] the triggers for inflammatory and allergic reactions after DES implantation are more complex because DES consist of 3 components: the antirestenotic drug, the drug carrier vehicle (polymer), and the stent platform. In particular, the polymer carrier has been shown to provoke eosinophilic/heterophilic infiltration and induce a marked inflammatory reaction of the arterial wall.[17,18] In addition, delayed healing reactions in response to DES, such as incomplete endothelialization over DES struts, have been detected by invasive approaches (angioscopy and optical coherence tomography) as well as in autopsy studies.[19,20] An additional phenomenon that has been observed after DES implantation is late acquired incomplete stent apposition, which is observed in 8% to 10% of patients.[21,22] In short, the combination of physical trauma induced by stent implantation and specific biological reactions after DES implantation might together contribute to coronary aneurysm formation after DES implantation.

The stent used in our case was a Sirulimus-eluting polymer coated stent, but technical details of angioplasty were not known as it was performed in another centre.

Proposed Classification of Coronary Artery Aneurysms

Classification of PCI associated CAA

Type	Description
Type -I	Aneurysm that demonstrates rapid growth with pseudo aneurysm formation detected within 4 weeks.
Type-II	Detected incidentally during angiography for recurrent symptoms or as part of protocol mandated follow-up e≥6 months after the procedure.
Type -III	Mycotic or infectious in etiology.

In a review of coronary artery aneurysm, Jiro Aoki and others described three different types of aneurysms after DES or BMS implantation.[23] Although there are no data regarding the prognostic important of differing aneurysm types, a system of aneurysm classification may be useful to guide therapies because some coronary aneurysms are fatal without prompt and appropriate treatment,[24,25,26] and some coronary aneurysms are not life-threatening events and need only careful observation without treatment.[27,28]

Type I aneurysm is a type of aneurysm that demonstrates rapid early growth with pseudoaneurysm formation detected within 4 weeks. [29, 30] This type is typically complicated by clinical pericarditis. Given the rapid time course of aneurysm formation, it is likely that arterial injury related to the procedure is the likely contributor to aneurysm formation in these cases rather than the chronic arterial response to the stent, polymer, and drug. The second kind of aneurysm described in the literature is that with a "subacute to chronic" presentation (type II) and is typically detected incidentally during angiography for recurrent symptoms or as part of protocol mandated follow-up (usually detected 6 months after the procedure).[31-33] These aneurysms appear to have the most varied clinical presentations; some patients are asymptomatic, but some have complaints of angina. It seems more likely in this scenario that a chronic arterial response to a metal stent, polymer, and/or drug, may be the basis for aneurysm formation in this subtype. The final reported subtype in the published literature is mycotic or infectious in etiology (type III).[34, 35] Large mycotic aneurysms infected with Staphylococcus aureus after DES or BMS implantation have been reported. In these rare cases, patients typically present with systemic manifestations and fever as the result of bacteremia. Whether the local immunosuppressive effects of eluted drugs from stents tend to increase the incidence of these rare infectious aneurysms is unknown. *Our case has characteristics of type III aneurysm.*

Diagnosis

CAA can be diagnosed by noninvasive and invasive techniques, such as echocardiography, CT,

magnetic resonance imaging (MRI), and coronary angiography. Coronary angiography remains the gold standard tool as it provides information about the shape, size, location, and co-existing anomalies such as coronary artery disease, and is also useful for setting up the strategy of surgical resection.[36] Coronary CT and MRI is an alternative to invasive coronary angiography. Intravascular ultrasound (IVUS) has become the "gold standard" technique that produces transluminal images of the coronary arteries, including information on the composition of the lumen and the arterial wall structure.[37] IVUS is very helpful in differentiating true from false aneurysms caused by plaque rupture.[37]

Proposed Treatment of Coronary Artery Aneurysms

The treatment of CAA consists of medical management, surgical resection, and stent placement; however, the appropriate treatment for CAAs depends on the precise clinical situation. In their study, Aoki et al.[38] proposed that the treatment of CAA be "individualized" using a combination of the size of the aneurysm, expansion history, pathophysiology, and symptoms to decide when and if to apply therapy alternatives.

Percutaneous intervention is a fairly new option with a markedly smaller data set. The described techniques include conventional stent implantation, coil embolization, autologous saphenous vein-covered stent grafting, and one case has been reported when DES implantation superimposed on a polytetrafluoroethylene (PTFE)-covered stent graft.[39]

Various surgical strategies have been described including resection, aneurysm ligation, marsupialization with interposition graft, and coronary artery bypass surgery. In symptomatic patients unsuitable for PCI, surgical excision or ligation of CAA combined with bypass grafting of the affected coronary arteries is the preferred option.[40]

Conclusions:

Coronary artery aneurysms after DES implantation are rare with variable clinical course. Some aneurysms naturally resolve, but some aneurysms can lead to life-threatening complications. Although the best treatment for coronary aneurysms after DES is controversial, we propose that a combination of aneurysm size, expansion history, pathophysiology, and symptoms be used to decide on treatment. Expanding pseudo aneurysms, infected aneurysms, and large, chronic (and expanding) aneurysms with symptoms should be treated. Further investigation is necessary to determine the pathophysiology, natural history, and best therapies for DES-associated aneurysms.

Conflict of Interest - None.

References:

1. Bell MR, Garratt KN, Bresnahan JF, Edwards WD, Holmes DR Jr.Relation of deep arterial resection and coronary artery aneurysms after directional coronary atherectomy. J Am Coll Cardiol 1992;20:1474–81.
2. Condado JA, Waksman R, Gurdiel O, et al. Long-term angiographic and clinical outcome after percutaneous transluminal coronary angioplasty and intracoronary radiation therapy in humans. Circulation 1997;96:727–32.
3. Slota PA, Fischman DL, Savage MP, Rake R, Goldberg S. Frequency and outcome of development of coronary artery aneurysm after intracoronary stent placement and angioplasty. STRESS Trial Investigators. Am J Cardiol 1997;79:1104–6.
4. Popma JJ, Leon MB, Moses JW, et al. Quantitative assessment of angiographic restenosis after sirolimus-eluting stent implantation in native coronary arteries. Circulation 2004;110:3773– 80.
5. Stone GW, Ellis SG, Cox DA, et al. A polymer-based, paclitaxeleluting stent in patients with coronary artery disease. N Engl J Med 2004;350:221–31.
6. Virmani R, Liistro F, Stankovic G, et al. Mechanism of late in-stent restenosis after implantation of a paclitaxel derivate-eluting polymer stent system in humans. Circulation 2002;106:2649 –51.
7. Luscher TF, Steffel J, Eberli FR, et al. Drug-eluting stent and coronary thrombosis: biological mechanisms and clinical implications. Circulation 2007;115:1051– 8.
8. Joner M, Finn AV, Farb A, et al. Pathology of drug-eluting stents in humans: delayed healing and late thrombotic risk. J Am Coll Cardiol 2006;48:193–202.
9. Swaye PS, Fisher LD, Litwin P, Vignola PA, Judkins MP, Kemp HG, *et al.* Aneurysmal coronary artery disease. Circulation 1983;67:134 8.
10. Kato H, Sugimura T, Akagi T, Sato N, Hashino K, Maeno Y, *et al.* Long term consequences of Kawasaki disease. A 10 to 21 year follow up study of 594 patients. Circulation 1996;94:1379 85.
11. Elahi MM, Dhannapuneni RV, Keal R. Giant left main coronary artery aneurysm with mitral regurgitation. Heart 2004;90:1430.

12. Indolfi C, Achille F, Tagliamonte G, Spaccarotella C, Mongiardo A, Ferraro A, *et al.* Polytetrafluoroethylene stent deployment for a left anterior descending coronary aneurysm complicated by late acute anterior myocardial infarction. Circulation 2005;112:e70 1.

13. Díaz Zamudio M, Bacilio Pérez U, Herrera Zarza MC, Meave González A, Alexanderson Rosas E, Zambrana Balta GF, *et al.* Coronary artery aneurysms and ectasia: Role of coronary CT angiography. Radiographics 2009;29:1939 54.

14. Nichols L, Lagana S, Parwani A. Coronary artery aneurysm: A review and hypothesis regarding etiology. Arch Pathol Lab Med 2008;132:823 8.

15. Bell MR, Garratt KN, Bresnahan JF, Edwards WD, Holmes DR Jr.Relation of deep arterial resection and coronary artery aneurysms afterdirectional coronary atherectomy. J Am Coll Cardiol 1992;20:1474 81.

16. Ge J, Liu F, Kearney P, et al. Intravascular ultrasound approach to the diagnosis of coronary artery aneurysms. Am Heart J 1995;130:765–71.

17. Virmani R, Guagliumi G, Farb A, et al. Localized hypersensitivity and late coronary thrombosis secondary to a sirolimus-eluting stent: should we be cautious? Circulation 2004;109:701–5.

18 . van der Giessen WJ, Lincoff AM, Schwartz RS, et al. Marked inflammatory sequelae to implantation of biodegradable and nonbiodegradable polymers in porcine coronary arteries. Circulation 1996;94: 1690–7.

19. Joner M, Finn AV, Farb A, et al. Pathology of drug-eluting stents in humans: delayed healing and late thrombotic risk. J Am Coll Cardiol 2006;48:193–202.

20. Kotani J, Awata M, Nanto S, et al. Incomplete neointimal coverage of sirolimus-eluting stents: angioscopic findings. J Am Coll Cardiol 2006;47:2108 –11.

21. Tanabe K, Serruys PW, Degertekin M, et al. Incomplete stent apposition after implantation of paclitaxel-eluting stents or bare metal stents: insights from the randomized TAXUS II trial. Circulation 2005;111:900 –5.

22. Ako J, Morino Y, Honda Y, et al. Late incomplete stent apposition after sirolimus-eluting stent implantation: a serial intravascular ultrasound analysis. J Am Coll Cardiol 2005;46:1002–5.

23. Hirsch GM, Casey PJ, Raza Ahmad A, Miller RM, Hirsch KJ. Thrombosed giant coronary artery aneurysm presenting as an intracardiac mass. Ann Thorac Surg 2000;69:611 3.

24. Singh H, Singh C, Aggarwal N, Dugal JS, Kumar A, Luthra M. Mycotic aneurysm of left anterior descending artery after sirolimuseluting stent implantation: a case report. Catheter Cardiovasc Interv 2005;65:282–5.

25. Alfonso F, Moreno R, Vergas J. Fatal infection after rapamycin eluting coronary stent implantation. Heart 2005;91:e51.

26. Alfonso F, Moreno R, Vergas J. Mycotic aneurysms after sirolimuseluting coronary stenting. Catheter Cardiovasc Intv 2006;67: 327– 8.

27. Nilsen DW, Melberg T, Larsen AI, Barvik S, Bonarjee V. Late complications following the deployment of drug eluting stents. Int J Cardiol 2006;109:398–401.

28. Kim HS, Park JH, Ko JK. Asymptomatic coronary artery aneurysm associated with paclitaxel eluting stent. Heart 2006;92:480.

29. Gupta RK, Sapra R, Kaul U. Early aneurysm formation after drugeluting stent implantation: an unusual life-threatening complication. J Invasive Cardiol 2006;18:E140 –2.

30. Zhang F, Qian JY, Ge JB. Rapid development of late stent malappositon and coronary aneurysm following implantation of a paclitaxeleluting coronary stent. Chin Med J (Engl) 2007;120:614–6.

31. Porto I, MacDonald S, Banning AP. Intravascular ultrasound as a significant tool for diagnosis and management of coronary aneurysms. Cardiovasc Intervent Radiol 2004;27:666–8.

32. Panja M, Basu S, Mondol S. A case of giant aneurysm following percutaneous coronary intervention. Indian Heart J 2005;57:731–3.

33. Lubell D, Gruberg L, Hermiller JB, Gonschior P. Post-stent very proximal left anterior descending coronary artery aneurysm. J Invasive Cardiol 2005;17:230 –2.

34. Jindal RK, George R, Singh B. Giant coronary aneurysm following drug-eluting stent implantation presenting as fever of unknown origin. J Invasive Cardiol 2007;19:313– 4.

35. Singh H, Singh C, Aggarwal N, Dugal JS, Kumar A, Luthra M. Mycotic aneurysm of left anterior descending artery after sirolimuseluting stent implantation: a case report. Catheter Cardiovasc Interv 2005;65:282–5.

36. Pahlavan PS, Niroomand F. Coronary artery aneurysm: A review. Clin Cardiol 2006;29:439 43.

37. Sanidas EA, Vavuranakis M, Papaioannou TG, Kakadiaris IA, Carlier S, Syros G, *et al.* Study of atheromatous plaque using intravascular ultrasound. Hellenic J Cardiol 2008;49:415 21.

38. Aoki J, Kirtane A, Leon MB, Dangas G. Coronary artery aneurysms after drug eluting stent implantation. JACC Cardiovasc Interv 2008;1:14 21.

39. Jindal RK, George R, Singh B. Giant coronary aneurysm following drug-eluting stent implantation presenting as fever of unknown origin. J Invasive Cardiol 2007;19:313– 4.

40. Badmanaban B, Mallon P, Campbell N, Sarsam MA. Repair of left coronary artery aneurysm, recurrent ascending aortic aneurysm, and mitral valve prolapse 19 years after Bentall's procedure in a patient with Marfan syndrome. J Card Surg 2004;19:59 61.