CASE REPORT

An unusual cause of ST elevation myocardial infarction (STEMI)

Mohammed Monem, Rajiv Rampat

St Peter's Hospital, Surrey, UK

Correspondence to
Dr Mohammed Monem,
monem@doctors.org.uk

Accepted 31 August 2014

SUMMARY

A 67-year-old Caucasian woman presented to clinic with a 2-month history of worsening shortness of breath on exertion and a single episode of chest pain 1 week before. Her ECG in clinic showed ST elevation inferiorly and she was admitted from clinic for further investigations as inpatient. She was initiated on the acute coronary syndrome protocol and underwent emergency left heart catheterisation on the day of admission. The coronary angiogram revealed large aneurysmal dilations in the right coronary artery and left main stem. A ventriculogram showed poor left ventricular (LV) systolic function in line with subsequent transthoracic echocardiogram, which revealed her to have an left ventricular ejection-fraction (LVEF) of approximately 20%. It was agreed with the cardiothoracic surgeons to treat the aneurysms non-operatively and start low-molecular weight heparin. Furthermore the underlying biventricular impairment was treated with ACE-inhibitors, β -blockers and diuretic therapy (loop and potassium-sparing). The strategy was to prevent further thrombus formation with the aneurysmal vessels and to achieve this the patient was initiated on lifelong warfarin. Other medical risk factors were optimised and patient started on statin medication. The aneurysm was monitored with serial CTs with a view to reconsider surgical intervention if any evidence of dilation. This case highlights an unusual cause of ST elevation myocardial infarction.

BACKGROUND

This case highlights a rare cause of ST elevation myocardial infarction (STEMI). The management is particularly challenging and needs a multidisciplinary approach with close involvement of the cardiothoracic surgical team.

CASE PRESENTATION

A 67-year-old Caucasian woman was referred to the outpatient cardiology clinic with a 2-month history of exertional dyspnoea and reduced exercise tolerance. One week prior to admission, she had an episode of central chest pain which was associated with clamminess and sweating but had been pain-free since. She did not have any significant medical history or significant cardiovascular risk factors. Her ECG in clinic showed ST elevation inferiorly.

The decision was made to admit her for further investigations. Once on the ward, she received a loading dose of aspirin and clopidogrel and had a left heart catheterisation later that day.

INVESTIGATIONS

Her ECG in clinic showed ST elevation in the inferior leads consistent with a inferior STEMI. In the light of her clinical presentation, it was felt that the index event occurred a week earlier and that this was a late presentation of a STEMI. Serial ECGs did not show dynamic changes further confirming that this was not an acute presentation.

Her coronary angiogram revealed massive aneurysmal dilations in the right coronary artery (RCA) and in the left main stem. The RCA had severe diffuse fusiform proximal and middle artery aneurysm with distal occlusion ([figure 1](#)). The left coronary artery had a left main stem aneurysm but good distal flow ([figure 2](#)).

The distal occlusion of the RCA was felt to be responsible for the ST elevation on her ECG. However, as her chest pain had occurred 1 week before and she had been pain free since, the decision was made to not open up the occluded distal RCA. The residual ST segment elevation most likely signified a completed myocardial infarction. Since the index event had happened over a week before, no thrombin inhibitor was administered.

Her brain natriuretic peptide (BNP) was elevated at 820 ng/L (normal <100). She had an inpatient

Figure 1 Coronary angiogram right anterior oblique view—diffuse fusiform aneurysm of the right coronary artery.

CrossMark

To cite: Monem M, Rampat R. *BMJ Case Rep* 2014. Published online: [please include Day Month Year] doi:10.1136/bcr-2014-203981

Figure 2 Coronary angiogram right anterior oblique view—left main stem aneurysm.

transthoracic echocardiogram which showed severe LV (EF 20%) and moderate right ventricle impairment. This was thought to be secondary to her previous inferior STEMI.

An inpatient cardiac CT measured the aneurysms at approximate 15 and 18 mm of the left and right coronary arteries, respectively (figures 3 and 4).

DIFFERENTIAL DIAGNOSIS

The aetiology of aneurysmal coronary arteries, include atherosclerosis of the vasculature (50%), autoimmune (Kawasaki) and congenital causes (17%), septic emboli (11%), connective tissue (including Marfan's) disease (<10%), arteritis including Takayasu and systemic lupus erythematosus (<10%) and finally trauma.

In this particular case, the aetiology is unclear. Her autoimmune screen was negative and there was no clinical evidence

Figure 3 Coronary CT—right coronary artery (arrow head, white) and left main stem artery (pointed arrow) aneurysms.

Figure 4 Volume rendered cardiac CT—right (arrow head, white) and left (pointed arrow) coronary aneurysm.

of connective tissue disorders, sepsis or trauma. There was no family history of premature or sudden cardiac death. It was felt that her presentation was likely to be congenital in origin though this was a diagnosis of exclusion.

TREATMENT

Her biventricular impairment was managed medically with frusemide, bisoprolol, ramipril and eplerenone.

Regarding her coronary aneurysms, the case was discussed with the cardiothoracic surgical team who advised medical management. As there was no evidence of viability on the echocardiogram, no percutaneous intervention was undertaken. To reduce the risk of further coronary thrombus given her abnormal arteries, the decision was made for life long anticoagulation unless the aneurysms were surgically treated. She was started on treatment dose low-molecular weight heparin (LMWH; 1.5 mg/kg once daily) and referred to the anticoagulation clinic to be started on warfarin. She was also started on clopidogrel in view of her STEMI for a total duration of 1 year.

The plan was for regular review in the outpatient cardiology clinic with serial CT imaging of her coronary arteries with a view to surgical intervention if there was evidence of further dilation.

OUTCOME AND FOLLOW-UP

On follow-up at 2 months, the patient was asymptomatic. Repeat cardiac CT did not show any increase in aneurysm size.

Repeat echocardiogram was performed 6 months later and this shows improvement in cardiac function with a LVEF between 40 and 45%.

Her BNP also improved to 337 from 820 on admission. She has been reviewed in the community heart-failure team for optimisation of her medication for heart failure. She continued to make steady progress through out.

DISCUSSION

Since the first description in 1761, there have been increasing reports of coronary aneurysms in the medical literature which is probably a result of the increasing use of coronary angiography and non-invasive cardiac imaging.¹

An aneurysm is defined as a localised dilation of coronary artery whose diameter is at least 50% larger than the adjacent coronary vessel. The incidence ranges between 1.4 and 5.3% based on angiography and autopsy series. It is predominately found in males (72–90% range) and in late middle age, with an average age of 63.5 on diagnosis by angiography. It has also been shown to have a dominant occurrence in the RCA.²

The presentation varies depending on the underlying cause the aneurysm. This ranges from no symptoms at one end of the spectrum to sudden cardiac death at the other end. Typically symptomatic patients have signs and symptoms of ischaemia due to coronary obstruction. Other characteristics such as the extent of collateral blood supply and the size of the aneurysm can affect the severity of presentation. Complications include thromboembolism, vasospasm, rupture, coronary artery dissection extending into the aorta and cardiac tamponade.³

A subgroup analysis of the CASS registry looking at the subset of patients with coronary artery aneurysms. No significant differences were noted in patient characteristics when compared to a cohort with non-aneurysmal coronary artery disease. There was no difference in 5-year mortality between patient with coexisting aneurysmal arteries and coronary stenosis versus patients with coronary stenosis alone. Coronary artery bypass grafting treatment yielded similar survival curves irrespective of the presence of coronary artery aneurysm. They concluded that aneurysmal coronary arteries appeared to be a variant of atherosclerosis and did not in itself confer a higher risk of MI or mortality.⁴

In contrast, a more recent retrospective analysis found that the presence of coronary aneurysm was an independent risk factor on long-term mortality rate. There was a significant 5-year mortality rate of 29% which was similar to the CASS registry. Given that these two studies were performed two decades apart, it would appear that advances in medical treatment and risk factor modification have not really changed the outcome of coronary artery aneurysm. Surprisingly, the survival rate did not seem to depend on the presence of concomitant coronary artery stenosis. The authors advocated aggressive risk factor modification independent of the presence of coexisting coronary stenosis.⁵

The mechanism of aneurysms is still not fully understood. Hypothesis include inflammation within the tunica media of the coronary artery leading to an increased breakdown of extracellular proteins and material. Metalloproteinase enzymes are present in elevated numbers in aortic aneurysm and have a potential to breakdown the components of the arterial wall material including elastin, collagen and laminin. In addition, a reduced level of matrix proteinase inhibitors can weaken the vascular wall leaving the vessel vulnerable to dilation under physical stress.^{6,7}

Atherosclerosis is believed to be the biggest cause of aneurysms causing an inflammatory process affecting the subendothelial layer resulting in aneurysm formation. This same process is likely to occur in patients with autoimmune vasculitis with or

without atherosclerosis. This includes Kawasaki disease which is the most common cause of coronary aneurysms in Japan.

Mycotic disease can present at any age and hypothesis relating to the mechanism of action include direct pathogen invasion of the wall and immune-complex deposition leading to weakening of the wall. Congenital causes may result from abnormal differentiation of the progenitor cells during cardiac development. Connective tissue abnormalities such as Marfans and Ehler-Danlos syndrome, are among the genetic causes. Finally trauma has been suggested to lead to direct damage to the endothelial and vascular wall, leading to aneurysm formations.^{8–11}

Coronary artery aneurysms have historically been treated both surgically and medically. The aim of treatment in acute settings is to prevent fatal complications including an expanding aneurysm which is at higher risk of rupture as well as causing significant myocardial ischaemia. Currently there are no clinical guidelines to guide physicians or surgeons on optimal management. Invasive options include either percutaneous or open surgical approach. In the open approach, coronary bypass surgery with or without ligation, aneurysmectomy or marsupialisation of the lesion can be employed. Follow-up of patients on the CASS registry showed no difference in outcome between patients who had bypass surgery for both aneurysm and coronary artery disease versus coronary artery disease alone.^{12,13}

Percutaneous approaches include coronary stent and coil placement. Percutaneous stenting has been compared to open surgery with no significant difference in mortality. However, the stented arm had a higher incidence of repeat angiography due to restenosis. There is a lack of prospective randomised trials comparing surgical versus percutaneous treatment.^{14,15}

Medical therapy involves antiplatelet and anticoagulation therapy to prevent and treat thrombosis. It has been suggested that up to 70% of coronary aneurysm have acute thrombosis.¹⁶

Thus each patient should be treated depending on the clinical presentation on a case-by-case basis. There are no clearly defined criteria to identify those patients who would benefit from an invasive approach or more conservative treatment. However, there is a consensus that in symptomatic patients and/or in the presence of significant coronary stenosis, invasive intervention is preferable. If the patient is not suitable in view of comorbidities, aggressive cardiovascular risk factor modification should be the mainstay of treatment.

Learning points

- ▶ Coronary aneurysms are a rare but important cause of ST elevation myocardial infarction.
- ▶ There are various medical conditions associated with coronary aneurysms but the exact mechanism of aneurysm formation is still not fully understood.
- ▶ Treatment options include medical, percutaneous and surgical treatment. This requires a multidisciplinary approach based on the specific clinical features of individual patients.

Acknowledgements The authors would like to thank all the staff and consultants at St Peter's Hospital, Chertsey, Surrey.

Contributors MM and RR have been involved in writing and editing the manuscript.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Morgagni JB. De Sedibus et Causis morborum. Venetus Tom I, Epis 27, Art 28, 1761.
- 2 Syed M, Lesch M. Coronary artery aneurysm: a review. *Prog Cardiovasc Dis* 1997;40:77–84.
- 3 Pahlavan PS, Niroomand F. Coronary artery aneurysm: a review. *Clin Cardiol* 2006;29:439–43.
- 4 Swayze PS, Fisher LD, Litwin PA, et al. Aneurysmal coronary artery disease. *Circulation* 1983;67:134–8.
- 5 Baman TS, Cole JH, Devireddy CM, et al. Risk factors and outcomes in patients with coronary artery aneurysms. *Am J Cardiol* 2004;93:1549–51.
- 6 Senzaki H. The pathophysiology of coronary artery aneurysms in Kawasaki disease: role of matrix metalloproteinases. *Arch Dis Child* 2006;91:847–51.
- 7 Lamblin N, Bauters C, Hermant X, et al. Polymorphisms in the promoter regions of MMP-2, MMP-3, MMP-9 and MMP-12 genes as determinants of aneurysmal coronary artery disease. *J Am Coll Cardiol* 2002;40:43–8.
- 8 Nichols L, Lagana S, Parwani A. Coronary artery aneurysm: a review and hypothesis regarding etiology. *Arch Pathol Lab Med* 2008;132:823–8.
- 9 Kayalar N, Burkhart HM, Dearani JA, et al. Congenital coronary anomalies and surgical treatment. *Congenit Heart Dis* 2009;4:239–51.
- 10 Ebina T, Ishikawa Y, Uchida K, et al. A case of giant coronary artery aneurysm and literature review. *J Cardiol* 2009;53:293–300.
- 11 Díaz-Zamudio M, Bacilio-Pérez U, Herrera-Zarza MC, et al. Coronary artery aneurysms and ectasia: role of coronary CT angiography. *Radiographics* 2009;29:1939–54.
- 12 Mavrogeni S. Coronary artery ectasia: from diagnosis to treatment. *Hellenic J Cardiol* 2010;51:158–63.
- 13 Keyser A, Hilker MK, Husser O, et al. Giant coronary aneurysms exceeding 5cm in size. *Interact Cardiovasc Thorac Surg* 2012;15:33–6.
- 14 Szalat A, Durst R, Cohen A, et al. Use of polytetrafluoroethylene-covered stent for treatment of coronary artery aneurysm. *Catheter Cardiovasc Interv* 2005;66:203–8.
- 15 Zeb M, McKenzie DB, Scott PA, et al. Treatment of coronary aneurysms with covered stents: a review with illustrated case. *J Invasive Cardiol* 2012;24:465–9.
- 16 Kelley MP, Carver JR. Coronary artery aneurysms. *J Invasive Cardiol* 2002;14:461–2.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.

BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow