An Asymptomatic Left Ventricular Pseudoaneurysm Found Incidentally at 12 Months Post Myocardial Infarction: Case Report and Review of the Literature

Christian C Brooks¹, Heather A Cooke²

¹. General Medicine, John Hunter Hospital, New South Wales, Australia.
². Department of Cardiology, John Hunter Hospital, New South Wales, Australia.

Corresponding author:
Christian Brooks
John Hunter Hospital,
Lookout Road, New Lambton Heights, NSW 2305, Australia.
E-mail: christian.brooks@health.nsw.gov.au

Highlights

Left ventricular pseudoaneurysms are a rare mechanical complication of myocardial infarction. If found acutely following infarction (within 2 weeks, with some advocating up to 3 months), surgical repair is recommended due to their high risk of rupture. Whilst associated with chest pain, dyspnoea and heart failure, some individuals are asymptomatic, with the diagnosis made incidentally on routine follow-up often months to years post infarction. Less is known about the natural history of these chronic pseudoaneurysms, with concerns around their propensity to rupture perhaps less than the mortality risk of surgical repair. We present the case of a 70 year-old asymptomatic man who was found to have a 1.6cm left ventricular pseudoaneurysm found incidentally on routine transthoracic echocardiogram at 12-months post posterior myocardial infarction. The consensus opinion of our institution’s multi-disciplinary team regarding further management of this patient, with reference to the current limited data on chronic pseudoaneurysms, will be discussed.

Keywords: Cardiac catheterisation; Echocardiography; Left Ventricular Pseudoaneurysm; Myocardial Infarction


© 2020 Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution CC-BY 4.0 license CC-BY-4.0 (http://creativecommons.org/licenses/by/4.0/), which permits use, distribution and reproduction, provided the original work is properly cited. Published by Barcaray (International) Publishing.

Introduction

In the era of thrombolysis and emergent percutaneous coronary intervention (PCI) for ST elevation myocardial infarction (STEMI), mechanical complications such as left ventricular pseudoaneurysm (LVP) are increasingly rare. Historically rates of LVP rupture have been quoted as high as 44%,[1] with a mortality rate of 48% in those whom did not receive surgical repair. [2] Following surgical repair postoperative mortality remains elevated at 23-35%.[2, 3]

More recent data, however, has demonstrated long-term survival with a more conservative approach in carefully selected patients. Moreno et al.[4] showed a cumulative survival of 89% at 1 year and 74% at 4 years in those treated conservatively, noting an 89% probability of survival at 4 years if non-cardiac death was excluded from analysis. A LVP has been described 15 years post detection in a patient treated conservatively.[5]

Considering the risks associated with cardiac surgery and this more recent data, it is possible that for some patients, conservative management is appropriate.

Case Presentation

A 70 year-old man presented to our institution following an episode of chest pain that he had experienced 3 days prior. The chest pain lasted 30-hours, radiated superiorly into his neck and was not associated with shortness of breath, diaphoresis or nausea.

His past medical history was notable for hypertension, for which he was prescribed telmisartan 80mg daily, amlodipine 5mg daily and hydrochlorothiazide 12.5mg daily.

Initial clinical examination was unremarkable, as he was normotensive (119/70 mmHg), with a heart rate of 77 beats per minute (regular), a respiratory rate of 17 breaths per minute,
a peripheral oxygen saturation of 97% on room air and a
temperature of 36.7°C. Both heart sounds were appreciated,
there were no cardiac murmurs or pericardial rubs, both lung
fields were clear to auscultation, jugular venous pressure was
not elevated and peripheral oedema was not present.
His ECG demonstrated non-dynamic ST depression in leads
V1-3 with prominent R waves consistent with posterior ischemia/
infarction (Figure 1).

High-sensitivity serum troponin on presentation was 8184
ng/L (< 26ng/L). The patient underwent coronary angiography
revealing 70% distal left anterior descending (LAD) stenosis, as
well as 90% stenosis of the mid obtuse marginal (OM1) branch
(culprit vessel) and 70% ostial stenosis of the OM2 branch of
the circumflex artery in a right dominant coronary system. On
ventriculography, left ventricular end diastolic pressure (LVEDP)
was noted to be elevated at 17mmHg yet left ventricular function
was reported as normal.

Medical therapy in the form of aspirin 100mg daily, clopidogrel
75mg daily, metoprolol 25mg twice daily and rosuvastatin 10mg
daily was commenced as treatment for the diagnosis of a delayed
presentation, non ST-elevation myocardial infarction (NSTEMI),
noting that if the patient had recurrence of symptoms, the OM1
and LAD lesions were amenable to stenting.

At 12 months, the patient remained asymptomatic and repeat
clinical examination was unremarkable. Routine transthoracic
echocardiogram (TTE), however, demonstrated a discrete LVP
measuring approximately 1.6cm confined to the mid posterior
wall, with normal left ventricular systolic function and no valvular
pathology was detected. Informed consent was obtained from
the patient, allowing us to generate this case report.

Discussion
The prevalence of LVP’s following myocardial infarction in a
series of 2600 consecutive patients was reported to be 0.23%.[6]
An LVP forms when left ventricular free wall rupture is contained
by adherent pericardium and fibrotic tissue.[7] Inferior myocardial
infarctions are responsible for about twice as many LVP’s as
anterior myocardial infarctions, which reflects the propensity of
LVP’s to form on the posterior, lateral, apical or inferior surface
of the left ventricle, as opposed to anteriorly.[2] Anterior free
wall rupture is more likely to lead to haemopericardium and
subsequently death. Recumbent positioning in hospitalized patients promotes inflammation of the posterior pericardium and
the formation of adhesions necessary to contain pooled blood.[8]

A LVP can be detected on a TTE, whereby there is a sharp
demarcation between normal myocardium and the aneurysm
which does not contain myocardium, via a narrow neck that is
less than 50% of the diameter of the aneurysmal sac.[9] If not
detected acutely, many pseudoaneurysms are found incidentally
upon investigation much later; in one case series, aneurysms
were detected a median of 7 months (range 1-11months) post
myocardial infarction.[3]

In identifying those likely to benefit from a conservative approach,
the distinction between an acute and chronic LVP has been
made. It is recommended that acute LVP’s, defined as those
developing within 2 weeks of infarct, be managed with urgent
surgical intervention.[10] Some advocate extending the window
for emergency surgery to LVP’s detected up to 3 months post
infarct.[3]

For an LVP diagnosed beyond 3 months following infarct opinions
vary, however it has been proposed that if the LVP is <3cm in
diameter, isn’t rapidly growing on serial imaging and the patient is
asymptomatic with an absence of significant valvular or coronary
disease requiring operative intervention, that a conservative
approach with regular cardiology follow-up is reasonable.[10]

Conclusion
For this patient, the consensus opinion from our institution’s
combined cardiology and cardiothoracic surgery multi-disciplinary
meeting was in accordance with the current (albeit limited)
literature on the management of chronic pseudoaneurysms that suggests taking a conservative approach initially with serial echocardiograms and follow-up consultations with a cardiologist every 6 months to monitor for the development of symptoms or pseudoaneurysm growth on serial imaging, with surgical intervention then reconsidered if either of these were to develop.

**Declarations of Interest**

The authors declare no conflicts of interest.

**Acknowledgements**

The authors state that they abide by the authors’ responsibilities and ethical publishing guidelines of the International Cardiovascular Forum Journal.[11]

**Patient consent.**

Consent was obtained from the patient to generate this case report.

**References**


