PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY IN ACUTE CARDIOGENIC SHOCK: AN OVERVIEW OF THE CURRENT STRATEGY WITH A CASE ILLUSTRATION

K W Lau, A Tan, A Johan

ABSTRACT

Cardiogenic shock from severe left ventricular dysfunction remains the most common cause of death in patients with acute myocardial infarction despite the advent of inotropic agents and intra-aortic balloon support. Lytic agents have not convincingly reduced mortality when administered in a shock scenario. Recently, observational studies have demonstrated survival benefit when mechanical revascularisation, particularly balloon angioplasty, is performed early in such patients.

In this article, we report a case of an extensive anterior myocardial infarction with cardiogenic shock who underwent successful acute balloon angioplasty and prolonged intracoronary thrombolysis and use it to illustrate the current evolving interventional approach in the treatment of such a complex clinical syndrome.

Keywords: *Cardiogenic shock, angioplasty, thrombolysis.*

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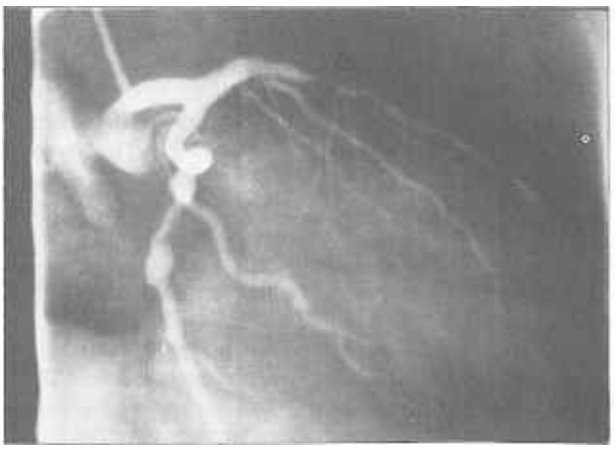
INTRODUCTION

Despite the introduction of coronary care and the wide availability of potent inotropic agents and intra-aortic balloon (IAB) counterpulsation support system, the high mortality associated with cardiogenic shock complicating acute myocardial infarction remains unabated[1,2]. In fact, cardiogenic shock which occurs in about 5% to 15% of acute infarct patients, is the primary cause of in-hospital death in these patients[1]. The systemic infusion of various thrombolytic agents, while effective in reducing mortality in the majority of patients with no haemodynamic compromise, is controversial in a shock scenario as there is no clear-cut demonstrable survival benefit following their application in such circumstances[1]. Although emergency bypass surgery following the insertion of intra-aortic balloon counterpulsation has reduced mortality, its use is limited by the need of a highly experienced surgical team and rapid access to a surgical suite[1-3]. Recently, percutaneous transluminal coronary angioplasty (PTCA) has emerged as a promising mechanical revascularisation alternative in patients with cardiogenic shock[1]. In this report, we will present the management approach of a case of extensive Q-wave myocardial infarction complicated by cardiogenic shock. This is followed by a review of the current status of PTCA and other adjunctive therapy in shock management.

CASE HISTORY

A previously asymptomatic 54-year-old man with multiple coronary risk factors was admitted with a $2^1/_2$ hour history of sudden central, compressive chest pain associated with profuse perspiration. The 12 lead ECG revealed changes consistent with an extensive acute anterior Q-wave infarction. The peak creatine kinase level was 4020 U/L. Initial physical examination revealed no evidence of left ventricular failure and a cuff blood pressure reading of 140/100 mmHg. Intravenous streptokinase (1.5 MU over 1 hour) was administered within 3 hours of the onset of chest pain together with 100 mg of chewable aspirin. An hour after the completion of the thrombolytic agent, the patient extended his infarction laterally and promptly went into shock (blood pressure of 80/60 mmHg with peripheral shut-down despite maximal inotropic support). Emergency cardiac catheterisation revealed uncollateralised single vessel disease with a 95% diameter stenosis of the proximal left anterior descending arterial segment (Fig 1) and TIMI grade II flow. The left ventricular ejection fraction was 30%. PTCA using a conventional over-the-wire system was attempted under intra-aortic balloon counterpulsation support within 6 hours of the onset of the index infarct. During the procedure, the infarct-related lesion developed recurrent acute thrombotic closure (Fig 2) necessitating repeated dilatation with a slightly oversized balloon inflated at low pressures for longer durations.

Fig 1 - A freeze frame in the right anterior oblique projection reveals a 95% diameter stenosis in the proximal left anterior descending (LAD) segment just before the first septal perforator (open arrow). There is under-filling of the LAD distal to the tight lesion.



Department of Cardiology
Singapore General Hospital
Outram Road
Singapore 0316

K W Lau, MBBS (Monash), MMed (Int Med)
Consultant Cardiologist

A Tan, FRACP
Head

A Johan, FRACP
Senior Consultant

Fig 2 - Acute recurrent thrombotic closure (open arrow) is seen during PTCA as the balloon is deflated and withdrawn into the guiding catheter. The guiding wire is left across the lesion.

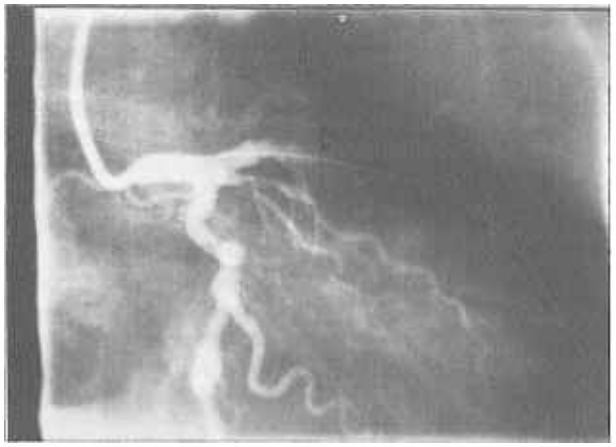
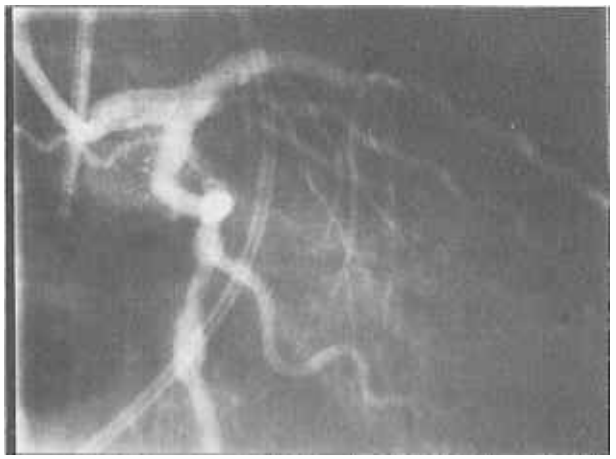


Fig 3 - A minor less complex residual lesion (open arrow) is seen after protracted intracoronary thrombolysis and repeat PTCA.



Despite this approach, there was still threatened closure of the dilated segment associated with ischaemia. The morphologic appearance of the lesion after dilatation was suggestive of an ulcerated lesion with overlying thrombus (Fig 3). At this stage, a "bailout" perfusion catheter was placed across the lesion successfully 'splinting' the latter and alleviating the ischaemia. Direct administration of intracoronary streptokinase and urokinase along with heparin were infused through the catheter over 2 days, at the end of which a repeat angiography was performed. The latter study revealed a lesion of about 40% but with a reduced volume of thrombus. A repeat uncomplicated PTCA was performed, reducing the stenosis to 20%. The clinical condition of the patient improved further and the intra-aortic balloon was removed. The blood pressure was maintained at about 130/80 mmHg and the patient was discharged with aspirin, warfarin, captopril. Follow-up angiography at one and six months post-PTCA revealed an enhancement in luminal appearance; the rough luminal surface was much smoother. The latter phenomenon, no doubt, is secondary to resolution of residual thrombus and reparative processes. Importantly, there was an increment in both regional and global left ventricular ejection fractions. The latter was noted to have improved by 10%, from 30% to 40% at the six-month angiogram.

DISCUSSION

This case report raises a number of crucial issues in the current management of cardiogenic shock. Firstly, acute PTCA in cardiogenic shock is not only feasible but highly effective in myocardial salvage when successfully executed. Secondly, the direct intracoronary thrombolytic intervention in abrupt thrombotic reclosure during infarct PTCA is a realistic and acceptable option. Thirdly, the availability of numerous novel intracoronary devices has confounded many interventional cardiologists who will need to know the specific niches of each device, particularly their potential role in such a situation as the case reported above. These issues will now be discussed in greater depth.

Role of PTCA in acute cardiogenic shock

The mortality of infarct-related cardiogenic shock remains extremely high (in excess of 80 to 90%) despite the use of pharmacologic agents and intra-aortic balloon counter-pulsation[1,2]. The current strategy in managing this syndrome seems to be that of urgent contrast angiographic delineation of the coronary anatomy followed by immediate interventional utilisation of intra-aortic balloon counterpulsation combined with rapid mechanical revascularisation. Intra-aortic balloon pump support alone can stabilise the haemodynamic status but does not seem to enhance survival[4,5].

Early coronary artery bypass surgery (CABG) with intra-aortic balloon support has been shown by a number of investigators to be an effective option with improvement in both acute and intermediate term survival, particularly when used early, within 16 to 24 hours following the onset of shock and in patients less than 75 years old[1,2,6]. However, it is not likely to be as widely applicable as PTCA which is more easily available, logistically simpler and less costly, achieves reperfusion more rapidly and appears to have a more consistent success rate compared to CABG. With the introduction of percutaneous cardiopulmonary support system and haemopump, especially in cases where the intra-aortic balloon pump may be rendered less effective or ineffective from extremely low systemic pressures, the clinical utility of PTCA may now be extended to more severely compromised patients[7,8]. Importantly, PTCA in shocked patients should only be performed on the infarct-related artery in the initial setting. In order to provide complete revascularisation, staged PTCA can then be attempted on other coronary arteries should there be amenable lesions present in them. Or, alternatively, once successful dilatation of the infarct-related artery and more haemodynamic stability is attained, the patient may then be sent for coronary artery bypass surgery on a less urgent basis thereby permitting the use of more durable internal mammary arteries instead of vein grafts.

It is well recognised that acute PTCA in cardiogenic shock generally affords a lower success rate and lower patency rate compared to elective PTCA[1,9]. It, nevertheless, prolongs survival when performed effectively. Pooled data and numerous smaller series show that it decreases mortality substantially (from about 80% to 30-40%) acutely[1,2,8-12] and maintains this survival benefit in the long-term[1,2,8-10,13]. Furthermore, those who survive to be discharged from hospital seem to have a reasonable quality of life with many returning to gainful employment[9].

Hence, observational reports thus far suggest that mechanical revascularisation, either CABG or PTCA, coupled with the use of adjunctive support devices prolongs survival in patients with extensive infarction and cardiogenic shock. It does so probably by the reversal of myocardial stunning, limitation of myocardial necrosis (and hence myocardial salvage), protection of the peri-infarct zone and prevention of acute infarct expansion[1,9]. O'Keefe et al[13] in fact noted that patients with the largest infarcts (including cardiogenic shock) with extensive amount of jeopardised myocardium appear to benefit most from infarct PTCA. The global left ventricular ejection fraction improved from a mean of 28% to 45% in patients with cardiogenic shock after successful dilatation, a scenario also observed in our patient.

Role of adjunct intracoronary thrombolytic agents

Abrupt atherosclerotic plaque rupture with superimposed thrombotic occlusion appears to be the pivotal initiating/perpetuating event in acute myocardial infarction[14]. The existing thrombus, furthermore, is extremely thrombogenic and provides a nidus for more thrombus formation. Hence, it is not surprising that PTCA of the thrombus-laden lesion in an acute infarct setting has been observed to be associated with a lower patency rate and higher acute occlusion rate than elective dilatation of thrombus-free lesions[15, 16]. In fact, a number of studies have identified the presence of intracoronary thrombus as a significant risk predictor of acute thrombotic closure, either in or out of the catheterisation laboratory[17-20]. The use of intracoronary thrombolytic therapy in such a situation is thus an acceptable if not an established adjunct to PTCA[12, 21-23]. Gulba et al[22] recently achieved an 81.5% recanalisation rate in acute thrombotic closure following the infusion of combined intravenous/intracoronary tissue-type plasminogen activator and repeat balloon dilatations. Suryapranata et al[21] similarly reported a high thrombus resolution rate with intracoronary streptokinase.

Other potential alternative therapeutic options

One could ask the question, "Could there be any other potential invasive alternative in the acute management of this patient?" This is especially pertinent in today's high technology era in which interventional cardiologists are expected to keep up with and gain experience in the never-ending avalanche of new therapeutic devices and are generally confused in the process. An in-depth analysis of the targeted use of all these devices and their clinical utility is beyond the scope of this article and has been extensively covered in previous publications[24-27]. However, a brief discussion will be attempted here. Importantly, the discussion which follows is at best a theoretical consideration as there has been no published study or trial on any of these devices in an acute infarct or cardiogenic shock setting .

Laser balloon angioplasty[26,27] which utilises continuous wave neodymium:yttrium-aluminum garnet (Nd: YAG) laser irradiation to heat up a distal balloon placed at the culprit lesion, probably shows the greatest promise in this situation. Preliminary studies have shown it to be highly effective in resolving acute reclosure due to either dissection or thrombosis or both, often re-establishing antegrade flow, salvaging myocardium and circumventing the need for emergency CABG. Theoretically, it seals off the intimal tear and dessicates clot. Unfortunately, its major caveats are a high restenosis rate and its high cost.

Directional atherectomy[26, 27], a catheter with a hand-held disposable motor at the proximal end and a rigid metal housing with an open window and cutter at the distal end, may also be useful in this patient as it effectively removes clot and is more suited for large vessels. Its disadvantages are the high profile of the device requiring some expertise in using it and limiting its wider application, its high restenosis rate, its 1% incidence of perforation and its rather high expense.

Although the niche for intracoronary stenting[24, 25] in a bail-out scenario in acute closure or threatened closure is no longer in doubt, its use is more appropriate for acute closure due to dissection rather than a large thrombotic load. All current stents in clinical use are metallic without exception and are thus highly thrombogenic. This unfavourable characteristic will most likely only aggravate the situation in this patient whose coronary lesion already has an immensely high propensity for thrombosis. Furthermore, stents should not be implanted when there is significant haemodynamic compromise as this will promote stasis and thrombosis of the stents. All these features make the deployment of stents unsuitable in this patient or any patient with cardiogenic shock and possibly de novo acute myocardial infarction in general.

CONCLUSION

The strategy in the approach to an infarct-related cardiogenic shock scenario, we believe, must surely be a maximally aggressive one for properly selected patients. They should have emergency cardiac catheterisation with optimal inotropic support and intra-aortic balloon deployment if necessary. Mechanical revascularisation should be attempted as early as possible, preferably within 24 hours of infarction. Patients with single vessel disease should have attempted PTCA of the culprit lesion. Adjunct intracoronary lytic agent is strongly recommended if a large thrombus burden is apparent. Those with multivessel disease and amenable lesions may have staged PTCA, with initial dilatation of the infarct-related lesion. On the other hand, those with multivessel disease unsuitable for PTCA or the presence of significant left main coronary artery (infarcted or not) should be sent directly for emergency CABG. This approach probably offers the best chance for survival.

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