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Pericardiocentesis in patients on thrombolytic drugs

Summary. Pericardiocentesis is the most important procedure for cardiac tamponade. The causes of pericardial effusion and tamponade are many. Common causes are broadly infective or non-infective pericarditis, iatrogenic ones, trauma and neoplasm. This clinical picture may not be a frequently encountered cardiac emergency in ICUs, but it has always been a serious issue, demanding early detection. Promptness in diagnosis and expertise in pericardiocentesis are the key factors in this context. This article discusses cases where the cause is primarily a complication of thrombolytic drugs for treating ST-elevation myocardial infarction (STEMI). A high level of suspicion in patients with total haemodynamic stability and sudden onset hypotension or cardiogenic shock remains the single most important issue for early detection. In such situations, rapid diagnosis with echocardiography and immediate pericardiocentesis helps to save the patient's life. Cardiac tamponade is a grave complication in STEMI patients receiving either streptokinase or tPA (alteplase, tenecteplase) in centres where a cathlab facility is not available for primary percutaneous coronary intervention (PCI).
Keywords: Cardiac tamponade, pericardiocentesis, ST-elevation myocardial infarction (STEMI), thrombolytic drugs, streptokinase (SK), tissue plasminogen activator (tPA).

INTRODUCTION

Cardiac tamponade is a condition causing compression of the heart due to the accumulation of fluid within the pericardial space. The resultant effect is impaired diastolic filling and low cardiac output in relation to the increased intrapericardial pressure. Pericardial diseases of any aetiology may cause cardiac tamponade. Prompt recognition of cardiac tamponade is critical since the underlying haemodynamic disorder can lead to death if not resolved by drainage of the pericardium, mainly by way of pericardiocentesis.

The incidence of cardiac tamponade as a complication of thrombolytic therapy is not very common. However, it always remains a possibility due to major haemorrhagic complications of thrombolytic drugs in managing ST-elevation myocardial infarction (STEMI) cases, either with streptokinase (SK) or different tissue plasminogen activators (tPA).

During the era of thrombolysis for STEMI patients with streptokinase to the more efficient and effective drugs of different tPA such as alteplase (second generation) and tenecteplase (third generation), the risk of fatal haemorrhage is a realistic fear for all cardiologists. There are documented cases where fatal intracerebral haemorrhage deve-

loped after successful thrombolysis of myocardial infarction [1]. Despite these situations, the risk benefit ratio has always favoured the use of thrombolytics in well-selected cases of STEMI sufferers.

Although the focus of STEMI treatment has shifted from thrombolysis to primary percutaneous coronary intervention (PCI), a clear majority of patients do not get this benefit. PCI has significant logistic limitations of availability, accessibility and affordability. PCI for the treatment of acute STEMI in India is being practised in several centres. However, the availability of this treatment has been widely urban-centric and limited to those individuals who can afford it and those who are insured.

Thrombolytic therapy continues to be the most commonly used management strategy for acute STEMI in India and, indeed, in most parts of the world [2]. Lack of cathlabs in most of the healthcare centres where the patients' first present with myocardial infarction remains the single most important and compelling reason for thrombolytic use.

The other important issues are mainly delayed presentation at the emergency department (more than 90 to 120 minutes) and apathy towards motivation for interventional therapy (the cost difference being 3–5 times that of thrombolytic therapy in India). The complication of cardiac tamponade can be diagnosed promptly with the help of echocardiography at the new onset of symptoms. In contemporary clinical practice, echocardiography is the gold standard for diagnosis of cardiac tamponade [3, 4].

BACKGROUND

Pericardiocentesis is the aspiration of fluid from the pericardial space that surrounds the heart. This procedure can be life-saving in patients with cardiac tamponade, even when cardiothoracic surgery is not available. Cardiac tamponade is a time-sensitive, life-threatening condition that requires prompt diagnosis and management.

Historically, the diagnosis of cardiac tamponade was based on clinical findings. Claude Beck, a cardiovascular surgeon, described triads of clinical findings that he found associated with acute cardiac tamponade. These triads consisted of hypotension, an increased venous pressure, and a quiet heart. This has come to be recognised as Beck's triad, a collection of findings most commonly produced by acute intrapericardial haemorrhage. Subsequent studies have shown that these classic findings are observed in only a minority of patients with cardiac tamponade. The detection of pericardial fluid has been facilitated by the development and continued improvement of echocardiography. Cardiac ultrasound is now accepted as the standard imaging modality for the assessment of pericardial effusions and the dynamic findings consistent with cardiac tamponade. With echocardiography, the location of the effusion can be identified, the size can be estimated (small, medium, or large), and the haemodynamic effects can be examined. These haemo-

dynamic effects can be detected by assessing for abnormal septal motion, right atrial or right ventricular inversion, and decreased respiratory variation of the diameter of the inferior vena cava [5, 6].

Cardiac tamponade as a complication of pericardial effusion, haemorrhagic or non-haemorrhagic, with all its significance and relevance, has already been discussed in several peer-reviewed journals. The most significant and elaborative narrations are available, including the scoring system based on aetiology, clinical symptoms and imaging, regarding cardiac tamponade in the European Heart Journal in 2014 [7]. Furthermore, there are recent ESC guidelines published in 2015 for the diagnosis and management of pericardial disease. Herein is an excellent depiction of scoring and administration protocols based on scoring on aetiology (step 1), clinical presentation (step 2) and imaging (step 3). The cumulative score (the sum of steps 1, 2 and 3) when it is greater than 6 demands urgent and immediate pericardiocentesis, ruling out the contraindications and, when there is a score of less than 6, the same can be postponed for 12–48 hours with certain exceptions [8].

This is very pertinent in the context of the ongoing discussion of pericardial effusion and cardiac tamponade precipitated by drugs such as thrombolytics and anticoagulants. Although there is no specific claim as to the volume of pericardial fluid causing tamponade, it is known that a rapid collection is more prone to cause life-threatening tamponade. From 10 ml to 50 ml of fluid in the pericardial sac is insignificant and is usually present there. A rapidly accumulating effusion, such as in haemopericardium, may result in tamponade with as little as 100 to 200 ml of fluid [9]. On the contrary, a slowly increasing pericardial fluid accumulation may build up to one to one and a half litres over a longer period without any significant effect on the diastolic filling of the heart. This is due to the adaptive stretching of the pericardium over time. A compliant pericardium can allow considerable fluid accumulation without causing tamponade. Hence, the amount of pericardial fluid needed to impair diastolic filling of the heart depends on the rate of fluid accumulation and the compliance of the pericardium.

Regarding pericardiocentesis, echocardiographic measurements are also necessary.

Class I category includes cardiac tamponade and effusion more than 20 mm in diastole. In this ongoing discussion, it is being highlighted that thrombolytics and haemopericardium cause cardiac tamponade only as a primary complication in the treatment of STEMI patients.

CLINICAL PICTURE AND DIAGNOSIS

Cardiac tamponade, as a thrombolytic complication, is an exceptional case as far as the opening of the culprit vessel is concerned. The assessment is primarily on the parameters of effective thrombolysis, most notably complete relief of chest pain, the quantitative rise of cardiac en-

zymes by several fold, a decline of ST elevation more than 50 %, the appearance of reperfusion arrhythmias and so on. These patients are completely stable haemodynamically with absence of any symptoms. One common factor in all these cases is “anterior wall myocardial infarction”. The clinical picture changes from total comfort and a sense of wellbeing to sudden onset of shortness of breath (dyspnoea), compressive chest discomfort, tachypnoea, tachycardia, anxiety and profound weakness. It is known that raised jugular venous pressure, cardiomegaly and “pulsus paradoxus” are other notable features of pericardial effusion causing cardiac tamponade but only “pulsus paradoxus” (> 10 mmHg) remains a clue to the likelihood of tamponade [10].

The onset of all these features raises the suspicion of several possibilities other than pericardial effusion. The risk of both pleural and pericardial effusion causing cardiac tamponade is also a possibility due to the major bleeding complications of thrombolytic drugs. In such situations, pleural aspiration may be needed as the first choice in treating these cases [11], leaving pericardiocentesis a subsequent option. Also, the possibility of reocclusion of the culprit vessel or extension of myocardial damage may arise in the differential diagnoses. However, the development of illness and progressive decline of systolic blood pressure below 90 mmHg, and rapid and low-volume pulse demand thorough evaluation, including repeat ECG and echocardiography and a few other allied investigations. These symptoms typically happen 3–4 hours to 24 hours after successful thrombolysis [12].

On the appearance of new symptoms, the first ICU bedside test to be carried out is a fresh ECG for any further changes, but this may remain inconclusive. These patients after reperfusion should never normally complain of a recurrence of chest pain and therefore this may be an indicator of extension of myocardial damage or reocclusion. Clinical examination requires half-hourly blood pressure monitoring wherein the majority of patients show a progressive lowering of systolic blood pressure to the stage of cardiogenic shock. Bedside echocardiography can quickly diagnose pericardial fluid collection, mostly on the right side of the heart compressing the right atrium and ventricles with a dilated inferior vena cava (IVC).

Extreme alertness of the ICCU staff, as well as cardiac specialists, plays a vital role in saving lives. Expertise in carrying out the bedside procedure of echocardiography-guided pericardiocentesis must be available. The operator and team should have a thorough and complete knowledge of the possible complications. Minor to catastrophic complications result from the cardiac puncture – malignant arrhythmia (most commonly ventricular tachycardia), epicardial blood vessel puncture, liver puncture, lung injury and surgical emphysema, and so on.

The incidence of cardiac tamponade is two cases per 10,000 population in the USA. No such statistical data are available for India. Renkin et al.

reported four cases of cardiac tamponade, an incidence of 1 %. The experience of the author and that of Renkin suggest that this complication, although uncommon, may have the potential to occur more frequently than reported. Haemopericardium with tamponade should be considered in any individual who becomes hypotensive hours after receiving thrombolytics for acute MI [13].

TREATMENT

Prompt recognition of cardiac tamponade is critical since the underlying haemodynamic disorder can lead to death if not resolved by percutaneous or surgical drainage of the pericardium. Echocardiography-guided pericardiocentesis has a high success rate (> 95 %) with relatively little risk. The morbidity rate is approximately 1–3 %. Concerning the use of thrombolytic drugs in the acute phase of myocardial infarction, the following considerations must be taken into account.

The usual criteria of evaluation of the benefits of treatment are coronary artery patency, left ventricular ejection fraction and patient mortality. The severity of blood clotting abnormalities and the frequency of haemorrhage are used to assess the usual risk criteria of evaluation of the benefits of treatment. The benefit-risk ratio of thrombolysis in the acute stage of myocardial infarction indicates that mortality is the only indisputable criterion of assessment [14]. The mortality rate from injuries directly caused by the procedure is less than 1 % [15].

In spite of the favourable data for the procedure of pericardiocentesis for the management of cardiac tamponade, at times this can be challenging. The reason is that there is a lack of validated criteria for risk stratification to guide clinicians in the decision-making process. This situation mostly revolves around issues such as:

i. which patients need immediate drainage of the pericardial effusion,

ii. whether echocardiography is sufficient for the guidance of pericardiocentesis, or

iii. in cases of “haemodynamic shock” with a significant reduction in stroke volume and/or evidence of “pulsus paradoxus” (a condition when pericardial pressure is higher than 10–12 mmHg and in the presence of compression of the right heart chambers).

After establishing the diagnosis of cardiac tamponade in such cases and having adequate skills for the emergency therapeutic measure of pericardiocentesis, the challenge remains of further haemorrhagic complications for the procedure as the patients are all on multiple drugs, mainly dual antiplatelets and heparin. The presence of anticoagulants in the system remains as a continuous threat and grounds for apprehension. But despite relative contraindications, the demand for pericardiocentesis is the only choice for saving lives. It is appropriate to mention here that there are conflicting reports on a resuscitative measure of tamponade and shock.

The following approaches should be considered. Preferably, patients should be monitored in an intensive care unit. All patients should receive oxygen, volume expansion with blood, plasma, dextran, or isotonic sodium chloride solution, as necessary, to maintain adequate intravascular volume. Sagrista-Sauleda et al. noted a significant increase in cardiac output after volume expansion [16].

Bed rest with leg elevation may help increase venous return. Positive-pressure mechanical ventilation should be avoided because it may decrease venous return and aggravate signs and symptoms of tamponade. After pericardiocentesis, leave the intrapericardial catheter in place using a sterile procedure and attaching it to a closed drainage system via a 3-way stopcock. The catheter can be left in place for 1–2 days. Serial fluid cell counts can be useful for helping to discover an impending bacterial catheter infection, which could be catastrophic.

In the author's own experience of managing this type of case, fortunately no major complications occurred. The maximum fluid (haemorrhagic) volume among thirteen patients was 184 ml. In all cases, the standard subxiphoid approach was the easiest way for pericardiocentesis, and in one case it was a parasternal needle insertion in the left fifth intercostal space under continuous echo monitoring. There was an immediate improvement in symptoms as well as the return of normal haemodynamic status. Serious consideration was given, as part of standard protocol, to inserting and retaining a drainage tube for 24 to 48 hours or to discontinuing the antiplatelets and heparin.

In none of the cases did the author need to keep a drainage tube mainly to prevent ascending infection. The basis for this judgement was hourly monitoring of all vital parameters and, most importantly, repeated echocardiographic examinations for reaccumulation of pericardial fluid and the constant vigilance of expert members in doing repeat pericardiocentesis. In all the cases, the medical team was always prepared for the further challenge of repetition of the pericardiocentesis procedure. There were minor fluid collections but not to the extent of cardiac tamponade. Although myocardial rupture or perforation was a possibility, none of the patients developed this serious complication. A period of observation with repeat echocardiography helped us enormously to rule out this incredibly ominous condition resulting in haemopericardium.

The author and his medical team also did not have any case with pleuro-pericardial haemorrhagic effusions. Patients continue to receive dual antiplatelet therapy (DAPT) but not heparin in any form. The promptness in deciding the mode of treatment of pericardial effusion causing cardiac tamponade was the key factor in saving lives. In all these cases the patients were in the age bracket of 55 to 65 years and the cases were primarily supervised by the author during the last two decades.

CONCLUSION

The use of thrombolytic drugs is of immense value in treating STEMI patients in centres where PCI is not available. Successful thrombolysis enhances patient survival due to the restoration of antegrade flow in the infarct-related vessel and is essential to salvage the myocardium at risk. This improves ventricular function by limiting infarct size and preserving good LV function and reducing short-term morbidity and mortality.

Although there are different complications inherently related to STEMI cases, the possibility of cardiac tamponade is only linked to myocardial perforation or rupture. Encountering the life-threatening condition of cardiac tamponade as a complication of thrombolytic drugs is a real possibility, but this is rare. By ruling out other possibilities for deterioration after a well-managed STEMI with thrombolytics and other supportive drugs and care, the possibility of cardiac tamponade needs to be borne in mind by the treating cardiac team. In the event of any of the symptoms mentioned above, immediate bedside echocardiography and a cardiologist expert in performing pericardiocentesis should be available. A high degree of suspicion is what is required for such cases. In the author's experience of encountering 13 cases of cardiac tamponade out of nearly 300 STEMI cases (a percentage which may be approximately 4 % statistically), salvaging the patients' lives with the help of echo-guided pericardiocentesis in this low number of cases remains extremely gratifying.

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Проведення перикардіоцентезу пацієнтам, які приймають тромболітичні препарати

Резюме. Перикардіоцентез – найважливіша процедура в разі виникнення серцевої тампонади. Існує велика кількість причин перикардіального випоту та подальшої тампонади. Типовими чинниками є інфекційний або неінфекційний перикардит, ятрогенний перикардит, травма або новоутворення. Клінічну картину може й не так часто спостерігають у відділеннях інтенсивної терапії, але такі випадки завжди потрібно виявляти на ранніх етапах. Своєчасність діагностики та досвідченість у проведенні перикардіоцентезу є ключовими факторами в таких ситуаціях. У статті наведені випадки, де причиною серцевої тампонади є ускладнення після прийому тромболітичних препаратів для лікування інфаркту міокарда з елевацією сегмента ST. Підвищений рівень уваги до осіб із попередньою стабільністю гемодинаміки та раптовою появою гіпотензії або кардіогенного шоку є найважливішим заходом для ранньої діагностики. У таких ситуаціях швидке виявлення проблеми за допомогою ехокардіографії та негайний перикардіоцентез можуть зберегти пацієнтові життя. Тампонада серця – це серйозне ускладнення в пацієнтів з інфарктом міокарда з елевацією

сегмента ST, яким проводять тромболітичну терапію стрептокіназою або тканинним активатором плазміногену в медичних центрах, де немає обладнання та можливостей для проведення первинного коронарного втручання.

Ключові слова: тампонада серця, перикардіоцентез, інфаркт міокарда з елевацією сегмента ST, тромболітичні препарати, стрептокіназа, тканинний активатор плазміногену.

REFERENCES

1. Arzu J, Muqueet MA, Sweety SA, Iqbal AM, Kabir FI, Mahmood M. Haemorrhagic stroke after thrombolysis with streptokinase. Mymensingh Med J. 2014 Oct;23(4):818–20.

2. Iyengar SS, Godbole GS. Thrombolysis in the era of intervention. J Assoc Physicians India. 2011 Dec; 59:26–30.

3. Tsang TS, Oh JK, Seward JB, Tajik AJ. Diagnostic value of echocardiography in cardiac tamponade. Herz. 2000 Dec;25(8):734–40.

4. Tsang TS, Oh JK, Seward JB. Diagnosis and management of cardiac tamponade in the era of echocardiography. Clin Cardiol. 1999 Jul;22(7):446–52.

5. Gumrukcuoglu HA, Odabasi D, Akdag S, Ekim H. Management of Cardiac Tamponade: A Comparative Study between Echo-Guided Pericardiocentesis and Surgery-A Report of 100 Patients. Cardiol Res Pract. 2011;2011:197838.

6. Shlamovitz GZ. Pericardiocentesis. Medscape. 2015 Apr 17.

7. Ristić AD, Imazio M, Adler Y, Anastasakis A, Badano LP, Brucato A, Caforio AL, Dubourg O, Elliott P, Gimeno J, Helio T, Klingel K, Linhart A, Maisch B, Mayosi B, Mogensen J, Pinto Y, Seggewiss H, Seferović PM, Tavazzi L, Tomkowski W, Charron P. Triage strategy for urgent management of cardiac tamponade: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2014;35:2279–84.

8. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J, Brucato A, Gueret P, Klingel K, Lionis C, Maisch B, Mayosi B, Pavie A, Ristić AD, Sabaté Tenas M, Seferovic P, Swedberg K, Tomkowski W, Achenbach S, Agewall S, Al-Attar N, Angel Ferrer J, Arad M, Asteggiano R, Bueno H, Caforio AL, Carerj S, Ceconi C, Evangelista A, Flachskampf F, Giannakoulas G, Gielen S, Habib G, Kolh P, Lambrinou E, Lancellotti P, Lazaros G, Linhart A, Meurin P, Nieman K, Piepoli MF, Price S, Roos-Hesselink J, Roubille F, Ruschitzka F, Sagristà Sauleda J, Sousa-Uva M, Uwe Voigt J, Luis Zamorano J; European Society of Cardiology (ESC). 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2015 Nov 7;36(42):2921–64.

9. Goldman L, Schafer AI. Pericardiocentesis, in Goldman-Cecil Medicine, 2-Volume Set. 25th Edition. Amsterdam: Elsevier Health Sciences Division; 2015.

10. Roy CL, Minor MA, Brookhart MA, Choudhry NK. Does this patient with a pericardial effusion have cardiac tamponade? JAMA. 2007 Apr 25;297(16):1810–8.

11. Furst B, Liu CJ, Hansen P, Musuku SR. Concurrent pericardial and pleural effusions: a double jeopardy. J Clin Anesth. 2016 Sep;33:341–5.

12. Renkin J, de Bruyne B, Benit E, Joris JM, Carlier M, Col J. Cardiac tamponade early after thrombolysis for acute myocardial infarction: A rare but not reported haemorrhagic complication. J Am Coll Cardiol. 1991 Jan;17(1):280–5.

13. Mohammad S, Austin SM. Hemopericardium with cardiac tamponade after intravenous thrombolysis for acute myocardial infarction. Clin Cardiol. 1996 May;19(5):432–4.
14. Castaigne A, Duval-Moulin AM, Dubois-Randé JL, Merlet P. [Methods for evaluating thrombolytic drugs]. [Article in French]. Arch Mal Coeur Vaiss. 1990 Feb;83 Spec No 1:9–14.
15. Kumar R, Sinha A, Lin MJ, Uchino R, Butryn T, O'Mara MS, Nanda S, Shirani J, Stawicki SP. Complications of pericardiocentesis: A clinical synopsis. Int J Crit Illn Inj Sci. 2015 Jul–Sep;5(3):206–12.
16. Sagristà-Sauleda J, Angel J, Sambola A, Permanyer-Miralda G. Hemodynamic effects of volume expansion in patients with cardiac tamponade. Circulation. 2008 Mar 25;117(12):1545–9.

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