Case report - Cardiac general

Post-infarct ventricular septal defect following thrombolysis
in intracranial bleed

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Abstract

Mechanical complications after myocardial infarction are uncommon with advances in medical reperfusion strategies. However, such strategies are associated with bleeding complications which typically contraindicate surgical management. We describe a patient with a post-infarction ventricular septal defect and an intraventricular hemorrhage following thrombolytic therapy for an acute myocardial infarction.

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1. Introduction

Mechanical complications, such as post-infarction ventricular septal defects (PI-VSD), following acute myocardial infarction have become less common with advances in reperfusion strategies. However, such strategies including the use of thrombolytic therapy are not without bleeding risks. We present a case describing the management of a patient who not only developed a life threatening bleeding complication of thrombolytic therapy but also a similarly acute mechanical complication.

2. Case report

Our patient, a 74-year-old female, otherwise healthy except for a previously placed proximal LAD stent, who lived at home presented with an acute stent thrombosis. She underwent emergent thrombolytic therapy and cardiac catheterization. Cardiac catheterization, despite complete reperfusion, revealed an acute post-infarct anterior ventricular septal defect (PI-VSD) with a Qp:Qs of 2:1. Secondary to altered mental status requiring intubation a CT-scan of the head was obtained and revealed an intraventricular hemorrhage (IVH) (Fig. 1). She was then transferred to our institution for management. Upon arrival she was unresponsive. A ventriculostomy was placed by the Neurosurgical team which resulted in an improvement in her neurological status to which she was opening her eyes, following commands, and moving all extremities. Extensive discussion was held with the family regarding the fatal prognosis with non-operative management of her PI-VSD as increased efforts to improve cerebral perfusion pressures only worsened the pulmonary over-circulation. Despite extremely high operative risks and in light of her improving neurological status and baseline functional status, the family opted for surgical management. Following 48 h of stabilization including placement of an intra-aortic balloon pump, it was felt her risks for rebleeding had minimized and she was starting to develop end-organ and hemodynamic comprise as she was becoming more hypoxemic and oliguric with evidence of clinical hypoperfusion. She was taken to the operating room and a large anterior wall infarction and apical anterior PI-VSD was encountered. Full heparinization (300 U/kg, goal ACT: 480 s) was performed and standard cardipulmonary bypass with a heparin bond-ed circuit (Medtronic, Minneapolis, MN) and a conventional roller-pump system (Terumo System 1, Terumo Cardiovascular Systems, Ann Arbor, MI) was used including maintaining systemic pressures above 65 mmHg. The initial ACT was 165 and ranged from 457 to 824 while on bypass. The cerebral ventriculostomy was allowed to drain with intracranial pressures <10 mmHg at all times. A left ventriculotomyt was performed through the infarct medial to the LAD and a Gortex patch closure of the PI-VSD was performed and a secondary patch reconstruction of the anterior wall was used to close the ventriculotomy. A LIMA-LAD revascularization only was performed as she had no other coronary disease. She was weaned from bypass with minimal inotropic support. Following protomine sulfate (10 mg per 100 U heparin administered) the final ACT was 138 s. From a cardiovascular standpoint her postoperative course was unremarkable. Her neurological status slowly returned...
to the point in which she was purposefully moving all extremities and opening her eyes. Repeat head CT 10 days post-op showed resolution of her IVH (Fig. 2). Neurology and neurosurgical consultation felt optimistic about her potential for recovery. Unfortunately the family did not think that she would have wanted a prolonged rehabilitation including the possible need for tracheostomy and feeding tube and the decision was made to withdraw support. She was terminally extubated and provided comfort care only.

3. Discussion

Intracranial hemorrhage is a well known, and dreaded, complication of thrombolytic therapy occurring in 0.65% of treated patients with a 60% 30-day mortality rate [1]. However, early neurosurgical management of intracranial hemorrhage, when compared to medical management, is associated with improved 30-day survival (65% vs. 35%, \( P<0.01 \)) and tended towards more nondisabling outcomes (20% vs. 12%, \( P=0.15 \)) [2]. In similar studies PI-VSD, although less common with modern reperfusion strategies, still occurs in 0.2% of patients [3]. Each problem in itself is potentially lethal if left untreated, however, acute IVH, unlike an acute embolic stroke, is typically considered a strong contraindication to CPB. Although some patients present in a stable condition and can tolerate a period of close observation with delayed surgical management of their PI-VSD, early repair is advocated to limit end-organ damage as medical management is typically associated with 100% 30-day mortality vs. ~30% for those treated surgically [4]. The competing hemodynamic demands necessitating increased cerebral perfusion pressure versus the need for afterload reduction to minimize pulmonary over-circulation mandate an all or nothing approach to these complex problems. Although long-term survival and further neurological recovery would have been desirable, and potentially obtainable given her immediate postoperative course, our case illustrates the feasibility of performing complex cardiovascular procedures necessitating cardiopulmonary bypass in selected patients with intraventricular bleeding.

We used conventional bypass techniques and medications based upon experience and predictability – factors important in an inherently high-risk patient. Nafamostat mesilate, a synthetic protease kallikrein inhibitor has also been used for CPB in the setting of intra-cranial hemorrhage [5]. However, experience is limited and caution should be urged as Aprotinin, also a kallikrein inhibitor, has been removed from clinical use secondary to increased risks of complications.

4. Conclusions

In selected patients with intracranial hemorrhage and unstable cardiovascular problems a multi-disciplinary invasive approach may result in a favorable outcome provided families are prepared for a possible prolonged, and unpredictable, recovery. While such interventions are considered to be extremely aggressive, it is important to realize that there may be small glimpses of hope in potentially futile situations.

References

eComment: Complications of late thrombolytic therapy in the elderly

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We read with great interest the article by Firstenberg et al. [1] presenting a case of post-myocardial infarction (MI) ventricular septal defect (VSD) and intracranial hemorrhage following thrombolytic therapy after acute stent occlusion. We would like to comment the authors’ article with our modest experience of similar complications.

Myocardial or aortic rupture, aortic dissection, intracranial hemorrhage, or even splenic rupture are potential fatal complications of thrombolytic therapy following acute myocardial infarction. They usually occur in elderly (>65 years of age), moderate body weight (<70 kg) female subjects. Hypertension at presentation and usage of tissue plasminogen activator (t-PA) rather than streptokinase have shown to be additional risk factors. Of the most important, timing of the lytic therapy is accepted as the critical factor for post-MI VSD and intracranial bleeding. Initiation of the therapy beyond 12 h following the onset of MI is a major risk. Although the mechanism behind such complications following thrombolytic agents is not clear, it is suggested that recanalization with medical thrombolysis may lead to microvascular hemorrhage, interstitial edema and contraction band necrosis as well as to a decrease in fibrinogen and elongated partial thromboplastin times especially with the usage of t-PA [2–4].

At our institution, we have been faced with one post-MI VSD and another intracranial bleeding in two female patients aged 74 and 78 years who underwent thrombolytic therapy at 12 and 18 h of the onset of symptoms for the left anterior descending coronary artery occlusion related acute MI. Both patients were managed accordingly and successfully (unpublished data).

In order to better study the risks of such complications, it would be more appropriate if the authors could give detailed information about the blood pressure of the patient on admission and at the time of the intervention. Additionally, what was the time interval between the diagnosis and the initiation of the thrombolytic therapy?

We would like to congratulate the authors for their successful management in such a challenging case.

References