

Review Article

Management of Internal Mammary Artery Spasm

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OPEN ACCESS**Abstract**

The article is dedicated to the management of internal mammary artery spasm intra- and postoperatively based on the accumulated evidence in the literature. It provides stepwise decision algorithms for safely resolving the spasm and prevention of relapse.

Keywords

- Internal mammary artery spasm
- Coronary artery bypass grafting
- Vasodilation

INTRODUCTION

The left internal mammary artery (IMA) is the most widely used graft and is predominantly anastomosed to the left anterior descending artery (LAD), which is considered as gold standard in coronary artery bypass surgery (CABG) [1].

Vasoreactivity is a complex process, which presents in homeostatic equilibrium between constriction and dilation, and it is induced by a number of different neurotransmitters, cascades and receptors. IMA owns histo-anatomical features of a somatic artery and capable of undergoing severe spasm. It has been described in literature case reports that intractable spasm of IMA lead to transmural myocardial infarction and death [2]. The real incidence of this dangerous complication is unknown due to several reasons. One of them is the absence of an immediate angiographic study in each case where ischemic episode occurred. Second, when a patient develops postoperative myocardial ischemia, usually he gets treated empirically with nitroglycerin and taper down noradrenaline, which may relieve the spasm. Third, if it is ultimately decided to take the patient to the cathlab, and by the time the patient reaches there, the spasm may spontaneously resolve which results in normal angiography.

The first report about IMA vasospasm was by Sarabu M. et al in 1987 in which the authors described two cases of postoperative spasm successfully managed by intragraft application of vasodilators [3]. One year later, Kong et al described the angiographic presentation of IMA spasm in a patient who underwent CABG [4].

In order to prevent IMA spasm, different pharmacological agents have been used in the last 50 years. The most famous one is the solution of papaverine [5,6], which initially was proposed to be injected intraluminally. However, due to the fact that it adversely affects the endothelium because of acidity, presently it is applied topically only. Despite many other drugs available for spasm prevention, papaverine remains in use in the majority of cardiac centers.

Looking through the publications in recent years regarding the use of different vasodilators to prevent IMA spasm, we found that the results of many studies have controversial evidence. We would not mention all of them, but we will be summarizing some of the drugs used in daily practice such as nitroglycerin, calcium antagonists and sodium nitroprusside which have been firmly proven to be effective. The use of topical vasodilators also gained a lot of interest among investigators. A group of investigators from Turkey compared different vasodilators for topical use, and found that they cause approximately the same level of IMA vasodilation [7]. However, other investigators showed absence of dilatory effect of topical vasodilators [8-10]. This questions the need to prevent spasm when blood flow is adequate. It is important to remember that forceful vasodilation may lead to reciprocal increase in production and storage of endogenous constrictors in order to maintain homeostasis, what may cause vasospasm afterwards.

We would like to emphasize that this article is dedicated to the treatment of IMA spasm, not to prevention. Based on the available literature, we created an algorithm to manage IMA spasm intra- and postoperatively.

Management of IMA spasm

IMA spasm is a challenging condition. Its management starts from establishing the baseline condition of the patient. Comorbidities such as hypertension, diabetes, and dyslipidemia have significant impact in developing endothelium dysfunction and increased sensitivity for endogenous vasoconstrictors [11-13]. Experiments in vitro demonstrated that hypertension reduces the response to passive stretching of the IMA, increases basal tone, and impairs nitric oxide release [14].

Revision of preoperative medication chart may reveal a drug causing IMA vasoconstriction. Knowing the mechanism of its action, one can identify a potential antagonist to relieve the spasm. Here is a list of pharmacological groups with proven vasospastic effect on the IMA: alpha-adrenoreceptor agonists, beta-blockers,

acetylcholine antagonists, calcium, triptans, glibenclamide and noradrenaline.

It is also important to look for and treat any metabolic or electrolyte imbalances such as hypokalemia [15], hypercalcemia [16], hypomagnesemia, and hypoglycemia, which may contribute to the IMA spasm. An increase of serum lactate suggests a general vasospastic condition; and the presence of simultaneous vasospasm in several grafts and coronary arteries suggests a metabolic disorder rather than a local imbalance. The presence of left main coronary artery stenosis is an extremely unfavorable factor calling for immediate actions [17]. If hypokalemia is present, it has been reported that the use of diltiazem [18] and levosimendan [19] fast resolve IMA spasm due to the opening of potassium channels in the artery.

Intraoperative management

Intraoperatively, IMA spasm presents as reduced or absent blood flow in the graft. That is why it is recommended to dissect the artery right after harvesting to assure flow adequacy; otherwise it will be a waste of time to manage the spasm when the patient is already on cardiopulmonary bypass. Generally, it is not a dangerous condition, though may limit the use of IMA.

Here is stepwise algorithm for the management of intraoperative IMA spasm (Figure 1):

1. Along with the above mentioned revision of comorbidities, preoperative medications and verification of current metabolic status, it is recommended to cannulate the IMA and inject a warm solution of a vasodilator – sodium nitroprusside (SNP) alone [20] or “combination solution” of calcium channel blocker (verapamil, dihydropyridine [21]) and organic nitrate (nitroglycerin) [22].

It is important to maintain the temperature of administered fluids at 37 °C, since it has been shown that application of normothermic (37°C) saline solution is as effective as topical papaverine at room temperature [23]. We recommend the use of SNP or the “combination solution” because they are highly potent, widely available, and safe. Several authors reported that sodium nitroprusside is more potent than nicardipine, nicorandil, fenoldopam, hydralazine, adenosine, and labetalol [20,21]. Nevertheless, according to other authors the “combination solution” is as effective as sodium nitroprusside [22]. Intraluminal route is more preferable than topical or in-pedicle injection; and one should remember to check the IMA afterwards for possible dissection. No intraluminal papaverine should be injected as it adversely affects the endothelium because of acidity.

2. Skeletonization of the IMA abolishes sympathetic nervous system influence, dilates the artery and increases blood flow. This was proven by measuring flow before and after skeletonization [24], as well as the absence of sensitivity to noradrenaline in free IMA grafts [25].

3. IMA spasm may be diffused and limited. The distal segment of IMA is usually more involved in spasm as vasoconstrictive receptors are more expressed in distal direction [11,26]. If the length of IMA allows, we recommend to shorten it, and to see whether flow improves. If not, then mechanical dilation will be required.

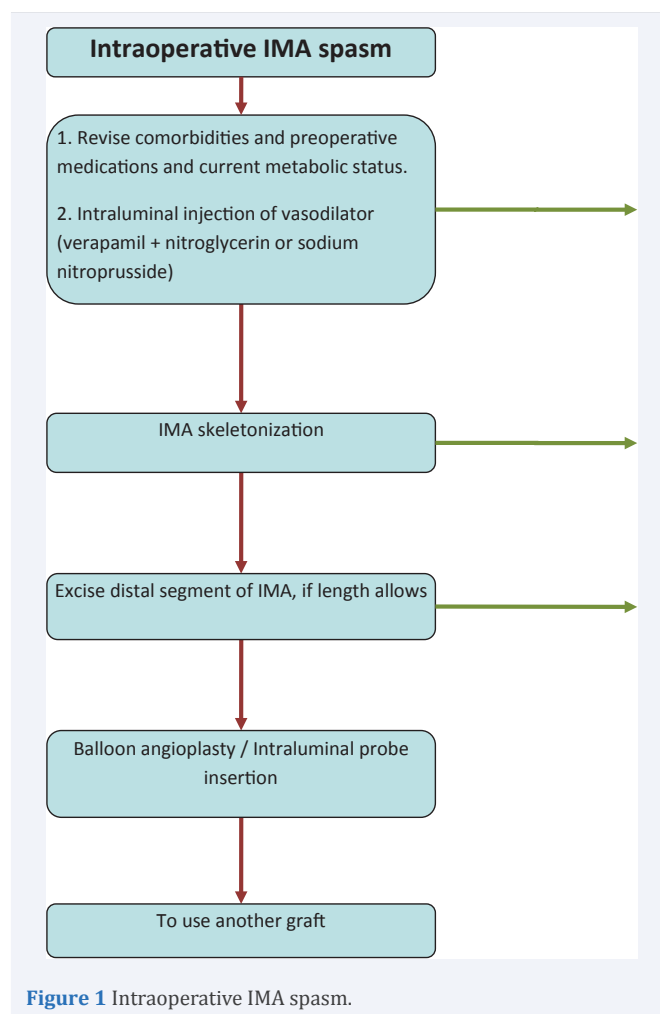


Figure 1 Intraoperative IMA spasm.

4. In our literature search, we found three reports about different atraumatic devices for routine dilation of the IMA [27-29]. Presently, they are not widely used due to the fact that they may injure the endothelium and thereby reduce the long term graft patency. The coronary angioplasty balloon may theoretically be inflated within the spasm area, though it has never been reported. The coronary steel probe introduction into IMA is a rough procedure and may be used only as a last resort. In our experience, we had two cases of reduced flow, where the probe perforated the IMA mid-segment.

5. If despite all the above measures, the IMA spasm persists, then it is time to consider using another graft.

When the spasm resolves (Figure 1), the IMA should be clamped by bulldog, not clipped. Flow should be refreshed and reassured every 20 min in order to remove degraded products from stagnated blood. The IMA should be kept in gauze soaked with warm solution of the above mentioned vasodilators.

Ultrasound flow measurement and photo dynamic eye system [30] may be useful tools to recognize spasm developed after the anastomosis is performed, and to treat the spasm accordingly avoiding unnecessary intervention [30]. Bolus administration of calcium chloride should be avoided after separation from CPB

as it may significantly reduce flow and triggers vasospasm of the IMA [16].

Postoperative management

IMA spasm rarely occurs in the postoperative period, and is characterised by ST-segment elevation in the grafted territory, rising cardiac enzymes, hemodynamic instability, and cardiac arrest. The best diagnostic method of IMA spasm is a coronary angiography where a contrast filling defect during spasm should be distinguished from atherosclerotic plaque [31] and kinking (particularly in a long or twisted vessel).

Spasm of the IMA usually occurs during the first hours postoperatively as a reaction for the surgical intervention. Nevertheless, a case of IMA spasm shown on angiography has been reported in a patient with classical angina four years postoperatively [32].

In the same manner, we created a stepwise algorithm for the management of postoperative IMA spasm: (Figure 2)

1. If coronary angiography shows evidence of IMA spasm, it means that the spasm is sustained and serious, and so prompt actions are required. The first action is injection of warm (37 °C) solution of SNP or "combination solution" into the IMA [3,15,20,21,33]. Together with this, it is necessary to revise current medications, inotropes, to check electrolytes, lactate, and glucose with subsequent rectification. These measures exhibited efficacy in a number of reports, though may be insufficient.

2. Next step would be establishing cardiocirculatory support, as it was reported that early institution of extracorporeal membrane oxygenation and controlled cardiocirculatory assistance significantly improves survival, especially in cases where IMA spasm associated with spasm of other grafts or coronary arteries [17, 34]. IMA spasm itself is matter of several hours and circulatory support is an insurance which helps to overcome short ischemic period [33]. Once patient's hemodynamic stability secured, further interventions can be done.

3. IMA angioplasty/stenting is a peculiar procedure and has been successfully performed in IMA spasm [35], stenosis and anastomotic stricture. Lack of experience for such interventions or diffuse spasm are limitations for latter.

4. Stenting of coronary artery (usually LAD) grafted by IMA could be an alternative, which helps to avoid myocardial infarction, though may create competitive flow. Interesting case was described by Pratt et al. where at the patient with retractable IMA spasm instead it was stented critical left main stenosis. At three months angiographic follow up was revealed patent IMA with occluded left main trunk [36].

5. If despite of all measures patient still has signs of ongoing ischemia due to the IMA spasm, it is necessary to exchange graft and preferably to do it soon. Vogt et al described a case with distal IMA spasm. Initially it was relieved by nitroglycerin intro IMA injection, but because of respasm they did redo bypass using vein graft, by this prevented myocardial infarction [37].

When spasm relieved it is important to maintain vessel patency administrating dilators and avoiding constrictors. Here

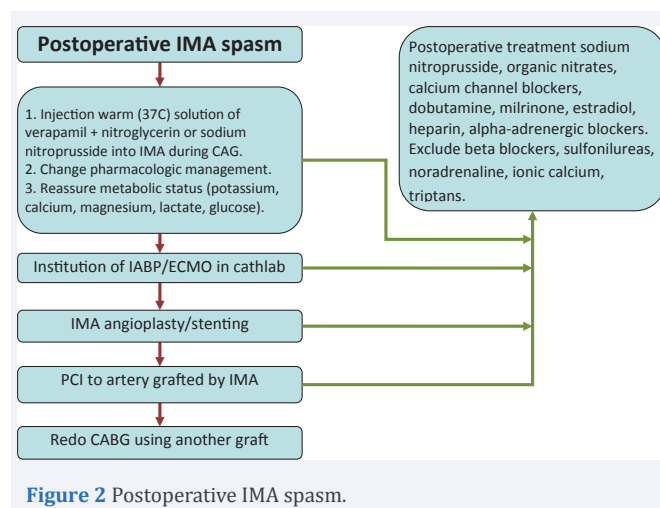


Figure 2 Postoperative IMA spasm.

is a list of drugs that cause IMA dilation: sodium nitroprusside [21], organic nitrates [38], calcium channel blockers [3], 17beta-estradiol (evidence only for females) [39], natriuretic peptides [40], low molecular weight heparin [41], alpha-adrenergic blockers (at benign prostate hyperplasia), resveratrol [42], iloprost [43], fasudil [44].

Inotropic drugs are routine in post cardiac surgery treatment and has strong impact on IMA vasoreactivity. Among of all drugs dobutamine appears to be superior to others in terms of IMA dilation [45]. Levosimendan [46-48] and milrinone [49-52] exhibited moderate dilatory effect through some different biochemical pathways. Adrenaline is influencing on IMA unpredictably, due to stimulation several antagonizing receptors. Noradrenaline in turn is a potent vasoconstrictor and generally should be rejected or reduced.

Regards diabetic patients sulfonilureas should be replaced to other hypoglycemic agents, such as thiazolidinediones [51] which displayed soft dilatory properties. Beta blockers may be substituted to ivabradin, or at least to nebivolol or carvedilol with their preferred additional vasodilatory effects.

COMMENTS

IMA spasm is a complication that precludes use graft intraoperatively and causes myocardial infarction postoperatively [2, 34]. Many techniques and drugs have been introduced to deal with this complication. Initial studies compared ability of different pharmacological agents to dilate IMA assuming spasm as a single self-limited process. Later it was understood that vasoreactivity is multifactorial process triggered via various metabolic pathways. That became the basis to study influence of dilators after precontraction IMA with known vasoconstrictive mediator. Results of these studies helped to choose proper drug depending on the situation. For the moment in a human being it has been identified more than ten endogenous mediators and almost to each of them discovered particular antagonistic drug.

There are more than hundred reports in current literature dedicated to pharmacological prevention IMA spasm. However until now there is no unified consensus about using exact medicament, its dosage and route of administration. Still the question remains unanswered whether prevention is required in

general [9]. What is interesting is that some drugs have additional vasoprotective properties what may improve long-term graft patency. Given the fact that intraoperatively grafts are passing through short period of ischemia between harvesting and being anastomosed during which is launching pathological cascades adversely affecting endothelium, adequate vasoprotection is preferable to preserve graft viability. For instance, it is known facts that early postoperative aspirin intake increase long-term patency and at the same time preoperative aspirin intake is avoided due to the risk of bleeding. Thus, it would be interesting to investigate long-term patency IMA with intraoperative topical application of aspirin. In the same manner could be studied other topical protector-dilators as natriuretic peptides, iloprost, revestrol etc.

Diagnosed IMA spasm is relatively rare complication and only few reports available in literature. It begs for more information in order to work out adequate treatment.

CONCLUSION

Intraoperatively IMA spasm limits its use, where adequate measures may resolve spasm retaining IMA as a preferred graft. Postoperative IMA spasm is highly lethal and unfamiliar condition, which requires prompt actions in order to avoid complications. We propose stepwise algorithms for intra and postoperative management that can serve as a guide, but does not call for absolute obedience, as every case still requires an individual approach.

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