International Journal of Research in Medical Sciences
*Vaidyanathan S et al. Int J Res Med Sci. 2017 Sep;5(9):4191-4194*
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20174011

**Case Report**

# Vasoplegic syndrome after off pump coronary artery bypass grafting surgery

**Swaminathan Vaidyanathan[1], Kumar Chidambaram[2], Anbarasu Mohanraj[1]***

[1]Department of Cardiac Surgery, Madras Medical Mission, Chennai, Tamil Nadu, India
[2]Department of Anaesthesiology, Madras Medical Mission, Chennai, Tamil Nadu, India

**Received:** 13 July 2017
**Accepted:** 09 August 2017

***Correspondence:**
Dr. Anbarasu Mohanraj,
E-mail: anbarasu.mohanraj@gmail.com



## ABSTRACT

We report a case of 48-years-old man who developed nor-epinephrine resistant vasoplegic syndrome after elective off pump coronary artery bypass grafting surgery. The failure of norepinephrine to improve the patients' hemodynamics promted us to start treatment with vasopressin. Within an hour, the hemodynamics begin to improve. After 4 hours, patient was stable enough to be weaned from vasopressin, extubated at the end of 24 hrs and shifted to ward at the end of 48 hours. Vasoplegic syndrome can occur commonly after cardiopulmonary bypass and incidence can go up to 10%. This case was reported to insist upon the fact that vasoplegic syndrome can occur even after off pump surgeries. Herein we discuss the aetiology, risk factors, pathophysiology and prophylactic measures to be taken, for the prevention of vasoplegic syndrome.

**Keywords:** Cardiac surgery, CABG, Cardiopulmonary bypass, OPCAB, Vasoplegia, Vasopressin

## INTRODUCTION

Vasoplegic syndrome is one of the dreaded complication that can occur in postoperative cardiac surgery patients. It is characterized by severe and persistent form of hypotension (mean arterial pressure<50 mm hg), tachycardia, normal or increased cardiac output (cardiac index>2.5l-min-m~2), decreased systemic vascular resistance (<800 dyne-sec-cm~5), low filling pressures and is poorly responsive or unresponsive to volume infusion.[1,2]

Earlier it was completely attributed to cardiopulmonary bypass, but off late various other risk factors have been identified for the development of vasoplegic syndrome.[1] The incidence of vasoplegic syndrome following cardiac surgery ranges from 5 to 10%.[3,4] Mortality rate of vasoplegic syndrome can go up to 25% following cardiac surgery.[5,6] We report a patient who developed vasoplegic syndrome after off pump coronary artery bypass grafting surgery.

## CASE REPORT

A 48-years-old man with history of posterior-inferior wall myocardial infarction was admitted to our institution for elective coronary artery bypass grafting surgery. He was euglycemic and normotensive. Electrocardiogram showed changes suggestive of old inferior wall Myocardial infarction. Echocardiography revealed mild LV systolic dysfunction (ejection fraction=42%) with regional wall motion abnormality of corresponding inferior-posterior wall. Coronary angiography revealed double vessel disease with 95% stenosis of left circumflex artery and 70% stenosis of proximal left anterior descending artery. His preoperative medications were, Tab. Rozuvastatin 20 mg od, Tab. Ramipril 5 mg

od, Tab. Metoprolol 12.5 mg bd, Tab. Nicorandil 10 mg bd.

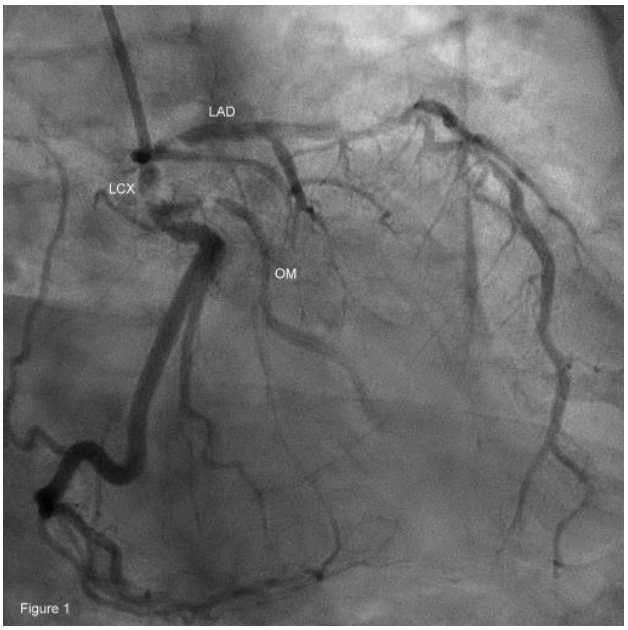


**Figure 1: Coronary angiogram.**

After multidisciplinary team meeting, he was taken up for elective surgery. A peripheral venous canola, radial arterial line, central venous line, and urinary cannula catheter were inserted. The anaesthetic technique included administration of inj fentanyl 250 mcg, inj midazolam 3 mg and inj vecuronium 8 mg intravenously with controlled mechanical ventilation, maintained by isoflurane. Heparin was administered at a dose of 1.5mg/kg and activated clotting time was found to be 640. Double vessel total arterial revascularization was performed off-pump by anastomosing pedicle left internal mammary artery to left anterior descending artery and right internal mammary artery to obtuse marginal (OM) artery as LIMA-RIMA-Y to OM. Both the target vessel is about 2 mm size, good quality vessels and conduits were about 1.6 mm and of good quality. At the end of the procedure, during protamine (1:1 ratio) administration patient developed gradual hypotension, tachycardia, which were initially treated unsuccessfully with fluid infusion. There were no ST segment changes.

Patient was started on inj noradrenaline at 0.05 mics/kg/min and was increased to 0.1 mics/kg/min to combat hypotension. Inj adrenaline @ 0.05 mics/kg/min and inj dopamine @ 5 mics/kg/min was also started. As the hemodynamics were not improving intra-arterial blood pressure catheter (IABP) was inserted. An option of reopening the chest to look into the position and lie of grafts and to redo the grafts was considered but deferred due to absence of ST changes, good left ventricular function with no obvious regional wall motion abnormality on trans esophageal echocardiography.

Flow track monitor inserted at this point revealed a Systemic vascular resistance of 248 dynes-sec-cm~5; CO 8.2L/min. Mean arterial pressure was around 50 to 55 mm of hg and heart rate was 90-100 beats / min, with severe metabolic acidosis. So, provisional diagnosis of norepinephrine resistant vasoplegia was made and vasopressin infusion was started at 2 Iu/hr. Within 30 min of starting vasopressin infusion, improvement in hemodynamics was noticed and patient was shifted to intensive care unit.

Over the next 4 hours, SVR increased to 903 dyne-sec-cm~5, C.O. stabilized to 6 l/min, and mean arterial pressure increased to 90-100 mm hg. Urine output was also good (maintained more than 1 ml/kg/minute). The associated metabolic acidosis and electrolyte imbalance was appropriately managed. Vasopressin infusion was tapered at 0.5 iu/hour and stopped after 4 hours. Nor epinephrine was also gradually tapered.

Patient was extubated and weaned off from IABP 24 hrs after the surgery. Chest drains were removed on 1st post op day. Patient was transferred to ward on 3rd post op day after tapering and stopping all the supports. He was discharged on 7th postoperative day. He was asymptomatic at 6 months follow-up.

## DISCUSSION

The aetiology of vasoplegic syndrome is multifactorial. It is mainly attributed to systemic inflammatory response activated specifically to cardiopulmonary bypass. However the emergence of vasoplegic syndrome in off pump cases leads to theorize other causal factors such as surgical stress, use of rasterized disposable devices, neutralization of heparin resterlized with protamine, transfusion of blood products, hypothermia, preoperative use of angiotensin converting enzyme inhibitors, occurrence of endotoxemia secondary to repeated episodes of hypotension throughout off pump surgeries as a result of mobilization and displacement of heart, preoperative cardiac failure with low ejection fraction.[1,7]

Fundamentally vasodilatation is due to inappropriate activation of the vasodilator mechanism and failure of vasoconstrictor mechanism in vascular smooth muscle despite high plasma catecholamine levels and activation of renin-angiotensin mechanism (R.A.S). Basic mechanisms implicated are,

- Activation of potassium channels which are adenosine triphosphate activated and calcium regulated. This occurs in the membrane of vascular smooth muscle
- Impairment of arginine vasopressin (AVP) system
- Increased level of nitric oxide (NO), due to activation of inducible form of NO synthase.[4]

Therefore, vasoplegic syndrome has been correlated with the release of vasodilator inflammatory mediators, AVP

system impairment, endothelial dysfunction, decreased myogenic reactivity due to catecholamine despite enhanced catecholamine release, and cytokines such as tumour necrosis factor (TNF) and interleukin 1 (IL 1) associated with increased NO production, resulting in marked relaxation of vascular smooth muscles.

Under normal physiological circumstances blood pressure is maintained by three separate systems, 1- sympathetic system, 2- RAS 3- vasopressinergic system. Most anaesthetic drugs reduce the influence of sympathetic system on cardiovascular tone. Therefore, under general anaesthesia there is believed to be an increased reliance on RAS and vasopressinergic system to maintain blood pressure. RAS antagonist such as angiotensin converting enzyme (ACE)inhibitors and angiotensin receptor blockers (ARB) block the RAS response to hypotension. Therefore, patients taking these agents have an increased risk of refractory hypotension during general anaesthesia. Tuman et al prospectively examined the influence of chronic preoperative ACE inhibitor use and other perioperative factors on the incidence of vasoplegic syndrome after cardiac surgery and they concluded that preoperative ACE inhibitor use is an independent risk factor.[8] Our patients were also on chronic preoperative ACE inhibitor therapy.

Occasionally onset of vasoplegic syndrome have been perceived soon after blood products transfusion. `Blood transfusion is known to activate polymorphonuclear leukocytes and cytokine release. It is known that protamine neutralization of heparin may cause circulatory side effects mediated via complement activation, histamine release. Protamine causes activation of leukocytes and increased blood levels of TNF alpha that result in an increased NO production and this leads to contractile dysfunction and depression of cardiac function. W.J. Gomes et al, reported four patients with vasoplegic syndrome after off pump surgery, similar to our patient and they noticed that increase in the level of TNF-alpha in one of their patient and they postulated that it may be a likely mediator to this syndrome.[1]

Management of vasoplegic syndrome is controversial. But treatment is based on use of drugs with vasoconstrictor effect. The currently used drugs are catecholamines with adrenergic alpha effects such as norepinephrine, Vasopressin and methylene blue. Our patient did not respond to norepinephrine but improved promptly to vasopressin. There have been many reports suggesting starting vasopressin and methylene blue as first line of drugs to vasoplegic syndrome. Papadopoulos et al reported low dose infusion of vasopressin in perioperative period is beneficial in prevention and treatment of post cardiotomy vasoplegic syndrome whereas Mehaffey et al concluded that early administration of methylene blue improves survival and reduces the risk of major adverse events in patients with vasoplegic syndrome following cardiac surgery.[4,9]

## CONCLUSION

Vasoplegic syndrome is an unusual complication after off pump cardiac surgery. Many factors influence the development of Vasoplegic syndrome and the role played by these factors and the extent to which they affect the pathophysiology of vasoplegic syndrome gives a complex integrated picture. For this reason, knowing the preoperative factors (for example preoperative use of ACE inhibitors) and intra operative factors (blood transfusion, hypothermia, protamine administration, causing susceptibility to vasoplegic syndrome is essential). Taking precautions against these factors helps to combat this condition.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

## REFERENCES

1. Gomes WJ, Erlichman MR, Batista-Filho ML, Knobel M, Almeida DR, Carvalho AC, et al. Vasoplegic syndrome after off pump coronary artery bypass surgery. Eur J Cardiothorac Surg. 2003;23:165-9.
2. Argenziano M, Chen JM, Choudhri AF, Cullinane S, Garfein E, Weinberg AD, et al. Management of vasodilatory shock after cardiac surgery: identification of predisposing factors and use a novel pressor agent. J Thorac Cardio-vasc Surg. 1998;116:973-80.
3. Levin RL, Degrange MA, Bruno GF, Del Mazo CD, Taborda DJ, Griotti JJ, et al. Methylene blue reduces mortality and morbidity in vasoplegic patients after cardiac surgery. Ann Thoracic Surg. 2004;77(2):496-9.
4. Papodopoulos G, Sintou E, Siminelakis S, Koletsis E, Baikoussis NG, Apostolakis E. Perioperative infusion of low dose vasopressin for prevention and management of vasodilatory vasoplegic syndrome in patients undergoing coronary artery bypass grafting-a double blind-randomized study. J Cardiothorac Surg. 2010;5:17-28.
5. Gomes WJ, Carvalho AC, Palma JH, Teles CA, Branco JN, Silas MG, Buffolo E. Vasoplegic syndrome after open heart surgery. J Cardiovasc Surg. 1998;39:619-23.
6. Mekontso-Dessap A, Hauel R, Soustelle C, Kirsch M, Thebert D, Loisance DY. Risk factors for post cardiopulmonary bypass vasoplegia in patients with preserved left ventricular function. Ann Thorac Surg. 2001;71(5):1428-32.
7. Ohri SK, Becket J, Brannan J, Keogh BE, Taylor KM. Effects of cardiopulmonary bypass on gut blood flow, oxygen utilization and intra-mucosal Ph. Ann Thorac Surg. 1994;57:1198-9.
8. Tuman KJ, McCarthy RJ, O'connor CJ, Holm WE, Ivankovich AD. Angiotensin-converting enzyme inhibitors increase vasoconstrictor requirements

after cardiopulmonary bypass. Anesthesia Analgesia. 1995;80(3):473-9.
9. Mehaffey JH, Johnston LE, Hawkins RB, Charles EJ, Yarboro L, Kern JA, et al. Methylene blue for vasoplegic syndrome after cardiac operation: early administration improves survival. Ann Thoracic Surg. 2017;20(2):178-81.

**Cite this article as:** Vaidyanathan S, Chidambaram K, Mohanraj A. Vasoplegic syndrome after off pump coronary artery bypass grafting surgery. Int J Res Med Sci 2017;5:4191-4.