



Case Report

Successful CPR in a Patient with Left Main Coronary Stenosis and Severe Aortoiliac Disease Following Intubation

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Abstract: The incidence of pre-bypass ischemia in patients undergoing coronary artery bypass grafting ranges between 10% to 50% [1]. We report in this paper a successful cardiopulmonary resuscitation (CPR) for a 75-year-old male patient with left main coronary disease who developed an asystolic cardiac arrest during induction, immediately after laryngoscopy and intubation. The arrest was managed by an immediate CPR and a prompt initiation of cardiopulmonary bypass. The patient had a very smooth and uneventful post operative course and was discharged from our institution 5 days after surgery without any organ damage. We discuss in this case report the factors that may have precipitated the arrest as well the elements -barbiturates, immediate CPR and tepid hypothermic bypass- that may have contributed to the excellent outcome.

Keywords: Left Main Coronary Disease, Laryngoscopy, Cardiac Arrest, CPR, Trepid Hypothermic Bypass

1. Case Report

A 75-year-old man (whose consent was obtained) was admitted to the hospital for elective catheterization following a brief episode of chest and jaw pain three days prior to admission. He is known to be dyslipidemic, diabetic on oral medications, mildly hypertensive, has a severe aortoiliac disease with total occlusion of the distal abdominal aorta and is a heavy smoker (50 py). He also has a glaucoma on eyes drops.

All lab values were within normal range but coronarography revealed severe left main stenosis (80%). The cardiac echography showed a normal ejection fraction with no wall motion abnormalities. He was transferred to Coronary Care Unit (CCU) for continuous monitoring. Small dose of Beta Blocker (Bisoprolol 2.5 mg/d) and 24000 U/d of heparin were started. Note that the patient was clinically and hemodynamically stable throughout his hospital stay. A

coronary artery bypass graft (CABG) was scheduled the next morning.

Premedication was achieved by Glycopyrrolate 0.2 mg, Promethazine 25 mg and Morphine 7 mg intra muscularly on call to the operating room (OR). Upon arrival to the operating theatre, he was calm but had a relative high blood pressure (170/80 mm Hg). Two peripheral intra venous lines and a left radial artery cannula were inserted. The insertion of an intra aortic balloon pump (IABP) was not considered because of the patient's known occlusion of the aorta. He was preoxygenated with 100% oxygen for 3 min, induced by Midazolam (0.05mg/kg), Thiopentone (4mg/kg), Sufentanyl (0.75 µg/kg) and the neuromuscular blockade achieved by Rocuronium (0.7 mg/kg).

Following induction the heart rate dropped from 90 to 65 beats per min (bpm) and the blood pressure from 170/80 to

120/60 mm Hg. During laryngoscopy and intubation which was achieved only after the second trial using a stylet and despite deepening the anesthesia level by supplemental dose of opioids (Sufentanyl 0.3 µg /kg) and Midazolam (0.02mg/kg), the blood pressure rose to 190/80 mm Hg and the heart rate to 120-130 bpm. Thirty seconds after the catecholamine response the patient became unstable, the blood pressure dropped to 70 mm Hg and was refractory to vasopressors (boluses of phenylephrine 100 micrograms then epinephrine boluses of 10 micrograms). A minute later the patient went into asystolic cardiac arrest. Immediate CPR was started generating a blood pressure above 90 mmHg. The chest was opened immediately and the external cardiac compressions-that were discontinued for less than one minute-were replaced by internal cardiac massage. Heparin was administered intravenously at 4 mg/kg. The aorta and the atrium were cannulated and cardiopulmonary bypass (CPB) was initiated 20 min after the onset of CPR. Maintenance of anesthesia was achieved by an infusion of Midazolam (0.2 µg/kg/min) Sufentanyl (0.6 µg/kg/hr) and Cisatracurium (0.15mg/kg/hr) and Sevoflurane 1%. Prophylactic doses of antibiotics (Zinacef 1.5g and Vancomycin 1g) were given intravenously.

After initiation of CPB, perfusion pressure was maintained at 70-80 mm Hg, with a blood flow of 4.5 to 5 L/min and a Hematocrit at 29%. A tepid hypothermia was induced and the mixed venous blood temperature dropped to 33.5°C. Two vein grafts to the left anterior descending (LAD) and circumflex were completed. A gentle rewarming was initiated, the aortic cross clamp removed (duration 35 min). The bypass support of 1hr 32 min was successfully discontinued without the use of any inotrope.

The chest was closed. A central line, Swan Ganz and a Foley catheter-which are usually placed after induction of anesthesia - were inserted at the end of the procedure because of the urgent opening of the chest. All the hemodynamic parameters were within normal range and the patient was transferred to the Coronary Surgical Unit (CSU).

The post operative course was uneventful and the patient was discharged five days after the procedure without any neurological sequel nor organ damage.

2. Discussion

The incidence of pre-bypass ischemia in patients undergoing CABG ranges between 10 to 50% [1]. Although most of ischemic events are not associated with hemodynamic changes, some are. The occurrence of tachycardia, hypertension and increase in left ventricular filling pressure jeopardize the oxygen supply demand relationship and reduce the Coronary Perfusion Pressure (CPP) resulting in ischemia and cardiac arrest.

Many factors may have precipitated ischemia then asystole in our case, although the patient was stable upon arrival to the operating theatre. In fact the morning dose of Bisoprolol was missed which explains the exaggerated increase in heart rate during laryngoscopy despite administering the usual dose

(normal) of anesthetics. In addition the patient was obese and had relative difficult airways (Mallampati class II-III). The trachea was intubated after the second trial using a stylet which increased the stressful period preceding intubation. Unfortunately anesthetic drugs were not able to blunt solely the catecholamine response.

Although the patient had a severe left main stenosis, an IABP was not inserted prior to induction, because of his known history of distal aortic occlusion.

Many papers discussed the role of the IABP prior to invasive cardiac procedures or cardiac surgery. Most of the authors recommend its use especially in unstable cardiac patients with severe lesions. The IABP will provide better blood flow in arteries that are not severely diseased and in collateral circulation. It will also decrease oxygen demand by decreasing afterload.

However, kern found that the IABP will have a minimal role in improving blood flow in arteries with severe stenosis. He concluded that in stable patients with critical lesions, an optimal medical preparation prior to cardiac surgery and a careful induction of anesthesia are sufficient in achieving a good outcome [2].

Although our patient went into cardiac arrest immediately after intubation and despite a relative long CPR of 20 min, he had a spectacular smooth post operative course and was discharged from hospital 5 days after surgery. This excellent outcome may be attributed to many factors; the asystolic cardiac arrest occurred after intubation and ventilation with 100% oxygen which increased the total body oxygen stores prior to arrest [3]. In addition immediate and vigorous CPR was launched and followed by prompt initiation of CPB. These factors helped preventing prolonged and pronounced global organ hypoperfusion and ischemia throughout this critical period.

On the other hand, the excellent neurological outcome may be related to the protective agents and methods used in our case such as administration of barbiturates, benzodiazepine and induction of hypothermia. The role of thiopentone in providing cerebral protection in global ischemia has been debatable. It may have a possible protection effect by reducing cerebral blood flow, volume, pressure and edema, by decreasing oxygen consumption and by acting as a free radical scavenger especially if used in high doses. Although a relative small dose was given to our patient, a probable cerebral protection effect was observed because it was combined to hypothermia and benzodiazepines [4, 5, 6].

The use of the classic cannulation site may have had a favorable impact too. In fact the surgeon elected a direct ascending aortic cannulation and not a femoral one because the patient had a severe aortoiliac disease. Although the femoral cannulation is usually used in such emergencies allowing a relative rapid cannulae insertion without interrupting external chest compressions, it has possible complications such as cerebral embolization and organ malperfusion caused by retrograde blood flow [7, 8]. These probable adverse events were avoided in our case because an aortic cannulation was rapidly achieved. Resuscitation

throughout this critical period was sustained by replacing external chest compressions with an efficient internal cardiac massage.

Finally hypothermia which is becoming an important therapeutic tool in advanced life support cases has been applied. The patient core temperature was dropped to 33.5°C followed by a gentle re-warming. This may have contributed to cerebral protection during cardiac arrest [9].

Few similar cases of post induction cardiac arrest followed by a good post operative course have been described in the literature. Dragan Mijuskovic *et al* reported a successful resuscitation in a female patient with critical aortic stenosis, severe mitral regurgitation and left main stenosis. She developed two cardiac arrests the first one during coronarography and the second after induction of anesthesia. The patient was successfully resuscitated but the post operative course was complicated by a cardiac tamponade then by a transient renal and liver failure which necessitated a prolonged hospitalization for 33 days [10].

3. Conclusion

Most if not all cardiac patient particularly the ones with critical lesions must be scrupulously prepared in the perioperative period (rigorous control of blood pressure and heart rate...) especially if an IABP cannot be inserted or is not available. Beta blockers which are recommended in high risk patients by the ACC/AHA [11] may help preventing any cardiac event throughout induction, laryngoscopy or any stressful procedure especially that the anesthetic drugs cannot block solely any catecholamine response which can be deleterious in such cases.

If cardiac arrest occurs a rapid and efficient management is mandatory because the prognosis and the outcome are dramatically related to an immediate and vigorous CPR, use of medications known to have cerebral protection effect and a prompt initiation of a tepid cardiopulmonary bypass.

References

- [1] Kaplan J, Wynands J: Anesthesia for myocardial revascularization in Cardiac Anesthesia ed (4), WB Saunders company, 1999. pp 715.
- [2] Kern M: Your patient has critical left main stenosis. Do you need an intra aortic balloon pump. Cath lab digest 16 (1), 2008.
- [3] Sirian R, Wills J: Physiology of apnea and the benefits of preoxygenation. Continuing Education Anesthesia, Critical care and Pain 9 (4): 105-108, 2009.
- [4] Tan PSK: The anesthetic management of circulatory arrest. Br J Hosp Med 43: 38, 1990.
- [5] Thomas AN, Anderton JM, Harper NJN: Anesthesia for the treatment of a giant cerebral aneurysm under hypothermic circulatory arrest. Anesthesia 45: 383, 1990.
- [6] Quasha AL, Tinker JH, Sharbrough FW: Hypothermia plus Thiopental: Prolonged encephalographic suppression. Anesthesiology 55: 636, 1981.
- [7] Kamiya H, Kallenbach K, Halmer D *et al*: Surgery for aortic disease. Comparison of ascending aorta versus femoral artery cannulation for acute aortic dissection type A. Circulation 120: S282-S286, 2009.
- [8] Van Arsdall GS, David TE, Butany J: Autopsy in acute type aortic dissection. Surgical implication. Circulation 98 (suppl): II-299-II-304, 1998.
- [9] Holzer M: For the hypothermia after cardiac arrest study group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. NEng J Med 346: 549-556, 2002.
- [10] Mijuskovic D, Stamenkovic DM, Borovic S *et al*: Successful resuscitation from two cardiac arrests in a female patient with critical aortic stenosis, severe mitral regurgitation and coronary artery disease. Vojnosanit Pregl 69 (8): 714-716, 2012.
- [11] Fleisher A, Fleischmann KE, Auerbach AD *et al*: 2014 ACC/AHA guidelines on perioperative cardiovascular evaluation and management of patients undergoing non cardiac surgery. J Am Coll Cardiol 64 (22): 2373-2405, 2014.