Early Hydralazine Induced Chest Pain, in Postoperative Hypertension

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CASE REPORT

A previously healthy (nonsmoker, non-alcoholic and non-drug abuser) 60-year-old woman underwent general anesthesia for an endoscopic sinus surgery operation in a medical center.

Her past medical history included HTN medication, which the patient had randomly stopped for the past 6 months. She had normal cardiac, respiratory, liver and renal functions with normal laboratory findings on preoperative examination.

Her preoperative echocardiogram showed an Ejection Fraction (EF) of 67%, relaxation abnormalities, mild tricus regurgitation, and was otherwise unremarkable. Her blood pressure (BP) was usually maintained around 130/80 mmHg and her heart rate (HR) was 60, it was agreed to continue the operations without any special medication.

In the operating room, her BP was 140/80 mmHg and HR 79. General anesthesia was performed with propfol 120 mg (2 mg/kg), rocuronium 50 mg (0.9 mg/kg) for induction, desflurane 1 Minimum Alveolar Concentration (MAC) and remifentanyl 0.1 ug/min/kg were used for maintenance.

There were no problems intraoperatively, or during extubation and she was sent to the recovery room with a BP of 150/90 mmHg and HR of 90.

Upon arrival at the recovery room, her BP was 180/100 mmHg and HR 95, but she did not complain of nose pain, only stuffiness.

After 5 minutes, her BP was 192/110 mmHg and HR 95, under the direction of anesthesiologists, hydralazine 5mg iv was given.

After 30 minutes, the patient’s chest pain was relieved and her BP was 160/80 mmHg and HR 100 at this time.

The biomarker results for cardiac necrosis were normal: creatinine kinase-MB; 1.0 ng/mL (reference range <6.3 ng/mL) and troponin-I; 0.02 ng/mL (refe-
rence range: <0.2 ng/mL) and myoglobin; 36.4 ng/mL (reference range 17.4–105.7 ng/mL). However she still complained of mild chest pains and echocardiogram was performed, but the results did not show any difference from her preoperative exams.

IV infusion of NTG 0.5 ug/min/kg was maintained while transferring to the ward, and she was discharged after 3 days without any problems.

**DISCUSSION**

The transient nature of postoperative HTN, and the unique clinical factors present in the postoperative period, requires that this clinical syndrome be given particular attention. (4)

Postoperative HTN (arbitrarily defined as systolic BP ≥190 mmHg and/or diastolic BP 100 mmHg on 2 consecutive readings following surgery) (2,6) may have significant adverse sequelae in both cardiac and noncardiac patients. Postoperative HTN often begins 10–20 minutes after surgery and may last up to 4 hours. (10) If left untreated, patients are at increased risk for bleeding, cerebrovascular events, and myocardial infarctions. (3)

Cause of postoperative HTN and acute pain-induced sympathetic stimulation leading to vasoconstriction; in the early postanesthesia period, associated with pain induced sympathetic stimulation, hypothermia, hypoxia, or intravascular volume overload from excessive intraoperative fluid therapy; and in the 24 to 48 hours after postoperatively as fluid is mobilized from the extravascular space. In addition, BP elevation secondary to discontinuation of long-term antihypertensive medication may occur postoperatively.
Postoperative HTN in the first course of management is to reverse precipitating factors (pain, hypervolemia, hypoxia, hypercarbia, and hypothermia).

NSAIDS and Opioids in pain control or inhibit sympathetic stimulation, and it can do effectively stable are two vital.

The approach to the treatment of perioperative HTN is considerably different than the treatment of chronic HTN.(5) The initial approach to treatment is prevention. One preventive approach is to substitute long-acting preparations of the patient’s long-term antihypertensive regimen starting, if possible, several days before surgery and to be given in the morning of the day of surgery.

In all cases, the possible causes of the patient’s BP elevation should be considered

In patients with a hypertensive emergency, it usually is necessary to treat with a parenteral antihypertensive agent. In the acute setting, the treatment goal is to decrease BP by no more than 25%.(1) Advancing these guidelines, the authors believe the immediate goal of therapy in hypertensive emergencies to reduce diastolic BP by 10% to 15%, or to approximately 110 mmHg, over a period of 30 to 60 minutes. Sodium and volume depletion can be significant, and gentle volume expansion with IV saline solution will serve to restore organ perfusion and prevent an abrupt decline in BP when antihypertensive regimens are initiated. This goal decreases the likelihood of too-aggressive control, which may result in target organ hypoperfusion.

Pharmacotherapy for hypertensive crisis involves a wide variety of agents with different mechanisms of action and pharmacologic properties. The agent of choice in any particular situation will depend on the clinical presentation. The ideal agent for treatment of hypertensive emergencies should be rapid acting, predictable and easily titrated, safe, inexpensive, and convenient.

Hydralazine has been used in this case as a direct-acting arteriolar vasodilator, often chosen as a first-line agent for critically ill patients with pregnancy-induced HTN that produces a rapid BP reduction, affecting diastolic more than systolic BP. Also be used in controlled hypotension for surgery, including

The onset of action after either IM or IV administration is approximately 5 to 15 minutes followed by a progressive and often precipitous fall in BP that can last up to 12 hours,(8,9) however; its maximum effect is usually noted between 10~80 minutes.

It should also be avoided in patients suspected of having a dissecting aneurysm and patients with intracranial processes (eg, head injury) in which increased intracranial pressure is an issue.

Its reduction of peripheral vascular resistance leads to a reflex tachycardia that can increase cardiac output. Therefore, hydralazine would not be a good choice in a patient with ischemic heart disease who may not tolerate the increased myocardial oxygen consumption associated with the cardiovascular effects.(7)

In contrast, the increased renal blood flow attributable to hydralazine can be considered a potential advantage in patients with impaired renal function.

In this case, the patient did not complain of pain, and because of the possibility of bleeding after endosinus-surgery, and the patient’s history of HTN, hydralazine was chosen to reduce BP.

30 minutes after injection of hydralazine the patient complained of chest pain, and ST depression on the ECG was observed.

The authors performed further tests (echocardiogram) to rule out myocardial infarction and NTG was administered.

Fortunately, the echocardiogram was unremarkable, but in research it was found that some hypotensive agents could be a cause for acute myocardial infarction in all patients. If there is a hypertensive crisis and BP
control is needed, first, pain control, hypoxia, hypercarbia, hypothermia, hypovolemia should be ruled out before the use of a hypotensive agent. Because the immediate use of a hypotensive agent could cause a myocardial infarction or ischemia due to an excessive use of myocardial oxygen.

**ABSTRACT**

수술 후 고혈압에서 조기에 투여된 Hydralazine에 의한 가슴통증

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Hydralazine은 소동맥확장에 직접 작용하는 약으로, 종종 임신성 고혈압 환자에게 사용된다. 이 약의 소동맥확장 작용은 심박출량을 증가시킬 수 있는 반사적 빈맥을 유발한다. 그러므로 허혈성 심장질환 환자에게서 심근의 산소소모량을 증가시킬 수 있어 Hydralazine은 좋은 선택이 아니다.

본 증례보고에서는 수술 후 고혈압의 조절에서 선행되어야 할 통증, 저체온, 저산소증, 혈관내 용적과부하 등의 조절이 이루어지기 전에 Hydralazine이 사용 되었고 결과적으로 가슴통증이 왔다. 저자는 여러 문헌을 통해 본 증례에 대한 고찰을 하였다.

**REFERENCES**