TÜBİTAK

Turkish Journal of Medical Sciences

http://journals.tubitak.gov.tr/medical/

Research Article

Turk J Med Sci
(2016) 46: 1634-1640

doi:10.3906/sag-1412-33

Management of acute postoperative hypertension for reducing cardiovascular complications in cancer patients: when and how aggressively?

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Received: 09.12.2014 • **Accepted/Published Online:** 04.02.2016 • **Final Version:** 20.12.2016

Background/aim: We sought to determine what factors cardiovascular complications (CVCs) correlate with in cancer patients with acute postoperative hypertension (APH) and to define when and how aggressively to treat this disorder in the present study.

Materials and methods: A retrospective observational study of cancer patients with postoperative hypertension at a single intensive care unit between July 2007 and June 2013 was conducted. The outcome of interest was the incidence of a CVC.

Results: There were 1162 patients enrolled, of which 92 (7.9%) experienced one or more CVCs. Preexisting hypertension (OR 38.3, 95% CI 3.2–457.4, P = 0.004) and choice of vasodilator (OR 6.5, 95% CI 2.3–18.6, P = 0.000) were predictors of a CVC. Cardiovascular complications were less likely to occur if pain was relieved efficiently during the postoperative period (OR 15.9, 95% CI 1.9–130.1, P = 0.01). Furthermore, there were increased CVCs among patients with postoperative hypertensive crises compared to those with stage 1 or stage 2 hypertension with the treatment threshold for systolic blood pressure (OR 5.7, 95% CI 2.0–16.5, P = 0.001) or diastolic blood pressure (OR 6.0, 95% CI 1.8–20.3, P = 0.004).

Conclusion: To reduce CVCs, APH may be defined and managed as a hypertensive crisis in cancer patients.

Key words: Acute postoperative hypertension, cardiovascular complication, cancer, surgery

1. Introduction

The immediate postoperative period is a time of increased risk of complications in surgical patients, with the blood pressure frequently affected. Acute postoperative hypertension (APH) refers to abnormally increased blood pressure in the period, which usually occurs in patients undergoing major surgery including cardiothoracic, vascular, head and neck, and neurological surgery (1–6). The reported frequency of APH varies with different surgical procedures, and the incidence is relatively higher in cardiovascular surgery (1–6). No standard definition of APH exists, making the variability in reported incidence difficult to reconcile (5–7).

The present reports describe that APH usually occurs within 2 h after surgery, and the duration is usually 6 h or less (4–7) and complications are higher if the elevated blood pressure lasts longer than 3 h (5). Therefore, most physicians agree that aggressive antihypertensive treatment is necessary for APH to prevent the incidence of severe complications. However, most commonly, 140/90 mmHg is the quoted threshold for the treatment of hypertension in cardiac surgery (4,5,7,8). For most noncardiac types of surgery, there is a lack of agreement about when and how aggressively to treat APH (4, 5, 7). The treatment thresholds used in clinical trials of postoperative hypertension have included either a fixed blood pressure value (e.g., blood pressure of >160/90 mmHg, or mean arterial pressure of >110 mmHg) or a relative change from baseline (e.g., an increase in systolic or diastolic pressure of ≥20%) (5). In our center, physicians apply the criteria for chronic primary hypertension to diagnose APH (9), and treatment of APH is frequently a bedside decision based on their professional backgrounds. Thus, how to manage APH in cancer patients has not been clearly established.

APH is due mainly to increased sympathetic tone and vascular resistance (10,11). Contributing factors may also include pain and increased intravascular volume (11). However, acute or chronic hypertension may or may not occur in the face of a shifted autoregulatory perfusion curve and may result in the need for higher blood pressure

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to perfuse end organs (11,12). This situation makes it very challenging to control blood pressure after surgery (11). Therefore, precisely describing the epidemiology of APH in this patient population who receive surgical tumor resection, as well as identifying any modifiable risk factors contributing to the development of cardiovascular complications (CVCs), is important to determine when and how aggressively to treat APH.

2. Materials and methods

2.1. Patients and ethics

Ethical approval for this study was provided by the Ethics Committee of Sun Yat-Sen University Cancer Center, Guangzhou, China. We performed anonymous analysis of routinely collected clinical data. The ethics committee of our institution waived the need for informed consent. A retrospective, observational study of cancer patients with APH at a single intensive care unit (ICU) between July 2007 and June 2013 was then conducted. A total of 9105 patients for anesthesia care were admitted to the intensive care unit (ICU). Eligible patients were identified by searching our billing database for the term "hypertension" as the primary procedure. Inclusion criteria were as follows: hypertension occurred within 24 h after surgery with systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg; patients received a short-acting vasodilator for APH; patients received no long-acting vasodilator within 24 h after surgery. Exclusion criteria were: patients with a previous history of coronary heart disease, the same type of arrhythmia before and after operation, or incomplete clinical data. A total of 1184 patients met the inclusion criteria and their charts were systematically reviewed. A total of 1162 patients were included and 22 patients were excluded, including 21 patients with preoperative arrhythmia or myocardial ischemia and 1 due to incomplete medical records.

2.2. Clinical variables

Preoperative medical comorbidities and preoperative smoking or drinking history were recorded from admission history documents. Duration of anesthesia was obtained from anesthetic records. ICU stay was recorded from hospital charts. After operation, patients were sent to the ICU under continuous monitoring of electrocardiograph, blood pressure, and blood oxygen saturation for 48 h. For the patients with postoperative hypertension, the following parameters were recorded: types of CVCs, vasodilators used, duration of anesthesia and types of surgery, pain grade (visual analog score, VAS) of postoperative analgesia, urine volume, and central venous pressure (CVP). All patients received general anesthesia with tracheal intubation and postoperative analgesic pump therapy (with 25 mg of morphine for 48 h, 0.5 mg/h) after surgery. Vital signs, including blood pressure, and CVCs were recorded every 30 min. The postoperative course during ICU stay included CVCs within 48 h after surgery. When postoperative hypertension occurred, doctors determined the need for treatment and selected the vasodilator to be administered. Due to the lack of standard definition or treatment threshold in noncardiac surgery, the doctors' professional background was an important factor affecting their decision to initiate treatment and the goals of blood pressure control. Medications included the following: sublingual nifedipine tablets (South China Pharmaceutical Group Ltd., Guangdong, China), intravenous injection of urapidil (Nycomed GmbH, Singen, Germany), and nitroglycerin (Shanxi Kangbao Biological Products Co., Ltd., Shanxi, China), infused intravenously by a micropump.

The primary outcome of interest was the incidence of CVCs, including arrhythmia, myocardial ischemia, and heart failure, which were defined using standard diagnostic criteria (13). Based on the Joint National Committee (JNC) on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, according to the most recent report, JNC7, patients were classified into three groups: stage 1 hypertension (SBP 140–159 mmHg and/or DBP 90–99 mmHg), stage 2 hypertension (SBP 160–179 mmHg and/or DBP 100–109 mmHg), and hypertensive crisis (SBP ≥180 mmHg and/or DBP ≥110 mmHg) (10). For choice of vasodilator, patients were split into three groups: nitroglycerin-, urapidil-, and nifedipine-treated patients. The patients were further divided into a group with high-risk surgery that included thoracic or cerebral surgery and a group with low-risk surgery that included all other types of surgery. The patients were also divided into two groups based on goal of blood control (yes or no), intraoperative blood loss (<300 or ≥300 mL), pain grade (VAS score of <5 or ≥5), and CVP (<10 mmHg or ≥10 mmHg). There is no treatment goal for postoperative hypertension after noncardiac surgery. Based on our review of the literature, the patients were stratified based on a 20% reduction of SBP or DBP 3 h after antihypertensive treatment (11,14,15).

2.3. Statistical analysis

Data were collected by a single researcher. All authors agreed on the definitions of CVCs prior to the commencement of data collection. Data were presented as mean (±SD) or number (proportion). Univariable analyses were conducted using two-tailed t-testing, chi-square analysis, and Fisher's exact test where appropriate. Significant univariate predictors, identified at $P < 0.05$, were then entered into multivariable logistic regression analysis to identified independent predictors. All statistical analyses were performed with SPSS 14 (SPSS Inc., Chicago, IL, USA), and $P < 0.05$ was considered significant. Manuscript preparation was done in accordance with the Strengthening the Reporting of Observational Studies

in Epidemiology (STROBE) Statement: Guidelines for Reporting Observational Studies [16].

3. Results

In this study, patients were predominantly male (69.3%) and the median age (SD) at the time of surgery was 61.1 (12.5) years. Median weight (SD) was 62.5 (15.7) kg and 43.3% or 19.7% of patients had a smoking or drinking history, respectively. Comorbidity was documented in 623 cases (53.6%). The most prevalent medical comorbidity was hypertension (39.0%), followed by diabetes mellitus (8.7%). The rate of high-risk surgery, which included thoracic or cerebral surgery, was 47.5%. The majority of patients (62.9%) were operated on for less than 4 h, and the blood loss during operation was less than 300 mL in 71.6% of patients (Table 1).

In 9105 patients undergoing surgery, 1184 patients suffered from postoperative hypertension, and the incidence was 13.0% (defined as blood pressure ≥140/90 mmHg).The initial mean treatment threshold of SBP was 160.6 mmHg (147–206 mmHg) and DBP was 97.6 mmHg (81–124 mmHg) in the 1162 patients not excluded from our study (Table 2). After treatment with a short-acting vasodilator for 3 h, SBP fell by 12.9% and DBP fell by 6.7%, respectively.

Overall, our analysis revealed that 92 out of 1162 patients (7.9%) developed one or more postoperative CVCs. The most frequently observed CVC was an arrhythmia without hemodynamic change (n = 87). Eighty-seven patients (94.6%) experienced a single CVC, and 5 patients (5.4%) who experienced myocardial ischemia or heart failure developed two or more complications. Twenty-nine episodes of sinus tachycardia, 27 episodes of atrial premature contraction, 26 episodes of atrial fibrillation, 11 episodes of ventricular premature contraction, 7 episodes of supraventricular tachycardia, 4 cases of myocardial ischemia, and 1 case of heart failure were observed (Table 3). The overall incidence of severe CVCs falls to 5 out of 92 (5.4%) when the patient who experienced isolated arrhythmia is removed from the analysis.

Table 4 shows the results of the univariate analysis performed to determine the significance for the variables to predict the development of CVCs. A preoperative diagnosis of hypertension was the only medical comorbidity significantly associated with a CVC (P = 0.013). Development of CVCs was significantly associated with the choice of vasodilator, and the incidence of CVCs was 6.5% and 6.1% in the urapidil-treated and nifedipine-treated patients, respectively, but 17.6% in the nitroglycerin-treated patients (P = 0.000). Those with pain relieved efficiently during the postoperative period developed fewer CVCs compared to those without (P = 0.007). Furthermore, there were increased CVCs among patients with postoperative hypertensive crises compared to those with stage 1 or stage 2 hypertension with the treatment threshold for SBP (16.7% vs. 7.0% vs. 7.5%, P = 0.012) or DBP (12.8% vs. 8.3% vs. 6.5%, P = 0.049). The incidence of CVCs in the patients who achieved the goal of blood pressure control and the patients who did not were 7.1% and 8.2%, respectively (P = 0.732). Moreover, there was no association between any of male sex, age, body weight, smoking, drinking, diabetes mellitus, type of surgery, duration of anesthesia, intraoperative blood loss, or CVP and CVCs (P > 0.05) (Table 4). All of the severe CVCs, including 4 cases of myocardial ischemia and 1 case of heart failure, developed in cases of preexisting hypertension combined with hypertensive crisis patients. No patients died in the ICU stay within 48 h.

Table 1. Demographic and clinical variables for patients with postoperative hypertension (n = 1162)

Total patients, no. (%)	1162 (100)
Male, no. (%)	805 (69.3)
Age, mean (SD), years	61.1 (12.5)
Weight, mean (SD), kg	62.5 (15.7)
Smoking history, no. (%)	503 (43.3)
Drinking history, no. (%)	229 (19.7)
Diagnosis, no. (%)	
Lung cancer	259 (22.3)
Esophageal cancer	212 (18.2)
Intracranial tumor	81 (7.0)
Colorectal cancer	196 (16.9)
Gastric cancer	141 (12.1)
Liver cancer	130 (11.2)
Urologic cancer	24 (2.1)
Gynecological cancer	20 (1.7)
Head and neck cancer	13 (1.1)
Other cancer	86 (7.4)
Medical comorbidity, no. (%)	
Hypertension	453 (39.0)
Diabetes mellitus	101 (8.7)
COPD	54 (4.6)
CVA/TIA	8 (0.7)
Chronic renal failure	7 (0.6)
Duration of operation, no. (%)	
Duration of anesthesia, <4 h	731 (62.9)
Blood loss, <300 mL	832 (71.6)

COPD, Chronic obstructive pulmonary disease; CVA, cerebrovascular accident; TIA, transient ischemic attack; SD, standard deviation.

Table 2. Initial treatment threshold for systolic and diastolic blood pressure in the study.

	Mean (SD)	Minimum	Maximum
SBP	160.6 (12.2)	147	206
DBP	97.6 (10.4)	81	124

SBP, Systolic blood pressure; DBP, diastolic blood pressure; SD, standard deviation.

Multivariate logistic regression analysis was conducted using input parameters as follows: preexisting hypertension, treatment threshold for systolic blood pressure or diastolic blood pressure, choice of vasodilator, and analgesia (Table 5). The full model containing all predictors was statistically significant (P = 0.000). Five of the variables were independent predictors of CVCs. Preexisting hypertension (OR 38.3 [95% CI 3.2–457.4], P = 0.004) and choice of vasodilator (OR 6.5 [95% CI 2.3–18.6], P = 0.000) were predictors of a CVC. Cardiovascular complications were less likely to occur if pain was relieved efficiently during the postoperative period (OR 15.9 [95% CI 1.9–130.1], P = 0.01). Furthermore, there were increased CVCs among patients with postoperative hypertensive crises compared to those with stage 1 or stage 2 hypertension with the treatment threshold for SBP (OR 5.7 [95% CI 2.0–16.5], P = 0.001) or DBP (OR 6.0 [95% CI 1.8–20.3], P = 0.004).

4. Discussion

Acute postoperative hypertension is a common complication after any major surgery. Different incidences have been reported in the literature of 1.1%–60% (5,17–20). In this study, the incidence of postoperative hypertension was congruent with previous data regarding postoperative hypertension in patients undergoing noncardiac surgery. Acute postoperative hypertension has an early onset and is typically of short duration (4–7). The risk factors for APH include the surgical technique, duration of anesthesia, patient characteristics, and pain management strategy (5). However, elevation of SBP or DBP in primary hypertension is associated with cardiovascular complications (21–23). Clinical guidelines suggest that when blood pressure is higher than 140/90 mmHg, antihypertensive treatment should be administrated in order to reduce the mortality rate and the occurrence of myocardial infarction, heart failure, or other cardiovascular complications (10,24). In contrast, for APH, the significance of transient postoperative increases in blood pressure, the definition, the treatment threshold and goal, and the potential adverse effects of vasodilators are debated (5).

In this study, the incidence of CVCs in postoperative hypertensive patients was 7.9%; there was only 1 case of heart failure and 4 cases of myocardial ischemia. The other cases were arrhythmias. The overall incidence of severe cardiovascular complications markedly decreased when the patient who experienced isolated arrhythmia is removed from the analysis. Therefore, the present data are unable to fully describe the relationship between severe CVCs and correlated risk factors. The incidence of cardiac arrhythmia was the major outcome in this study.

A high incidence of cardiac arrhythmia was found in these patients diagnosed with primary hypertension before operation. Furthermore, all of the severe complications, including 1 case of heart failure and 4 cases of myocardial ischemia, were also found in these patients. Patients with untreated severe hypertension have a higher incidence of stroke, myocardial ischemia, and renal failure because of chronic changes in the autoregulation of end organ perfusion (4–7). Postoperative hypertension is due mainly to increased sympathetic tone and vascular resistance in

Table 3. Frequency of individual postoperative cardiovascular complications (n = 92).

Complications	Number of CVCs	Proportion of total CVCs (%)
Sinus tachycardia	29	27.6
Atrial premature contraction	27	25.7
Atrial fibrillation	26	24.8
Ventricular premature contraction	11	10.5
Supraventricular tachycardia	7	6.7
Myocardial ischemia	4	3.8
Heart failure	1	0.9
Total	105*	100

CVC, cardiovascular complication. *Several patients experienced more than one cardiovascular complication.

Table 4. Univariate analysis to determine the variables for cardiovascular complications in all 1162 patients with postoperative hypertension.

Variables	Total (n = 1162)	Patients without CVCs (n = 1070)	Patients with CVCs (n = 92)*	P
Male, no. (%)	805 (69.3)	747(70.8)	58 (63.0)	0.177
Age, mean (SD), years	61.1 (12.5)	60.5 (12.1)	62.4 (10.7)	0.254
Weight, mean (SD), kg	62.5 (15.7)	61.5 (17.4)	62.9 (13.9)	0.129
Smoker, no. (%)	503 (43.3)	463 (41.3)	40 (43.4)	0.969
Drinking habit, no. (%)	229 (19.7)	211 (19.7)	18 (20.0)	0.971
Comorbidity, no. (%)				
Hypertension	453 (39.0)	406 (37.9)	47 (51.1)	0.013
Diabetes mellitus	101 (8.7)	91 (8.5)	10 (10.9)	0.440
Duration of operation, no. (%)				
Surgery type, high-risk	552 (47.5)	501 (46.8)	51 (55.4)	0.112
Anesthesia, ≥4 h	431 (37.1)	396 (37.0)	35 (38.0)	0.844
Blood loss, ≥300 mL	330 (28.4)	311 (29.1)	26 (28.3)	0.870
Postoperative management, no. (%)				
Treatment threshold, SBP ≥180 mmHg	78 (6.7)	65 (6.1)	13 (14.1)	0.012
Treatment threshold, DBP ≥110 mmHg	133 (11.5)	116 (10.8)	17 (18.5)	0.049
Goal of BP control, yes	599 (51.5)	550 (51.4)	49 (53.3)	0.732
Choice of vasodilator, nitroglycerin	159 (13.7)	131 (12.2)	28 (30.4)	0.000
Analgesia, VAS <5	953 (82.0)	887 (82.9)	66 (58.7)	0.007
CVP, ≥10 mmHg	304 (26.2)	279 (26.1)	25 (27.2)	0.818

BP, Blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; VAS, visual analog score; CVP, central venous pressure; CVC, cardiovascular complications; SD, standard deviation. *Several patients sustained more than one cardiovascular complication.

patients with underlying primary hypertension (10,11). Therefore, for cancer patients, primary hypertension should be fully evaluated and managed before operation. Candidates for tumor resection should continue their antihypertensive medications through the morning of surgery and then resume the medications as soon as possible after the procedure (10,11). In addition, cardiac arrhythmic events are often observed in cancer patients undergoing thoracic surgery, and the possibility that they were caused by surgical trauma cannot be excluded (25). However, our study failed to detect a difference in the incidence of CVCs in the patients with high-risk surgery, including thoracic surgery, compared to those with low-risk surgery, in contrast to contemporary reports (25). Moreover, the effects of selection of duration of anesthesia and operation, and intraoperative blood loss on the development of postoperative CVCs, especially on cardiac arrhythmia, were small in this study.

Regarding postoperative management, it is standard practice in our institution to treat postoperative hypertensive patients with antihypertensive medications when the blood pressure is over 140/90 mmHg. It is possible that routine postoperative antihypertension does not predispose these patients to CVCs; however, when and how aggressively to treat hypertension in the immediate postoperative period has not been clearly established.

Table 5. Multivariable analysis to determine the predictors for cardiovascular complications in all 1162 patients with postoperative hypertension.

Variables	OR (95% CI)	P
Hypertension	38.3 (3.2–457.4)	0.004
Choice of vasodilator	6.5 (2.3–18.6)	0.000
Treatment threshold, SBP	5.7 (2.0–16.5)	0.001
Treatment threshold, DBP	6.0 (1.8–20.3)	0.004
Analgesia, VAS <5	15.9 (1.9–130.1)	0.01

SBP, Systolic blood pressure; DBP, diastolic blood pressure; VAS, visual analog score.

In this study, we found that there were increased CVCs among the patients with hypertensive crises, and all of the severe CVCs occurred in these patients. Thus, a postoperative hypertensive crisis may conceivably elevate the risk of sustaining a CVC, and the high incidence of CVCs observed is less surprising when severe hypertension is considered. The incidence of CVCs in patients with stage 1 or stage 2 postoperative hypertension was lower. Furthermore, the incidence of CVCs between the patients who achieved the goal of blood pressure control quoted in this study and the patients who did not were not markedly different. Therefore, we propose that APH may be defined as a classification of a hypertensive crisis, and patients with hypertensive urgency should have their blood pressure reduced within 24 to 48 h, whereas patients with hypertensive emergency should have their blood pressure lowered immediately. In addition, we found that the treatment of postoperative hypertension with intravenous nitroglycerin administered by micropump was a factor for CVCs. It may be due to the rapid and strong effect of nitroglycerin to lower blood pressure leading to adverse events, such as sinus tachycardia. This somewhat conflicts with the report of Bussmann et al., which implied that there was not significant difference in side effects between nitroglycerin and nifedipine in the treatment of hypertension (26). We thought that it may be because of using nitroglycerin sublingually, not being administered by micropump. These data suggest that this antihypertensive strategy did not significantly reduce complications, but the antihypertensive treatment induced somewhat an increase of adverse events. On the other hand, postoperative pain can cause agitation, anxiety, nervousness, and fear in patients, resulting in an increase in catecholamines and angiotensin, which would elevate myocardial automaticity and excitability, leading to cardiac arrhythmias (25). For the pain of incisions, regular analgesics are generally used. In this study, a VAS score of <5 was found in the majority of patients (82.0%), and fewer CVCs developed in these patients. Therefore, pain being relieved efficiently is useful for reducing CVCs in hypertensive patients after tumor resection.

The impact of primary hypertension on survival results mainly from the long-term contraction of small arteries leading to gradual changes in internal organs and pathological lesions in vital organs, such as the heart, which ultimately results in death (27,28). However, APH is mainly due to vasoconstriction of the resistance vessels in the circulatory system under a stress condition, and these events are often temporary and self-correcting (4–7,29–31). Thus, except for a hypertensive crisis, postoperative hypertension might not be sufficient to cause damage to vital organs such as heart and result in CVCs. Due to differences in etiology and pathological processes between primary and postoperative hypertension, we suggest that it is inappropriate to apply the classification system and management guidelines for chronic hypertension to the treatment of postoperative hypertension.

Our study demonstrated that postoperative hypertension in cancer patients is common. Those who developed CVCs were more likely to have preexisting hypertension and side effects of vasodilators. Pain relieved efficiently decreased the development of CVCs. There were increased CVCs among the patients with hypertensive crises. Postoperative stage 1 or stage 2 hypertension itself did not increase the development of CVCs, and short-acting vasodilators did not decrease the developed CVCs. Therefore, to reduce CVCs, APH may be defined and managed as a hypertensive crisis in cancer patients. Regarding postoperative stage 1 or stage 2 hypertension, preexisting hypertension and potentially reversible causes, such as postoperative pain, should be carefully managed, but routine short-acting vasodilators may not be used.

Our study has several limitations. First, this work was a retrospective study that investigated patients with APH after surgery. Hence, the study may be subject to recruitment bias. Second, it is essential to acknowledge that postoperative hypertension is due mainly to increased sympathetic tone (11), which results in CVCs, including arrhythmia. In the present study, the incidence of cardiac arrhythmia was the major complication. Although administration of beta blockers is preferred to prevent CVCs, in our center, they are rarely used regardless of preoperation or postoperation timing during the study period.

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