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ORIGINAL ARTICLE

Is microvascular decompression surgery a high risk for postoperative nausea and vomiting in patients undergoing craniotomy?

Keita Sato · Seijyu Sai · Takehiko Adachi

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Abstract
Purpose Patients undergoing microvascular decompression surgery often experience postoperative nausea and vomiting (PONV). However, there is little information about the incidence of PONV after microvascular decompression. We hypothesized that microvascular decompression is an especially high-risk procedure for PONV in patients undergoing neurosurgery, and investigated risk factors related to PONV after neurosurgery.
Methods All patients who underwent craniotomy in our institution during a period of 2 years were investigated retrospectively. Medical charts were reviewed to identify PONV during the 24-h postoperative period and related risk factors. Multivariate logistic regression analysis was conducted to elucidate the impact of microvascular decompression on PONV after craniotomy.
Results Among 556 craniotomy cases, 350 patients met the inclusion criteria. Multivariate logistic regression analysis showed that microvascular decompression was an independent risk factor for PONV after craniotomy (odds ratio 5.38, 3.02–9.60), in addition to female gender, nonsmoker status, amount of intraoperative fentanyl administered, and cerebrovascular surgery.
Conclusion In this retrospective study, microvascular decompression surgery was an especially high-risk factor for PONV in patients undergoing craniotomy. It may be necessary to adopt a combination of prophylactic methods to reduce the incidence of PONV after microvascular decompression.

K. Sato (✉) · S. Sai · T. Adachi
Department of Anesthesiology, Kitano Hospital, 2-4-20 Ogimachi, Kita-ku, Osaka-city 530-0025, Japan
e-mail: keita.satohh@gmail.com

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Introduction

Patients with hyperactive dysfunctional cranial nerve syndromes, such as trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia, who are refractory to medical therapy are candidates for surgery. Microvascular decompression (MVD) under posterior fossa craniotomy is among the most popular surgical therapies for these patients. MVD entails dissecting along the intracranial portion of the nerve, identifying the offending blood vessels that encroach on the nerve, and placing an insulating Teflon pad between the vessel and the nerve [1].

In the general surgical population, postoperative nausea and vomiting (PONV) was observed in 20–30 % of patients [2, 3], whereas in neurosurgical patients, a 40–80 % incidence of PONV within 24 h after craniotomy was reported [4–10]. PONV is unpleasant and may result in serious complications in neurosurgical patients consequent to elevated intracranial and arterial pressure.

Although we observe a high incidence of PONV after MVD, there is little information regarding this incidence. Recently, Tan et al. [11] reported MVD and acoustic neuroma resection were associated with an increased likelihood of PONV in the recovery room compared with other tumor resections in a retrospective case–control study. However, recovery room stay was 120–140 min in their study, and there have been no reports that showed MVD was associated with increased risk for PONV after the early postoperative period. Therefore, we investigated the risk factors for PONV in the 24-h period after

neurosurgery, focusing on whether MVD was a high-risk factor for PONV in patients undergoing neurosurgery.

Materials and methods

All patients who underwent craniotomy in our institution between April 2009 and March 2011 were investigated retrospectively. This study was approved by the institutional research ethics review committee, which granted a waiver of written informed consent. Exclusion criteria were patients younger than 18 years of age, emergency operation cases, Hardy's operation cases, awake craniotomy cases, patients who were not extubated in the operation room, patients with more than one craniotomy procedure during the same admission, and pregnant patients. Medical charts, anesthesia records, and nursing records were reviewed. The following patient data were collected: gender, age, body mass index (BMI), American Society of Anesthesiologists (ASA) physical status, smoking history, and preoperative corticosteroid administration. Collected intraoperative data included the type of surgery (tumor resection, cerebrovascular surgery, or MVD), intraoperative body position, craniotomy location (supratentorial or infratentorial), operation time, volume of fluid administered, volume of intraoperative blood loss, volume of intraoperative urinary output, anesthetic time, induction agents, anesthetic techniques of maintenance, use of remifentanil, intraoperative corticosteroid and droperidol administration, amount of intraoperative fentanyl administered, and reversal agents of muscle relaxants. Postoperative data included the postoperative use of corticosteroids, opioids, dexmedetomidine, and metoclopramide, the sedation level at night after surgery, and the presence of PONV during the 24-h postoperative period.

The primary outcome of this study was the incidence of PONV during the 24-h postoperative period. Under the clinical path in our institution, patients undergoing craniotomy are cared for in the intensive care unit for at least one night after surgery, and the nursing staff evaluate the symptoms of PONV at least every 4 h after surgery. Dexmedetomidine was usually administered at a fixed dose of 20 μg/h (0.35 μg/kg/h for average body weight in the study patients) for sedation and analgesia in the intensive care unit in our practice.

Nausea was noted as none, slight, or present, and we considered nausea was present only when noted as being present. Emesis was noted as none or present, and we considered the presence of emesis as noted. Because it was difficult to distinguish between nausea and retching in reviewing the medical charts in this retrospective study, we addressed PONV as one outcome and did not discriminate between nausea and vomiting. Because evaluation of the sedation level was not performed accurately in the intensive care unit, we estimated the sedation level based on the consciousness level recorded and nursing charts.

Data are expressed as the mean ± SD. A *t* test was used to compare continuous variables and the Fisher exact test or χ^2 test was used to compare categorical variables between patients with PONV and those without PONV in univariate analysis. Postoperative use of metoclopramide was removed from the univariate analysis because this agent was generally used for the treatment of PONV in our practice, not for prophylaxis. Multivariate logistic regression analysis was performed to elucidate the impact of MVD on the incidence of PONV, using the variables for which the *P* values were <0.10 in univariate analysis. This criterion, *P* value <0.10, was chosen to more accurately remove confounding factors from the model of multivariate analysis by permitting the variables that had a *P* value of less than 0.10 to be included in candidates of independent variables. Cramer's measure of association was used to evaluate multicollinearity between independent variables in the multivariate analysis. If the Cramer's measure of association was more than 0.85, a less important variable was removed from the independent variables, in consideration of the clinical significance. The following variables exhibited *P* values <0.10 in univariate analysis: 'female gender,' 'non-smoker,' 'preoperative corticosteroid administration,' 'types of surgery (tumor resection, cerebrovascular surgery, or MVD),' 'intraoperative body position,' 'craniotomy location,' 'amount of intraoperative urinary output,' 'intraoperative corticosteroid administration,' 'amount of intraoperative fentanyl administered,' and 'postoperative corticosteroid administration.' The variable 'intraoperative body position' was removed from the multivariate analysis because of multicollinearity with 'craniotomy location.' Because types of surgery were divided into three groups (tumor resection, cerebrovascular surgery, or MVD), the variable 'types of surgery' was converted to a dummy variables making 'tumor resection' the reference group in the multivariate analysis. Patients with missing data were excluded from the analysis. All statistical analyses were performed with SPSS version 11.0 software (IBM, New York, NY, USA), and *P* values <0.05 were considered significant.

Results

There were 556 cases that underwent craniotomy during the 2 years. We excluded 118 emergency cases, 54 cases with Hardy's operation, 12 cases that were not extubated in the operation room, 9 cases that were less than 18 years old, 8 cases of awake craniotomy, 1 pregnant case, 1 case with more than one craniotomy procedure during the same

admission, and 3 cases with missing data. Finally, 350 cases met the inclusion criteria (Fig. 1). Among the 350 patients, tumor resection was performed in 151 patients (43 %), cerebrovascular surgery in 85 patients (24 %), and MVD in 114 patients (33 %). Sevoflurane was used for maintenance of anesthesia in 345 patients (99 %). Nitrous oxide was not used. In the intensive care unit, dexmedetomidine was administered to 324 patients (93 %) for postoperative sedation and analgesia; 7 patients (2 %) were considered to be under deep sedation or drowsy at night in the intensive care unit after surgery, but all patients returned to be normal on the next morning after surgery. PONV occurred in 202 patients (58 %). Among the patients undergoing MVD, 83 (73 %) suffered from PONV.

The characteristics of the patients with PONV and those without PONV are shown in Tables 1 and 2. In patients with PONV, there were more female patients (patients with PONV, 73 %; patients without PONV, 51 %; $P < 0.001$) and more non-smokers (88 % vs. 80 %; $P = 0.04$) in comparison with those without PONV. Type of surgery and intraoperative body position were different between the patients with PONV and those without PONV. Patients with PONV were more likely to have undergone infratentorial craniotomy (51 % vs. 35 %; $P = 0.003$) and had less intraoperative urinary output (754 ± 550 vs. 949 ± 655 ml; $P = 0.003$). Patients with PONV had less intraoperative (12 % vs. 30 %; $P < 0.001$) and postoperative (16 % vs. 39 %; $P < 0.001$) corticosteroid administration. Trends suggesting less preoperative corticosteroid administration (2 % vs. 6 %; $P = 0.09$) and more intraoperative fentanyl administration (403 ± 216 vs. 359 ± 212 μg; $P = 0.06$) were observed in patients with PONV, but these differences were not significant.

In the multivariate logistic regression analysis (Table 3), five variables were found to be independent risk factors for PONV in patients undergoing craniotomy: female gender, non-smoker status, the amount of intraoperative fentanyl administration, MVD, and cerebrovascular surgery.

Discussion

In this retrospective study, we have investigated risk factors for PONV in the 24-h period after surgery in patients undergoing craniotomy. Our hypothesis was that MVD was a risk factor for PONV after craniotomy. In the multivariate analysis, MVD was found to be an independent risk factor for PONV, in addition to female gender, non-smoker status, the amount of intraoperative fentanyl administered, and cerebrovascular surgery.

Neurosurgery is considered as a surgical factor for PONV according to the consensus guidelines for managing postoperative nausea and vomiting [12]. Although there are controversies regarding the causal association between the type of surgery and increased PONV risk [13], we hypothesized that MVD was an independent risk factor for PONV, similar to strabismus surgery in children, which is a well-established independent risk factor for postoperative vomiting [14].

The incidence of PONV after craniotomy has been reported in double-blinded prospective randomized controlled trials that have mainly focused on prevention of PONV using 5-HT_3 antagonists [4–7, 9, 10]. In these studies, patients treated with placebo exhibited a 39–61 % incidence of emesis and 14–87 % incidence of nausea within 24 h after craniotomy. In other prospective studies, a 40–50 % incidence of PONV within 24 h after craniotomy was reported [7, 8]. In the present study, the overall incidence of PONV within 24 h after craniotomy was 52 %, which was comparable with these reports.

As to the incidence of PONV after MVD, there is little information available to date. Meng and Quinlan [15]

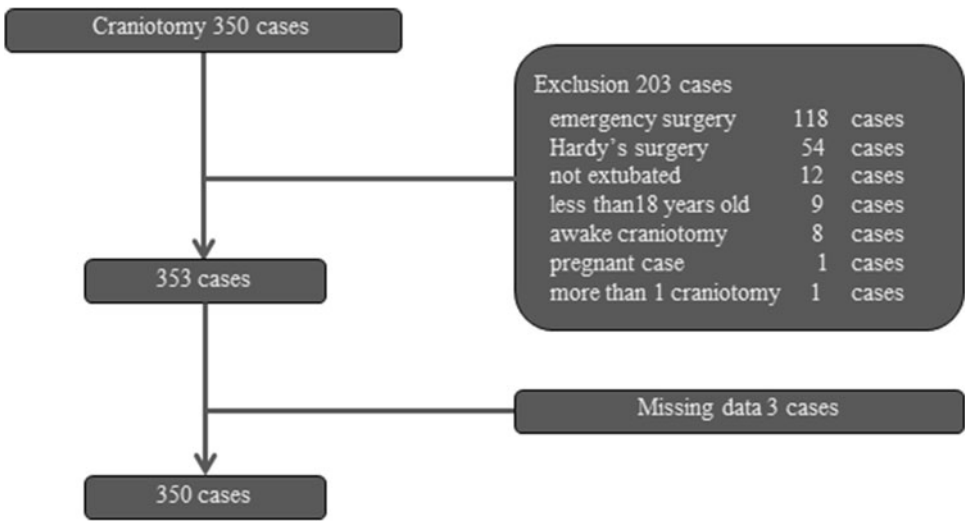

Fig. 1 Flow diagram of the study participants

Table 1 Demographics of the study patients with postoperative nausea and vomiting (PONV) and those without PONV relative to patient factors and surgical factors

	Total ($n = 350$)	PONV (+) ($n = 202$)	PONV (−) ($n = 148$)	P value
Patient factors				
Gender				
Female	212 (61 %)	147 (73 %)	75 (51 %)	<0.001
Age (years)	58 ± 13	58 ± 13	59 ± 14	0.70
BMI (kg/m^2)	22.9 ± 3.7	22.7 ± 3.9	23.3 ± 3.5	0.14
ASA-PS				
1	91 (26 %)	61 (30 %)	30 (20 %)	0.11
2	253 (72 %)	138 (68 %)	115 (78 %)	
3	6 (2 %)	3 (1 %)	3 (2 %)	
Non-smoker	296 (85 %)	178 (88 %)	118 (80 %)	0.04
Preoperative corticosteroid	14 (4 %)	5 (2 %)	9 (6 %)	0.09
Surgical factors				
Types of surgery				
Tumor resection	151 (43 %)	57 (28 %)	94 (64 %)	<0.001
Cerebrovascular	85 (24 %)	62 (31 %)	23 (16 %)	
MVD	114 (33 %)	83 (41 %)	31 (21 %)	
Body position				
Supine	170 (49 %)	89 (44 %)	81 (55 %)	<0.001
Prone	29 (8 %)	8 (4 %)	21 (14 %)	
Lateral	151 (43 %)	105 (52 %)	46 (31 %)	
Craniotomy location				
Supratentorial	194 (55 %)	98 (49 %)	96 (65 %)	0.003
Infratentorial	156 (45 %)	104 (51 %)	52 (35 %)	
Blood loss (ml)	155 ± 306	170 ± 370	137 ± 190	0.32
Fluid administration (ml)	2,802 ± 932	2,768 ± 912	2,870 ± 945	0.31
Urinary output (ml)	837 ± 602	754 ± 550	949 ± 655	0.003
Operation time (min)	286 ± 103	291 ± 97	281 ± 111	0.41

Data are expressed as number (%) or mean ± SD

PONV postoperative nausea and vomiting, *BMI* body mass index, *ASA-PS* American Society of Anesthesiologists physical status, *MVD* microvascular decompression

assessed risk factors for PONV in patients undergoing MVD in a retrospective fashion. They reported an overall incidence of PONV of 60 % during the first 24 h, despite the use of intraoperative prophylactic ondansetron in 99 % of their patients. However, whether MVD represented a higher risk for PONV compared with other types of craniotomy was uncertain from their study.

Recently, Tan et al. [11] reported MVD and acoustic neuroma resection were associated with an increased likelihood of PONV during the first 2-h period after surgery in a retrospective case–control study. Because there were few patients who had acoustic neuroma surgery in our population, we did not discriminate between acoustic neuroma surgery and other tumor resection, which differed from their study. As to MVD, our study is consistent with theirs, and added further information of MVD as a risk factor for PONV after the early postoperative period.

In the present study, we assessed the risk factors for PONV in the 24-h period after craniotomy. In addition to female gender, non-smoker status, and the amount of intraoperative fentanyl administered, which are well-known risk factors for PONV [12, 13, 16], the multivariate analysis showed that MVD was an independent risk factor for PONV in patients undergoing craniotomy. In fact, more than 70 % of patients undergoing MVD experienced PONV. Nausea and vomiting can be induced by various pathways through the vomiting center, including vagal afferents of the gastrointestinal tract, the chemoreceptor trigger zone in the area postrema, and the vestibular system [17]. Medial retraction of the cerebellum is performed near these structures during MVD [1] and may predispose to the development of PONV. However, as infratentorial craniotomy was not an independent risk factor for PONV in our study, which is consistent with other studies [8, 18, 19], the

Table 2 Demographics of the study patients with PONV and those without PONV relative to anesthetic factors and postoperative factors

	Total ($n = 350$)	PONV (+) ($n = 202$)	PONV (−) ($n = 148$)	P value
Anesthetic factors				
Anesthesia time	391 ± 112	396 ± 104	287 ± 122	0.47
Induction agents				
Propofol	243 (69 %)	139 (69 %)	104 (70 %)	0.47
Thiopental	100 (29 %)	58 (29 %)	42 (28 %)	
Sevoflurane	2 (0.5 %)	2 (1 %)	0 (0 %)	
Maintenance				
Propofol	5 (1 %)	2 (1 %)	3 (2 %)	0.65
Sevoflurane	345 (99 %)	200 (99 %)	145 (98 %)	
Use of remifentanil	312 (89 %)	187 (93 %)	132 (89 %)	0.34
Intraoperative corticosteroid	69 (20 %)	25 (12 %)	44 (30 %)	<0.001
Intraoperative droperidol	5 (1 %)	3 (1 %)	2 (1 %)	1.00
Total fentanyl administration (μg)	383 ± 215	403 ± 216	359 ± 212	0.06
Use of neostigmine	176 (50 %)	105 (52 %)	71 (48 %)	0.52
Postoperative factors				
Postoperative corticosteroid	90 (26 %)	32 (16 %)	58 (39 %)	<0.001
Use of opioid	3 (1 %)	3 (1 %)	0 (0 %)	0.27
Use of dexmedetomidine	324 (93 %)	190 (94 %)	134 (91 %)	0.22
Drowsy at night after surgery	7 (2 %)	2 (1 %)	5 (3 %)	0.12
Use of metoclopramide[a]	108 (31 %)	98 (49 %)	10 (7 %)	

Data are expressed as number (%) or mean ± SD

PONV postoperative nausea and vomiting

[a] Used for the treatment of postoperative nausea and vomiting

Table 3 Multivariate logistic regression analysis for postoperative nausea and vomiting

	Odds ratio (95 % CI)	P value
Female	3.22 (1.89–5.49)	<0.001
Non-smoker	2.28 (1.15–4.53)	0.02
Corticosteroid administration		
Preoperative	–	0.86
Intraoperative	–	0.37
Postoperative	–	0.14
Types of surgery		
Tumor resection	1.00 (reference)	
Cerebrovascular	4.80 (2.57–8.98)	<0.001
MVD	5.38 (3.02–9.60)	<0.001
Infratentorial craniotomy	–	0.23
Urinary output	–	0.30
Total fentanyl administration	1.002 (1.001–1.003)	0.001

CI confidence interval, *MVD* microvascular decompression

cause underlying PONV after MVD remains uncertain. Meanwhile, cerebrovascular surgery was also a significant risk factor for PONV after craniotomy. Considering that 50 % of patients with spontaneous intracranial hypotension exhibit nausea and vomiting [20], reduction of the cerebrospinal fluid may influence the incidence of PONV after each type of craniotomy.

Although dexamethasone has been reported to reduce the incidence of PONV in general surgical patients [21–24], corticosteroid administration was not associated with any reduction in the incidence of PONV in our study. Thus, it might be necessary to adopt a combination of the prophylactic methods to decrease the incidence of PONV after MVD. Several prophylactic methods have been suggested in patients undergoing craniotomy: 5-HT_3 receptor antagonists have proven effective for the prevention of postoperative emesis after craniotomy in a meta-analysis [25], electroacupoint stimulation has been reported to reduce PONV after craniotomy [8], and the type of anesthetic has also been reported to influence the incidence of PONV after craniotomy [26]. In addition, reduction of amount of fentanyl administered was suggested to be effective in our study. Local anesthesia or peripheral nerve block can also contribute to reducing the amount of opioid used for postoperative analgesia. Multimodal approaches of these prophylactic methods should be considered to reduce this problem.

Our study has several limitations. First, we do not have any information about the past history of PONV and

motion sickness, which are major risk factors for PONV. Second, this was a single-center, retrospective, observational study, which has significant inherent limitations in the external validity and the generalizability of the findings. Third, the incidence of PONV might be underestimated because of the nature of a retrospective study [27], although the symptom of PONV was evaluated at fixed intervals according to the clinical path at our institution.

In conclusion, we have retrospectively assessed risk factors for PONV in patients undergoing craniotomy, focusing on MVD. Our results showed that MVD was an independent risk factor for PONV, in addition to female gender, non-smoker status, the amount of intraoperative fentanyl administered, and cerebrovascular surgery.

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