Incidence and Management of Epistaxis after Endoscopic Skull Base Surgery

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Abstract
Background/Aims: Endonasal endoscopic surgery continues to gain acceptance as a minimally invasive, effective approach for benign and malignant pathology of the paranasal sinuses and skull base. Postoperative epistaxis could potentially result in rapid and devastating consequences due to hemorrhage. Our objective was to assess the incidence and causes of epistaxis after endoscopic skull base surgery.

Methods: Retrospective review of patients undergoing endoscopic skull base surgeries from 2007 to 2012. The main outcome measure was epistaxis requiring a visit to a hospital or clinic occurring within 30 days of surgery. Results: In our cohort of 330 consecutive patients, 10 (3%) experienced postoperative epistaxis, including 3 who had multiple episodes (14 events). A majority of the patients were controlled with packing in the emergency room (8/14 events). One patient required chemical cautery and 5 required control in the operating room. The only patient characteristic that reached significance was abstinence from alcohol (p value = 0.04). However, patients with epistaxis were more likely to be male, older and have hypertension. Conclusion: This study confirms that the risk of epistaxis after endoscopic skull base surgery is low and similar to sinonasal surgery for inflammatory conditions.

Introduction
Endonasal endoscopic surgery continues to gain acceptance as a minimally invasive, effective approach for benign and malignant pathology of the paranasal sinuses and skull base. The risk of major postoperative complications after endoscopic skull base surgery is low, ranging from 1–6% in two recent meta-analyses, and <16% in a single institution’s series of 800 patients [1–3]. Postoperative epistaxis is a complication after endoscopic skull base surgery that could potentially result in rapid and devastating consequences due to hemorrhage. Treatment of these patients is also more complex because the reconstructed skull base requires careful nasal packing due to the risk of disrupting the repair site or potentially introducing packing material intracranially.

The goal of this study is to determine the incidence and management of postoperative epistaxis after endoscopic skull base surgery, and to identify potential risk factors for bleeding.
Methods

Our study was approved by the institutional review board of the University of California at Los Angeles. We retrospectively reviewed the clinical data of all consecutive patients who underwent endoscopic skull base surgery between August 2007 and February 2012 at a single tertiary academic medical center. Patient characteristics that were analyzed included age, sex, pathologic diagnosis, tobacco and alcohol use, history of prior endonasal surgery, medical history (such as hypertension, Cushing’s disease and coagulopathies), tumor location, tumor type, malignant versus benign pathology, use of nasoseptal flap, operative blood loss, length of hospital stay and regular use of blood thinners prior to surgery or postoperatively. Blood thinners included the use of aspirin, clopidogrel bisulfate (Plavix), warfarin sodium (Coumadin), nonsteroidal anti-inflammatory drugs and fish oil. Patients were asked to stop aspirin, nonsteroidal anti-inflammatory drugs and fish oil 10 days prior to surgery. Clopidogrel bisulfate is stopped 14 days before surgery. Warfarin sodium is maintained until 1 week before surgery and the normalization of INR is ensured before surgery. At the end of surgery, bipolar cautery is used for hemostasis. The nasal cavity is then packed with microfibrillar collagen slurry (J.D.S.) or Nasopore (stryker Corporation, Kalamazoo, Mich., USA) (M.B.W.). For patients requiring skull base reconstruction, some cases required placement of Merocel (Medtronic, Jacksonville, Fla., USA) against the repair site which is removed after 2 weeks (J.D.S.) or a Foley catheter balloon (M.B.W.) removed prior to hospital discharge. The main outcome measure was epistaxis occurring within 30 days of surgery, which required a visit to a hospital or clinic.

We examined the bivariate association between each of the variables and the occurrence of postoperative epistaxis. The Fisher exact test was used to compare the categorical variables in patients with postoperative epistaxis versus those without epistaxis (tables 1–3). In order to compare the continuous variables between the 2 groups, the Student t test was used to compare the mean age, while the Wilcoxon rank sum test was used to compare median blood loss during surgery and the length of hospital stay (table 3).
We identified 330 patients who underwent endonasal approaches to the skull base between August 2007 and February 2012. There were 145 males (44%) and 185 females (56%). Tables 1, 2, and 3 list all of the patient characteristics that were analyzed. The most common tumor pathologies, as listed in table 2, were pituitary adenomas (n = 224), sinonasal malignancies (n = 18), Rathke’s cleft cysts (n = 18), meningiomas (n = 17) and clival chordomas (n = 10). Tumor pathology was not a significant factor for postoperative epistaxis (p value = 0.24).

A total of 10 patients (3%) experienced postoperative epistaxis. Of these, 6 had a single episode, 2 had 2 episodes and 1 had 3 episodes of epistaxis, of a total of 14 events (4.2%). The median time to the first episode of epistaxis in these 10 patients was 6.5 days (range 0–10 days). The clinical characteristics of each patient who had postoperative epistaxis are shown in table 4.

The site of bleeding was identified in 5 of the 14 (36%) events. Most of the patients were controlled with packing in the emergency room (8/14 events), and 1 also had chemical cautery of the sphenopalatine artery in the emergency room with silver nitrate. Five patients had bleeding that required control in the operating room. Two of these patients presented with epistaxis, but were also found on evaluation to have visual-field defects due to intrasellar and intrasphenoid blood. None of our patients required embolization or a blood transfusion. No patients in the study had a cerebrospinal fluid (CSF) leak result during management of the epistaxis.

In our analysis of patient characteristics and surgical data in order to identify risk factors for epistaxis, only alcohol abstinence reached statistical significance (p = 0.04) (tables 1–3). Of the 330 patients in our study, 100 admitted to drinking alcohol at least weekly, and none of these patients had epistaxis. None of the 10 patients who had postoperative bleeding drank alcohol.

No other patient characteristic reached statistical significance. A history of hypertension (p = 0.48), the use of blood thinners (p > 0.99), prior nasal surgery (p = 0.45) and the use of a nasoseptal flap (p = 0.71) were not statis-
tically significant risk factors for postoperative bleeding. However, patients with epistaxis were more likely to be male (70 vs. 43%), to have hypertension (40 vs. 28%) and to be older (mean age 54 vs. 48 years).

Discussion

Epistaxis is a potential complication after endoscopic skull base surgery that has the potential for severe morbidity. Bleeding from large caliber vessels such as the carotid and sphenopalatine arteries can result in rapid and severe postoperative hemorrhage. Also, as many of these patients have had large areas of the skull base reconstructed with precisely placed tissue and bone and use of a nasoseptal flap, nasal packing can be much more challenging to perform due to fears of disrupting the repair and causing a CSF leak.

In this study, we found a 3% rate of postoperative epistaxis. Fortunately, the bleeding was usually minor and could typically be managed with careful anterior nasal packing alone by an otolaryngology-trained physician. One patient required chemical cautery with silver nitrate on postoperative day 9. After anesthetizing the nose with 4% lidocaine and 0.25% phenylephrine spray, cautery was performed using a 30-degree endoscope and Frazier-tip suction followed by Vaseline gauze packing. No complications were associated with nasal packing or cautery in the outpatient setting or emergency room. However, 1.5% of the patients in this study still required intervention in the operating room to control bleeding. After surgical management of epistaxis, there were no recurrent episodes of bleeding and no subsequent CSF leaks. Both patients who presented with intrasellar blood and associated visual changes had improved vision after epistaxis management.

In our cohort of 330 patients, no statistically significant risk factors were identified for postoperative epistaxis, including hypertension, the use of preoperative blood thinners, a history of surgery or use of a nasoseptal flap. One might expect a higher rate of epistaxis after use of a nasoseptal flap due to the additional dissection required for flap harvest. In the initial description of the nasoseptal flap, Hadad et al. [4] reported a 2.3% rate of epistaxis in 43 patients. In our series, 70% of the patients that had epistaxis had a nasoseptal flap used as part of the surgery, but this was not statistically significant. In these patients, the source of bleeding could not be identified in 71% of the cases (5/7), and in 1 case the source of bleeding was actually found to be the contralateral sphenopalatine artery.

Alcohol use was also not a risk factor for bleeding in this study; we found that none of the patients who admitted to at least weekly alcohol use had postoperative epistaxis. In fact, our data suggests that use of alcohol might be a negative risk factor for postoperative epistaxis after endoscopic skull base surgery (p = 0.04). This is surprising, as heavy alcohol use has previously been associated with spontaneous epistaxis [5]. Even a moderate amount of red wine has been reported to be associated with decreased blood clotting by decreasing plasma fibrinogen and factor VIIc and increasing tissue plasminogen activator [6]. To our knowledge, there is no published study suggesting that alcohol intake may decrease the risk of bleeding.

As is expected, the 3% rate of epistaxis after endonasal endoscopic surgery for skull base tumors is slightly higher than the rate of postoperative epistaxis rate after endoscopic sinus surgery (ESS) for chronic rhinosinusitis and polyposis. This is most likely a result of the more extensive dissection around arteries and the presence of large mucosal defects that leave multiple bleeding edges. In a recent study of over 3,000 patients undergoing ESS for chronic sinusitis, the rate of postoperative epistaxis requiring treatment was 0.8% [7]. In another more recent study by Ramakrishnan et al. [8], only 0.76% of 58,732 patients who underwent ESS had hemorrhage requiring transfusion, with a majority of cases occurring within the first 30 days. No patient in our study required transfusion.

In our study, nasal packing alone was sufficient to stop the epistaxis in 4 of the 10 patients. In only 2 cases was the sphenopalatine artery identified as the source of bleeding. One of these patients required chemical cautery in addition to nasal packing, and the other patient required hemostatic control and cauteryization in the operating room. Five patients required surgical management in the operating room to control the bleeding. In 4 of these cases, the sites of bleeding were mucosal and were usually from the cut edges of the septum or turbinates. No initial episode of epistaxis occurred after 10 days in any of the patients, suggesting that the risk of bleeding 2 weeks after surgery is low, despite a potential for bleeding occurring with postoperative debridement.

Conclusion

This study confirms that the risk of epistaxis after endoscopic skull base surgery is low, and is similar to that of sinonasal surgery for benign and inflammatory condi-
tions. While there is a potential for significant morbidity associated with postoperative bleeding in this patient population, epistaxis is usually mild, and patients can usually be managed as outpatients with nasal packing or cautery.

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References