Risk factors for post-carotid endarterectomy hematoma formation

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Purpose: To identify risk factors for post-carotid endarterectomy (CEA) hematoma formation and establish the incidence of this complication at The Ottawa Hospital - Civic Campus (TOH-CC).

Methods: A chart review of all patients who underwent CEA at TOH-CC from January 1, 1996 to December 31, 1997 was completed. Identified cases of post-CEA wound hematoma were entered into a case-control study using age and sex-matched controls from within the cohort. These matched pairs were assessed for 31 potential risk factors including demographic details, co-existing medical conditions, preoperative medications, intraoperative management, and postoperative parameters. Risk factors associated with post-CEA hematoma with $P < 0.05$ were entered into a backward step-wise logistic regression model for multivariate analysis.

Results: Charts from 249 patients were reviewed and 29 cases of post-carotid endarterectomy hematoma were identified (12% incidence). Six of the initial 31 potential risk factors emerged as univariate predictors of post-CEA hematoma formation ($P < 0.05$): general anesthesia, carotid shunt placement, intraoperative hypotension, non-reversal of heparin, neurosurgery service, and preoperative aspirin use. Following logistic regression only non-reversal of heparin, intraoperative hypotension, and carotid shunt placement were identified as multivariate predictors of post-CEA hematoma formation. More time was spent in critical care settings (ICU/PACU) ($P < 0.01$) and there was increased perioperative mortality ($P = 0.04$) within the hematoma group.

Conclusions: Post-CEA hematoma formation is associated with increased morbidity and mortality. Non-reversal of heparin, intraoperative hypotension, and carotid shunt placement are multi-variate predictors of post-CEA hematoma formation.

Objectif : Identifier les facteurs de risques de formation d'un hématome post-endartériectomie carotidienne (EAC) et établir son incidence à The Ottawa Hospital - Civic Campus (TOH-CC).

Méthode : On a révisé les dossiers de tous les patients qui ont subi une EAC à TOH-CC du 1 janvier 1996 au 31 décembre 1997. Les cas d'hématome post-EAC ont été identifiés et inscrits à une étude cas-témoins appariés selon l'âge et le sexe à partir des sujets de la cohorte. Ces paires de sujets ont été évalués selon 31 facteurs de risque potentiels incluant les données démographiques, la coexistence de problèmes médicaux, la médication préopératoire, la prise en charge peropératoire et les paramètres postopératoires. Les facteurs de risque associés à l'hématome post-EAC avec $P < 0,05$ ont été introduits dans un modèle rétrospectif de régression logistique multifactorielle en paliers.

Résultats : Les dossiers de 249 patients ont été examinés et 29 cas d'hématome post-endartériectomie carotidienne ont été identifiés (incidence de 12 %). Six des 31 facteurs de risque potentiels désignés au début sont apparus comme des prédicteurs unifactoriels de la formation d'hématome post-EAC ($P < 0,05$) : l'anesthésie générale, la mise en place d'un shunt carotidien, l'hypotension peropératoire, le non-renversement de l'héparine, le service de neurochirurgie et l'utilisation d'aspirine préopératoire. À la suite de la régression logistique, seules la non-inversion de l'héparine, l'hypotension peropératoire et la mise en place du shunt carotidien sont apparus comme des prédicteurs multifactoriels. Le temps passé à l'unité des soins intensifs (USI/salle de réveil) a été plus long ($P < 0,01$) pour le groupe présentant un hématome et la mortalité périopératoire était plus élevée ($P = 0,04$).

Conclusion : La formation d'un hématome post-EAC est associée à une morbidité et à une mortalité croissantes. Le non-renversement de l'héparine, l'hypotension peropératoire et la mise en place d'un shunt carotidien sont des prédicteurs multifactoriels de la formation d'un hématome post-EAC.

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STROKE is the most frequent cause of neurological disability in adults and the third leading cause of death over the age of 65 yr in Canada.[1,2] In 1991, the North American Symptomatic Carotid Endarterectomy Trial (NASCET) and European Carotid Surgery Trial (ECST) showed a reduction in the incidence of stroke and death in patients with high grade (70-99%) carotid stenosis treated with carotid endarterectomy (CEA) *vs* medical management.[3,4] Since that time, the use of CEA in the treatment of atherosclerotic carotid artery stenosis has risen markedly [5] and updated CEA guidelines were published by the American Heart Association (AHA) in 1995.[6]

The attendant morbidity and mortality of a procedure must be considered along with the proposed benefits in order to assess its net benefit for a patient. One source of CEA surgical risk is airway compromise secondary to post-CEA hematoma formation.[7-10] Reduction in airway cross-sectional area and volume can be demonstrated radiologically in all patients following CEA.[9] The published incidence of clinically significant post-CEA hematomas typically ranges from 1-3% but rates as high as 26% have been reported.[8-13] Risk factor identification may improve the anticipation, prevention and management of these events, thereby reducing a patient's operative risk. The purpose of this study was to identify risk factors for post-CEA hematoma formation and establish the incidence of this complication at The Ottawa Hospital - Civic Campus (TOH-CC).

Methods

Following Research Ethics Committee approval, the medical records computer database and search codes were used to identify all patients who had undergone CEA at TOH-CC from 01/01/96 to 31/12/97. The charts of these patients were reviewed and demographic information including: surgeon, anesthesiologist, anesthetic technique, and general outcome data (post-CEA hematoma/stroke/death) was gathered for each patient. For the purposes of this study, post-CEA hematoma was defined as postoperative airway compromise in CEA patients severe enough for the PACU nurses to contact an anesthesiologist for assessment, with or without subsequent airway intervention (i.e. intubation and/or surgical reexploration). A case-control study was conducted on the cases of post-CEA hematoma using age and sex-matched controls from within the cohort. These matched pairs were assessed for 31 potential risk factors including demographic details, co-existing medical conditions, preoperative medications, intraoperative management, and postoperative parameters (Appendix-I). Univariate analysis of factors associated with post-CEA hematoma was conducted using unpaired Student's t test, Fisher's exact test, and chi-square statistic where appropriate. A P value of < 0.05 was considered to represent an association with post-CEA hematoma. Repeated univariate statistical testing may lead to type I error and cannot differentiate between correlated risk factors. Multivariate analysis using logistic regression may be used to reduce both such errors. Factors identified in the univariate analysis with an association to post-CEA hematoma were entered into a backward step-wise logistic regression analysis. Odds ratios and 95% confidence intervals (CI) describe the association between significant variables and post-CEA hematoma in both the univariate and multivariate analysis. Statistical analysis was completed using SPSS 7.0 for Windows, Chicago, IL.

Results

Two-hundred and forty-nine charts were reviewed and 29 hematomas, eight strokes and three deaths were identified (Table I). Of the 29 patients who developed hematomas, 16 (55%) required reintubation and five (17%) underwent surgical re-exploration. The mean time from arrival in PACU to hematoma development was six hours and 41 min (± 337 min) but ranged from 45 min to three days. More time was spent in critical care settings (ICU and/or PACU) by the patients who developed hematomas (3.1 ± 2.9 *vs* 1.0 ± 0.2 days; $P < 0.01$). A trend toward longer hospitalization in patients suffering post-CEA hematomas was noted (12.0 ± 18.5 dy *vs* 5.3 ± 5.5 dy; $P = 0.07$) but did not reach statistical significance. An increase in mortality was also associated with post-CEA hematoma formation ($P = 0.04$).

Five of the eight strokes in this study were identified within six hours of the CEA. Five were thromboembolic in origin, one was hemorrhagic and two were identified clinically without further radiographic clarification of their etiology. The strokes were evenly distributed between those patients who had or had not received protamine.

Of the three perioperative deaths in this study, one was cardiac in origin (myocardial ischemia/cardiogenic shock), and two resulted from cerebrovascular accidents (one thromboembolic, one hemorrhagic). The hemorrhagic stroke occurred in a 63 yr old man who was re-heparinized for unstable angina following CEA. He was found to be unresponsive several hours after heparinization, and an immediate CT scan revealed a massive intracerebral hemorrhage. No further interventions could be offered by the neurology or neurosurgery services and the patient died three days later. The thromboembolic stroke occurred in a

TABLE I General Demographic and Outcome Information

Characteristic	*Cohort*	*Cases*	*Controls*
Number (n)	249	29	29
Age	68 ± 10	68 ± 8	68 ± 8
Male/Female (%)	65/35	48/52	48/52
Neuro/Vascular (n)	147/102	22/7	14/15
Regional/GA (n)	117/132	8/21	22/7
Stroke (n/%)	8 (3%)	3 (10%)	2 (7%)
Death (n/%)	3 (1%)	2 (7%)	0
Hematoma (n/%)	29 (12%)	29 (100%)	0

Neuro = Neurosurgery Service; Vascular = Vascular Surgery Service;
Regional = Regional Anesthesia; GA = General Anesthesia.

TABLE II Univariate Predictors of Post-Carotid Endarterectomy Hematoma Formation

Risk Factor	*Odds Ratio*	*Confidence Interval*	*P*
General Anesthesia	8.3	2.5 - 26.8	<0.01
Carotid Shunt Placement	6.0	1.9 - 18.8	<0.01
Intraoperative Hypotension	5.8	1.9 - 18.1	<0.01
Non-Reversal of Heparin	4.1	1.3 - 13.1	0.01
Neurosurgery Service	3.4	1.1 - 10.3	0.03
Preoperative Aspirin Use	3.4	1.0 - 11.4	0.04

TABLE III Multivariate Predictors of Post-Carotid Endarterectomy Hematoma Formation

Risk Factor	*Odds Ratio*	*Confidence Interval*	*P*
Non-Reversal of Heparin	3.7	1.0 - 13.4	0.04
Intraoperative Hypotension	3.6	1.0 - 13.1	0.05
Carotid Shunt Placement	3.5	1.0 - 13.0	0.05

58 yr old man who was found to have a dense hemiplegia immediately following emergence from general anesthesia. Despite urgent re-exploration, evacuation of intraluminal carotid artery thrombus (no protamine had been given), and postoperative heparinization, the patient failed to recover and died from his massive thromboembolic stroke eight days later.

Six of the initial 31 potential risk factors were identified as univariate predictors of post-CEA hematoma formation ($P < 0.05$): general anesthesia, carotid shunt placement, intraoperative hypotension, non-reversal of heparin, neurosurgery service, and preoperative aspirin use (Table II). Though there were more hematomas within the neurosurgery service ($P = 0.03$), no surgeon from either service had a disproportionate excess of post-CEA hematomas ($P = 0.35$) and no correlation was made between any anesthesiologist and hematoma formation. A trend toward correlation of intra/postoperative hypertension with hematoma formation was also noted, but neither reached statistical significance ($P = 0.07$ and 0.06 respectively).

When the six univariate predictors of post-CEA hematoma formation were entered into a backward step-wise logistic regression model, only three factors remained as multivariate predictors of post-CEA hematoma formation: non-reversal of heparin, intraoperative hypotension, and carotid shunt placement (Table III).

Discussion

Carotid endarterectomy has been established as the gold-standard treatment for patients with severe symptomatic or asymptomatic carotid stenosis provided the associated risk of stroke and/or death is < 3% in asymptomatic patients and < 6% in symptomatic patients.[14] The incidences of CEA associated stroke and death are 1.0 - 8.6%[15–17] and 0.8 - 3.6%[15,18,19] respectively. The 3% incidence of stroke and 1% mortality rate in this study compares favourably with the published CEA outcome data.

Neck hematoma formation is another relatively common source of post-CEA morbidity and mortality because of the rapid, potentially life-threatening airway compromise that may develop.[7–10] Wide variance in the reported incidence of post-CEA hematoma primarily results from differing inclusion criteria. When post-CEA hematoma is defined as postoperative neck swelling/airway obstruction requiring tracheal reintubation/wound reexploration,[8,10–13] an incidence of 1-3% is commonly cited. If, however, it is defined as a radiographically evident reduction in airway cross-sectional area/volume, the reported incidence rises to 26.3%.[9] Post-CEA hematoma was defined in this study as: postoperative airway compromise in CEA patients severe enough for the PACU nurses to contact an anesthesiologist for assessment, with or without subsequent airway intervention. This definition was chosen because some degree of neck hematoma/edema is present radiographically in all post-CEA patients,[9] and clinically evident airway compromise may rapidly follow the onset of symptoms (even preceding marked external neck swelling).[7–10] Consequently, the 12% incidence of post-CEA hematoma in this study is intermediate between incidences cited elsewhere.[8–13] This study found that in over half of the cases where anesthesia staff were called for airway reassessment in PACU, intubation or reintubation of the patient's airway was required.

From a group of 31 potential risk factors, this study identified general anesthesia, carotid shunt placement, intraoperative hypotension, non-reversal of heparin, neurosurgery service, and preoperative aspirin use as univariate predictors of post-CEA hematoma formation. Following logistic regression, only non-reversal of heparin, intraoperative hypotension, and carotid shunt placement remained as multivariate predictors of post-CEA hematoma formation. The neurosurgery service and general anesthesia most likely emerged as univariate risk factors because the neurosurgeons at TOH-CC favour general anesthesia with frequent carotid shunt placement, non-reversal of heparin and/or postoperative heparinization.

Previous studies have found that approximately 60-80% of post-CEA hematomas result from capillary oozing and about 20-40% occur secondary to arterial bleeding.[8,10,12] One possible mechanism for the association of intraoperative hypotension with post-CEA hematoma is that hypotension during closure may give a false impression that adequate hemostasis has been achieved. Co-existing diseases such as hypertension, smoking and COPD may complicate a smooth emergence from anesthesia. Hypertensive swings and coughing at the time of extubation may induce bleeding at the surgical site that was not apparent at lower intraoperative blood pressures, resulting in hematoma formation.

Temporary intraluminal carotid shunt placement is used routinely by many surgeons to ensure adequate cerebral perfusion pressure (CPP) during carotid cross-clamping.[15,16,20] In the context of CEA performed under general anesthesia without CPP monitoring, intraluminal shunts reduce the incidence of perioperative neurological deficits.[20] However, these shunts may result in neurological complications due to emboli from atheromatous plaques, carotid artery dissection, or thrombosis secondary to intimal damage caused by the shunt.[21–24] The reasons for the association of carotid shunt placement with post-CEA hematoma are unknown, but may be related to the additional arteriotomies created by the placement of the shunt.

This study identified an association between failure to reverse heparin at the end of surgery and the development of post-CEA hematoma. Thrombus formation at the operative site and subsequent arterial thromboembolism has been cited as the primary cause of post-CEA stroke.[15,27–29] Therefore, withholding protamine has been advocated as a means to reduce the incidence of post-CEA stroke.[30] Wells *et al.* found that, although thrombin generation is enhanced by protamine administration, considerable thrombin generation persists despite full biochemical anticoagulation with heparin.[31] Studies using animal models to examine the impact of reversal or non-reversal of heparin on postoperative thrombus formation have yielded mixed results.[32–34] Treiman *et al.* conducted a retrospective study on the effect of reversal or non-reversal of heparin on the incidence of postoperative stroke and wound hematoma in 697 CEA patients. An increase in post-CEA wound hematoma occurred in the patients who did not receive protamine. A trend toward reduction of post-CEA stroke was noted in those patients who received protamine,.[35] These results suggest that the administration of protamine should not adversely affect the postoperative stroke rate while reducing the incidence of post-CEA hematoma.

Cervical plexus block is a well established anesthetic technique for CEA.[21–23,25] Regional anesthetic techniques offer the advantages of direct intra-operative cerebral function monitoring, selective shunting (based on the presence or absence of neurological deterioration with carotid cross-clamping) and greater intra/postoperative hemodynamic stability.[11,21–23,26]

Decisions based on the results of this study must be tempered with the knowledge that case-control studies are retrospective and are prone to bias (selection, measurement) and overmatching. Retrospective studies can establish association (i.e. post-CEA hematomas with increased peri-operative mortality) but prospective studies are required to determine causation. Despite its limitations, the risk factors identified by this study may prompt re-evaluation of current anesthetic and surgical practice patterns, and target areas for further research. In particular, prospective studies are needed to resolve the controversy surrounding the relationship between heparin reversal or non-reversal and post-CEA thromboembolus formation. In the mean time, the reversal of heparin is not associated with an increased incidence of post-CEA stroke, and routine reversal of heparin should be considered to reduce the likelihood of post-CEA hematoma. Finally, when compared with general anesthesia, regional anesthesia for CEA provides greater intraoperative and postoperative hemodynamic stability, direct intraoperative monitoring of cerebral function, and a greater opportunity for selective use of intraluminal shunting. Further prospective studies are needed to confirm that increased use of regional anesthesia for CEA may, by reducing the occurrence of these risk factors, decrease the incidence of post-CEA hematoma along with its potentially devastating complications.

APPENDIX Potential Risk Factors for Post-Carotid Endarterectomy Hematoma Formation

OR Team	*PMH and FH*	*Intraoperative Factors*	*Postoperative Factors*
Surgeon	Hypertension	General Anesthesia	PACU admission BP
Service	Coagulopathy	Regional Anesthesia	Hypertension Rx
Anesthetist	FH of Coagulopathy	Hypotension requiring Rx	Postoperative Heparin
	Preoperative INR	Hypertension requiring Rx	Postoperative INR
Pre-Op Meds	Preoperative PTT	Protamine Use	Postoperative PTT
Aspirin	Smoking	Surgicell Use	Time to Hematoma Dx
NSAID	COPD	Thrombostat Use	
Ticlopidine	Obesity	Carotid Shunt Placement	
Coumadin		Surgical Drain Placement	
Heparin		Operative Time	

OR = Operative; Pre-Op Meds = Preoperative Medications; PMH = Past Medical History; FH = Family History; Hx = History; COPD = Chronic Obstructive Pulmonary Disease; Rx = Treatment; BP = Blood Pressure; Dx = Diagnosis.

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