Subdural hematoma after thoracoabdominal aortic aneurysm repair: An underreported complication of spinal fluid drainage?

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Objective: **Cerebrospinal fluid (CSF) drainage is a commonly used adjunct to thoracoabdominal aortic aneurysm (TAAA) repair that improves perioperative spinal cord perfusion and thereby decreases the incidence of paraplegia. To date, little data exist on possible complications, such as subdural hematoma caused by stretching and tearing of dural veins, should CSF drainage be excessive. We reviewed our experience with patients in whom postoperative subdural hematomas were detected.**

Methods: **The records of 230 patients who underwent TAAA repair at the Johns Hopkins Hospital between January 1992 and February 2001 were reviewed.**

Results: **Eight patients had subdural hematomas (3.5%). The four men and four women had a mean age of 60.6 years; two of these patients had a connective tissue disorder. All patients had lumbar drains placed before surgery, including one patient who underwent operation emergently for rupture. Drains were set to allow drainage for CSF pressure greater than 5 cm H_2O in all but one patient set for 10 cm H_2O; spinal cooling was not performed in any patient. All drains were removed on the third postoperative day. In patients in whom subdural hematomas developed, the mean amount of CSF removed after surgery was 690 ± 79 mL, which was significantly greater than the amount drained from patients in whom subdural hematomas did not develop (359 ± 24 mL; $P = .0013$, Mann-Whitney *U* test). Six patients had postoperative subdural hematomas detected during hospitalization (mean postoperative day, 9.3; range, 2 to 16), and two patients were seen in delayed fashion after discharge from the hospital at 1.5 and 5 months. Four patients died of the subdural hematoma (50%); only one of these patients had neurosurgical intervention. All four survivors responded to neurosurgical intervention and are neurologically healthy. Two patients, both of whom were seen in delayed fashion, needed a lumbar blood patch. Multivariate logistic regression identified the volume of CSF drained as the only variable predictive of occurrence of subdural hematoma ($P = .01$).**

Conclusion: **Subdural hematoma is an unusual and potentially catastrophic complication after TAAA repair. Prompt recognition and neurosurgical intervention is necessary for survival and recovery after acute presentation. Epidural placement of a blood patch is recommended if a chronic subdural hematoma is detected. Care should be taken to ensure that excessive CSF is not drained perioperatively, and higher (10 cm H_2O) lumbar drain popoff pressures may be necessary together with meticulous monitoring of patient position and neurologic status. (J Vasc Surg 2002;36:47-50.)**

Spinal cord ischemia, either paraplegia or paraparesis, is one of the disastrous complications after thoracoabdominal aortic aneurysm (TAAA) repair. Modern series report approximately a 5% to 15% incidence rate of spinal cord ischemia after TAAA repair,[1-7] with significantly decreased survival rates among patients with spinal cord injury.[2,4] Many clinical strategies to prevent spinal cord ischemia have been investigated, including identification and reimplantation of critical spinal cord intercostal tributary arteries, distal aortic perfusion, systemic or regional hypothermia, and minimization of spinal cord oxygen demand pharmacologically. However, no optimal strategy has emerged.

Cerebrospinal fluid (CSF) drainage is a commonly used adjunct to TAAA repair that may improve perioperative spinal cord perfusion[8-10] and subsequently decrease the incidence of spinal cord ischemia.[11-14] However, to date, only one report exists on possible complications of CSF drainage, such as subdural hematoma.[15] We reviewed our experience with patients who underwent TAAA repair in whom postoperative subdural hematomas were detected.

METHODS

The records of 230 patients who underwent TAAA repair at the Johns Hopkins Hospital between January 1992 and February 2001 were reviewed retrospectively. Lumbar drains were routinely placed the evening before elective surgery by the anesthesia team or, if fluoroscopic guidance was needed, by the neurosurgical and interventional neuroradiology teams. The popoff level of the CSF drain was routinely 5 to 10 cm H_2O, as set from the ear canal, depending on the attending surgeon preference. CSF drainage began in the operating room and continued

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Table I. Patients with subdural hematoma after TAAA repair

Patient	*Age (y)*	*Race*	*Gender*	*Risk factor*	*Crawford classification*	*CSF popoff*	*CSF drained*	*Postop day*	*Presentation*	*Symptom*	*Intervention*	*Outcome*
1	26	W	M	Marfan's	I	5	1067	164	Chronic	HA, V	NS OR, lumbar patch	Alive
2	73	B	M	Hypertension	III	5	773	16	Acute	FDP	None	Died
3	80	W	F	Hypertension	IV	5	548	11	Acute	Aphasia	NS OR	Alive
4	74	W	F	Hypertension	III	5	513	13	Acute	Somnolent	NS OR	Alive
5	38	W	M	Ehler-Danlos	II	5	974	4	Acute	BD	NS OR	Died
6	64	B	F	Hypertension	III	5	581	10	Acute	Weakness	None	Died
7	82	W	F	Hypertension	II	5	566	2	Acute	BD	None	Died
8	48	W	M	Hypertension	II	10	497	45	Chronic	HA, V	Lumbar patch	Alive

CSF popoff is valve level in cm H_2O; *CSF drained* is total volume of CSF drained perioperatively in mL; *postop day* is postoperative day subdural hematoma was detected.
W, White; *B*, black; *M*, male; *F*, female; *HA*, headache; *V*, vomiting; *FDP*, fixed and dilated pupils; *BD*, bloody CSF drainage; *NS OR*, neurosurgical operative drainage.

for 3 days after surgery. Spinal cooling or instillation of pharmacologic agents was not performed in any patient. All patients had distal aortic perfusion with sequential aortic clamping performed as part of the TAAA repair, and all patients received heparin (100 mg/kg) during surgery. Routine anticoagulation therapy was not performed after surgery in any patient. The patient head position was maintained at no more than 30 degrees upright perioperatively until the patient was alert and mental status could be checked, regardless of whether the drain was present or not. The correct drain position was maintained regardless of head position. Routine postoperative screening for asymptomatic subdural hematomas was not performed; rather, all patients with altered mental status or paresis underwent head computed tomographic scans that were positive for the eight patients reported.

Comparison of the factors between patients with and without subdural hematomas was performed with the Mann-Whitney *U* test for nonparametric variables and the Fisher exact test for categoric variables. Multivariate analysis was performed with logistic regression. *P* values were considered significant if they were .05 or less.

RESULTS

Eight patients had subdural hematomas (Table I), for an incidence rate of 3.5% after TAAA repair. There were four men and four women. Six patients had atherosclerotic disease (mean age, 70.2 years), and two patients had connective tissue disorders (mean age, 32 years). The Crawford classification of the TAAAs were one type I, three type II, three type III, and one type IV. All patients had lumbar drains placed before surgery, including one patient who underwent operation emergently for rupture (patient 6). Drains were set to allow drainage of CSF at pressures greater than 5 cm H_2O in seven patients and greater than 10 cm H_2O in the other (patient 8). All drains were removed on the third postoperative day.

Six patients had an acute presentation of a subdural hematoma, at a range from postoperative day 2 to 16 (mean, 9.3). Four of these six patients died of the subdural hematoma (67%); only one of the four patients who died had neurosurgical intervention. Both survivors of acute subdural hematoma (33%), however, responded to neurosurgical decompression and are neurologically healthy.

Two patients had subdural hematomas in a delayed fashion after discharge from the hospital, at 1.5 and 5 months. These patients needed a lumbar epidural space blood patch to seal their chronic CSF leak. No patient with an acute presentation had a blood patch performed. Both patients with chronic subdural hematomas responded clinically to the blood patch, with decompression in one patient, and are neurologically healthy.

Comparison between the eight patients in whom subdural hematomas developed and those patients in whom hematomas did not develop after TAAA repair is shown in Table II. No statistically significant difference was seen in the mean age, percentage of women, incidence rate of connective tissue disorders, or occurrence of postoperative paraplegia in patients with or without subdural hematomas. The operative number of packed red blood cell, but not platelet, transfusions was greater in patients in whom subdural hematoma ultimately developed. There was also a greater amount of CSF removed perioperatively in patients in whom subdural hematomas developed compared with the amount drained from patients in whom subdural hematomas did not develop (Table II). Patients with subdural hematomas were more likely to die during hospitalization than patients without subdural hematoma (Table II).

Multivariate analysis of risk factors that affect the occurrence of postoperative subdural hematoma is reported in Table III. The only factor that was statistically significant and predictive of occurrence of subdural hematoma was the volume of CSF drained via the lumbar drain. With the mean amount of CSF drained from patients with and without subdural hematomas (690 mL and 359 mL, respectively; Table II), the multivariate analysis predicted an odds ratio of 7.29 for occurrence of subdural hematoma in this patient series (Table III).

Table II. Comparison of patients with and without subdural hematoma after TAAA repair

	Subdural hematoma	*No hematoma*	P *value*
No. of patients	8	222	
Age (y)	61.4 ± 6.9	66.7 ± 0.8	.78*
Female gender	50%	39%	.72†
Connective tissue disorder	2 (25%)	28 (12.6%)	.29†
EBL (L)	18.7 ± 5.0	11.8 ± 0.8	.06*
PRBC transfused (U)	19.1 ± 2.3	14.2 ± 0.7	.04*
Platelets transfused (U)	8.3 ± 1.9	11.9 ± 0.7	.13*
CSF drained (mL)	690 ± 79	359 ± 24	.0013*
Permanent paralysis	1 (12.5%)	11 (5.0%)	.35†
In-hospital death	4 (50%)	34 (15.3%)	.03†

*Mann-Whitney *U* test.
†Fisher exact test.
EBL, Estimated blood loss; *PRBC*, packed red blood cells.

Table III. Multivariate analysis of factors affecting occurrence of subdural hematoma

Factor	P *value*	*Odds ratio*	*95% CI*
Age	.31	1.04	0.96-1.13
Connective tissue disorder	.43	3.27	0.18-60.77
Gender	.60	0.60	0.09-4.02
Race	.53	2.26	0.18-27.71
Hypertension	.24	0.17	0.01-3.27
COPD	.99	0.01	0-∞
Permanent paralysis	.50	0.31	0.01-9.20
PRBC transfused (U)	.19	1.06	0.97-1.17
Platelets transfused (U)	.17	0.85	0.68-1.07
CSF drained (mL)	.01	1.006	1.001-1.01

COPD, Chronic obstructive pulmonary disease; *PRBC*, packed red blood cells.

DISCUSSION

Subdural hematoma is an unusual and potentially catastrophic complication after TAAA repair, with an incidence rate of 3.5% in our series. We report eight cases of patients with postoperative subdural hematomas, six of whom had an acute presentation, with a 67% mortality rate. Two patients were seen after hospital discharge with chronic subdural hematomas and needed lumbar epidural blood patches to control the chronic CSF leak. Multivariate analysis identified the volume of CSF drained perioperatively as the only factor predictive of occurrence of postoperative subdural hematoma (Table III). Multivariate analysis disclosed that excess drainage was associated with a seven-fold increased risk of subdural hematoma.

Subdural hematoma after TAAA repair has only been reported once before.[15] In that case report, a patient was seen acutely with a subdural hematoma on postoperative day 5; neurosurgical intervention was declined, with subsequent patient death. The lumbar drain popoff pressure was 10 mm Hg, equivalent to approximately 13 cm H_2O, but no report was made of the volume of CSF drained. The subdural hematoma was attributed to the large needle size (16 gauge) used to puncture the dura with subsequent CSF leakage through the drainage site, compounded by change in patient position. However, subdural hematomas have been reported after dural puncture with smaller gauge needles.[16,17] Furthermore, we routinely use a 16-gauge needle to puncture the dura for our lumbar drain placement, and most of our patients do not have subdural hematomas develop.

Although CSF drainage is a recognized adjunct to TAAA repair, few guidelines exist for proper CSF drainage pressures. We initially routinely set our lumbar drain popoff pressures at 10 cm H_2O but questioned whether setting the popoff pressure lower, at 5 cm H_2O, would increase CSF drainage and thereby decrease the incidence rate of paraplegia. On the basis of this experience, we speculate that excessive CSF drainage stretches and tears dural veins, leading to a subdural hematoma. Subdural hematomas have been previously reported as a consequence of excessive CSF drainage because of lumbar puncture[18-20] or drainage catheters[21] and have been related to CSF hypovolemia.[22] Although subdural hematomas have been associated with disseminated intravascular coagulation,[23] no patient in this series had evidence of disseminated intravascular coagulation (data not shown).

Our estimate of a 3.5% incidence rate of subdural hematomas after TAAA repair is likely to be an underestimate of the true incidence rate of this complication. This retrospective study identified only patients with symptomatic subdural hematomas because asymptomatic patients were not tested. However, clinically significant acute subdural hematomas, with their rapidly progressing and dramatic presentations, were likely to be identified.

All six patients in this series with acute subdural hematoma had the CSF drain popoff valve set at 5 cm H_2O. The mortality rate was 67% in this subgroup, with neurosurgical drainage offering the only hope of survival (Table I). Consequently, this experience has led us to recommend routinely setting the CSF catheter to drain at 10 cm H_2O. We also maintain the patient head position at no more than 30 degrees upright perioperatively, until the patient is alert and the mental status can be checked, to avoid potential fluctuations in CSF and cerebral perfusion pressure.[24,25]

No patient with a drain popoff pressure set at 10 cm H_2O had an acute subdural hematoma; the only patient in this series who had a subdural hematoma with a drain popoff pressure set at 10 cm H_2O had a chronic presentation (Table I). Chronic presentation of a subdural hematoma is likely the result of the same mechanism as an acute presentation, with CSF hypovolemia occurring slowly through the dural puncture site. This explains the rationale for application of an epidural blood patch to seal the puncture site[20,22] and the occurrence of the subdural hematoma independently of the perioperative drain popoff pressure setting.

We report a 3.5% incidence rate of subdural hematomas after TAAA repair and have identified excessive CSF drainage as a risk factor for its occurrence. Changes in consciousness or new onset of paresis should prompt recognition and neurosurgical intervention that is necessary for survival and recovery. Epidural placement of a blood patch is recommended to stop the chronic leakage of CSF at the lumbar puncture site if a chronic subdural hematoma is detected. Care should be taken to ensure that excessive CSF is not drained perioperatively, and higher (10 cm H_2O) lumbar drain pressures and meticulous monitoring of patient position and neurologic status may be necessary.

REFERENCES

1. Hollier LH, Money SR, Naslund TC, Proctor CD Sr, Buhrman WC, Marino RJ, et al. Risk of spinal cord dysfunction in patients undergoing thoracoabdominal aortic replacement. Am J Surg 1992;164:210-4.
2. Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg 1993;17:357-70.
3. Grabitz K, Sandmann W, Stuhmeier K, Mainzer B, Godehardt E, Ohle B, et al. The risk of ischemic spinal cord injury in patients undergoing graft replacement for thoracoabdominal aortic aneurysms. J Vasc Surg 1996;23:230-40.
4. Cambria RP, Davison JK, Zannetti S, L'Italien G, Atamian S. Thoracoabdominal aneurysm repair: perspectives over a decade with the clamp-and-sew technique. Ann Surg 1997;226:294-305.
5. Coselli JS, LeMaire SA, Miller CC III, Schmittling ZC, Koksoy C, Pagan J, et al. Mortality and paraplegia after thoracoabdominal aortic aneurysm repair: a risk factor analysis. Ann Thorac Surg 2000;69:409-14.
6. Acher CW, Wynn MM, Hoch JR, Kranner PW. Cardiac function is a risk factor for paralysis in thoracoabdominal aortic replacement. J Vasc Surg 1998;27:821-30.
7. Safi HJ, Miller CC III, Carr C, Iliopoulos DC, Dorsay DA, Baldwin JC. Importance of intercostal artery reattachment during thoracoabdominal aortic aneurysm repair. J Vasc Surg 1998;27:58-68.
8. Miyamoto K, Ueno A, Wada T, Kimoto S. New and simple method of preventing spinal cord damage following temporary occlusion of the thoracic aorta by draining the cerebrospinal fluid. J Cardiovasc Surg 1960;1:188-97.
9. Blaisdell FW, Cooley DA. The mechanism of paraplegia after temporary aortic occlusion and its relationship to spinal fluid pressure. Surgery 1962;57:351-5.
10. Saether OD, Juul R, Aadahl P, Stromholm T, Myhre HO. Cerebral haemodynamics during thoracic and thoracoabdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg 1996;12:81-5.
11. McCullough J, Hollier LH, Nugent M. Paraplegia after thoracic aortic occlusion: influence of cerebrospinal fluid drainage. Experimental and early clinical results. J Vasc Surg 1988;7:153-60.
12. Hill AB, Kalman PG, Johnston KW, Vosu HA. Reversal of delayed-onset paraplegia after thoracic aortic surgery with cerebrospinal fluid drainage. J Vasc Surg 1994;20:315-7.
13. Safi HJ, Miller CC III, Azizzadeh A, Iliopoulos DC. Observations on delayed neurologic deficit after thoracoabdominal aneurysm repair. J Vasc Surg 1997;26:616-22.
14. Coselli JS, LeMaire SA, Schmittling ZC, Koksoy C. Cerebrospinal fluid drainage in thoracoabdominal aortic aneurysm surgery. Semin Vasc Surg 2000;13:308-14.
15. McHardy FE, Bayly PJM, Wyatt MG. Fatal subdural haemorrhage following lumbar spinal drainage during repair of thoraco-abdominal aneurysm. Anaesthesia 2001;56:168-70.
16. Pryle BJ, Cater JA, Cadoux-Hudson T. Delayed paraplegia following spinal anaesthesia. Spinal subdural haematoma following dural puncture with a 25 G pencil point needle at T12-L1 in a patient taking aspirin. Anaesthesia 1996;51:263-5.
17. Cantais E, Behnamou D, Petit D, Palmier B. Acute subdural hematoma following spinal anesthesia with a very small spinal needle. Anesthesiology 2000;93:1354-6.
18. Ruff RL, Dougherty JH Jr. Complications of lumbar puncture followed by anticoagulation. Stroke 1981;12:879-81.
19. Spanu G, Berlanda P, Rodriguez y Baena R. Spinal subdural haematoma: a rare complication of lumbar puncture. Case report and review of the literature. Neurochirurgia (Stuttg) 1988;31:157-9.
20. Vos PE, de Boer WA, Wurzer JA, van Gijn J. Subdural hematoma after lumbar puncture: two case reports and review of the literature. Clin Neurol Neurosurg 1991;93:127-32.
21. Chan KW, Datta NN. Iatrogenic acute subdural hematoma due to drainage catheter. Surg Neurol 2000;54:444-6.
22. Chung SJ, Kim JS, Lee MC. Syndrome of cerebral spinal fluid hypovolemia: clinical and imaging features and outcome. Neurology 2000;55:1321-7.
23. Kawakami Y, Ueki K, Chikama M, Shimamura Y, Naito T. Intracranial hemorrhage associated with nontraumatic disseminated intravascular coagulation: report of four cases. Neurol Med Chir (Tokyo) 1990;30:610.
24. Meixensberger J, Baunach S, Amschler J, Dings J, Roosen K. Influence of body position on tissue-pO2, cerebral perfusion pressure and intracranial pressure in patients with acute brain injury. Neurol Res 1997;19:249-53.
25. Moraine JJ, Berre J, Melot C. Is cerebral perfusion pressure a major determinant of cerebral blood flow during head elevation in comatose patients with severe intracranial lesions? J Neurosurg 2000;92:606-14.

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