Complications of Lumbar Puncture Followed by Anticoagulation

ROBERT L. RUFF, M.D., PH.D., AND JOHN H. DOUGHERTY, JR., M.D.

SUMMARY The complications associated with lumbar puncture (LP) were compared in 2 groups of 342 patients. The first group of patients was anticoagulated after the LP, and the second was not. The incidence of minor headache or back pain was similar in the 2 groups (Group 1–62%, Group 2–64%). The anticoagulated patients had a higher incidence of paraparesis (Group 1, 5 patients; Group 2, 0 patients; p < .05) and severe back or lumbosacral radicular pain lasting more than 48 hours (Group 1, 18 patients; Group 2, 6 patients; p < .025). Seven of the anticoagulated patients developed spinal hematomas (5 with paraparesis, 2 with severe back pain). Among the anticoagulated patients the risk of a major complication was increased by traumatic LP (p < .001), starting anticoagulation within one hour of the LP (p < .001), or aspirin treatment at the time of the LP (p < .001). This study suggests that if LP is done, delaying anticoagulation for at least one hour and avoiding concurrent aspirin therapy may decrease the risk of developing an extraparenchymal spinal hematoma.

SEVERAL REPORTS have suggested that lumbar puncture (LP) followed by anticoagulation will increase the risk of spinal hemorrhage. In this retrospective study we examined the risk that anticoagulation confers on LP, and identified factors which modified this risk.

Methods

Two groups of patients admitted to the New York Hospital-Cornell Medical Center between 1970 and 1980 were studied. Group 1 consisted of 342 patients with a clinical diagnosis of acute cerebral ischemia who were anticoagulated after LP. The second group consisted of 342 patients with cerebral ischemia (164), meningitis (126), or multiple sclerosis (52) who had an LP but were not anticoagulated. Patients with coagulopathy were excluded. Patients in groups 1 and 2 had a similar distribution of age, sex, heart disease, diabetes, and hypertension. Anticoagulation consisted of 10–14 days of intravenous heparin followed by conversion to warfarin. The evaluation period was the acute hospitalization which lasted from 15–46 days. Hypertension was defined as a diastolic blood pressure persistently elevated above 95 mm Hg, or a past history of hypertension with continuing anti-hypertensive therapy. Patients were considered diabetic if they had a repeated past history of hyperglycemia and glucose intolerance. Cardiac disease was defined by the presence of valvular heart disease, coronary artery disease or congestive heart failure. Medication usage was determined by chart review of the drugs that the patients were taking at the time of admission and the medications that were prescribed during their hospitalization.

All LPs were done with 20-gauge needles. A traumatic LP was differentiated from a true subarachnoid hemorrhage (SAH) by: a decrease in the red blood cell count in serially collected tubes; the absence of blood pigments, oxyhemoglobin, or bilirubin in the supernatant fluid; or the appearance of bloody fluid after repositioning the spinal needle when the initial fluid was clear. Probabilities were calculated using the Chi-square or two-tailed Fisher Exact test.

Results

Minor Complications. The 2 groups of patients had a similar incidence of minor complications (Group 1–62%, Group 2–64%). The minor complications were headache alone (Group 1–22%, Group 2–20%), backache alone (Group 1–28%, Group 2–23%), and both headache and backache (Group 1–12%, Group 2–13%). None of the minor complications was debilitating.

Major Complications. There was a 6.7% incidence of major complications in Group 1 compared to 1.8% in Group 2 (p < .001, table 1). The major complications were paraparesis (Group 1–5 patients, Group 2—none; p < .05) and severe back or lumbosacral radicular pain lasting more than 48 hours (Group 1–18 patients, Group 2–6 patients; p < .025). Four of the 5 patients with paraparesis had myelography and 3 proceeded to laminectomy. The findings at surgery were spinal subarachnoid hematoma in 2 and spinal epidural hematoma in the other. The operated patients who had surgery all had good recovery of function. The patient who did not have surgery had a spinal epidural hematoma demonstrated at myelography which was decompressed with a spinal needle. The patient who refused myelography remained paraparetic and died 3 months later. Autopsy demonstrated a chronic spinal subdural hematoma. None of the patients with severe back pain developed paraparesis or had myelography. Seven of these 18 patients from Group 1 subsequently died of unrelated causes and at autopsy had findings of a chronic spinal epidural hematoma in one, and an organized spinal subdural hematoma in another. Thus, 7/23 patients in Group 1 with major LP-related complications had
documented spinal hematomas which were epidural in
3, subarachnoid in 2, and subdural in 2.

In Group 1 the risk of a major complication (table 1) was significantly increased if the patient had a traumatic LP (76% vs 2%, p < .001), if anticoagulation was started within one hour of the LP (17% vs 1.5%, p < .001), or if the patient was treated with aspirin at the time of the LP (17% vs 2.4%, p < .001).

Heart disease, hypertension, diabetes, age, or sex did not affect the incidence of major complications in anticoagulated patients.

In Group 2, the only factor which increased the risk of a major complication was a traumatic LP (7.7% vs 0.7%, p < .001). More patients received aspirin in Group 2 (128) than in Group 1 (96), but aspirin therapy did not increase the incidence of LP-related major complications in patients who were not anticoagulated.

### Discussion

The incidence of LP-related minor complications of headache and/or backache for both groups was similar to that reported by Tourtellotte et al,^13^ Paraplegia occurred only in anticoagulated patients, and severe back pain was 3 times more frequent in the anticoagulated patients. Paraplegia was caused by extraparenchymal spinal hematomas. The autopsy finding of chronic subdural or epidural spinal hematomas in 2/7 patients with prolonged back pain suggests that several of the other patients with severe back pain may have had extraparenchymal spinal hematomas.

Anticoagulation and LP are known to increase the risk of developing a spinal hematoma. Table 2, compiled from reports in the literature, indicates that LP or anticoagulation were associated with extraparenchymal spinal hematoma as follows: subarachnoid-29%, subdural-44%, combined subarachnoid and subdural-88%, and epidural-59%.

In this study and in several previous studies, a traumatic LP increased the risk of developing an extraparenchymal spinal hematoma in patients with or without anticoagulation.  

Favaro et al reported that aspirin therapy might increase the risk of spinal hematoma in patients who are anticoagulated after LP. The increased risk of immediate anticoagulation after LP was not previously noted.

Although spinal hematomas usually produce local pain and rapidly progressive spinal cord dysfunction,^1^ there may be a more benign course with minimal pain or dysfunction.  

Spinal subarachnoid hematomas may also produce prominent meningeal signs which lead to the mis-diagnosis of SAH of intra-cranial origin.

Anticoagulation is accepted treatment for closely recurrent transient ischemic attacks or stroke-in-evolution. To be safe, however, such therapy requires accurate diagnosis of nonvascular and hemorrhagic lesions. Prior to the advent of computed tomography, LP alone was used to differentiate between hemorrhagic and ischemic lesions. Several authors have suggested that computed tomography is the only diagnostic procedure required to evaluate patients with cerebral ischemia prior to anticoagulation.  

However, computed tomography is not always feasible.

This study suggests that if LP is done, delaying anticoagulation for at least one hour, and avoiding concurrent aspirin therapy, may decrease the risk of developing an extraparenchymal spinal hematoma.

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