

Original Contributions

EMERGENCY DEPARTMENT CONTROL OF BLOOD PRESSURE IN INTRACEREBRAL HEMORRHAGE

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□ Abstract—Background: Early treatment of elevated blood pressure (BP) in patients presenting with spontaneous intracerebral hemorrhage (ICH) may decrease hematoma enlargement and lead to better neurologic outcome. **Study Objective:** To determine whether early BP control in patients with spontaneous ICH is both feasible and tolerated when initiated in the Emergency Department (ED). **Methods:** A single-center, prospective observational study in patients with spontaneous ICH was performed to evaluate a protocol to lower, and maintain for 24 h, the mean arterial pressure (MAP) to a range of 100–110 mm Hg within 120 min of arrival to the ED. An additional goal of placing a functional arterial line within 90 min was specified in our protocol. Hematoma volume, neurologic disability, adverse events, and in-hospital mortality were recorded. **Results:** A total of 22 patients were enrolled over a 1-year study period. The average time to achieve our target MAP after implementation of our protocol was 123 min (range 19–297 min). The average time to arterial line placement was 84 min (range 36–160 min). Overall, 77% of the patients tolerated the 24-h protocol. The in-hospital mortality rate in this group of patients was 41%. **Conclusions:** Adopting a protocol to reduce and maintain the MAP to a target of 100–110 mm Hg within 120 min of ED arrival was safe and well tolerated in patients presenting with spontaneous ICH. If future trials demonstrate a clinical benefit of early BP control in spontaneous ICH, EDs should implement similar protocols. © 2011 Published by Elsevier Inc.

□ Keywords—intracerebral hemorrhage; hypertension; therapy

INTRODUCTION

Spontaneous intracerebral hemorrhage (ICH) is seen in more than 67,000 patients per year in the United States and results in unacceptably high mortality and neurologic morbidity. The 30-day reported mortality rate of these patients is between 35% and 52%, with half of all deaths occurring within the first 2 days. The majority of surviving patients are left with severe disability, with only 20% of individuals returning to a level of functional independence at 6 months (1).

Contemporary theory regarding the pathogenesis of the initiation and propagation of ICH focuses on the role of intravascular fibrinoid necrosis creating “vulnerable” rupture sites at or near the bifurcation of susceptible intracranial arterioles. Chronic hypertension further reduces vascular compliance and increases the likelihood of spontaneous rupture (2). Persistently elevated blood pressure (BP) is thought to contribute to the propagation of ICH through early hematoma expansion and rebleeding. Early hematoma growth is seen in nearly one-third of all patients, most commonly in the first several hours from symptom onset, and is a poor prognostic indicator (3–9).

Despite increasing data suggesting that elevated BP in patients with ICH is associated with early hematoma growth, poor neurologic outcome, and death, there are no evidence-based guidelines supporting a specific treatment algorithm for managing the elevated BP in these patients. The 2007 American Stroke Association/American Heart Association guidelines on the management of ICH give a class IIb recommendation (i.e., usefulness/efficacy less well established by evidence or opinion) with a level of evidence C (i.e., consensus opinion of experts) to “consider a modest reduction of blood pressure” if the mean arterial pressure (MAP) is elevated > 130 mm Hg and to “consider aggressive reduction of blood pressure” if the MAP is > 150 mm Hg (1).

The goal of our study was to assess the feasibility and tolerability of early BP reduction in patients with elevated BP and spontaneous ICH in an Emergency Department (ED) setting. Secondary goals evaluated the following outcomes: change in hematoma volume at 24 h, in-hospital mortality, and functional neurologic outcome at 24 h and 3 months.

MATERIALS AND METHODS

Study Design

This was a prospective observational study conducted at a single hospital from January 15, 2007 to January 15, 2008 using a multidisciplinary protocol developed jointly by the Department of Emergency Medicine and the Divisions of Neurology, Neurosurgery, and Critical Care. The study was approved by the hospital institutional review board committee. Informed consent was waived, as this was a feasibility and safety study regarding an intervention that was routinely recommended by the Divisions of Neurology and Neurosurgery at our hospital.

Study Setting and Patients

Alameda County Medical Center, Highland Campus in Oakland, California, is an urban teaching hospital with an affiliated emergency medicine residency-training program that treats approximately 72,000 patients per year in the ED.

All adult patients with a spontaneous ICH, determined by non-contrast head computed tomography (CT) scan, who presented during the study period and who had a triage MAP > 120 mm Hg were eligible for inclusion. Exclusion criteria included age < 18 years, pregnancy, coagulopathy (bleeding disorder, low-molecular weight heparin or warfarin therapy, platelet count $< 50,000/$

mm³, or international normalized ratio > 2.0), pre-existing neurologic disability (e.g., requiring assistance for activities of daily living), symptom onset > 48 h before being seen in the ED, or a history (e.g., trauma) or CT scan suggesting secondary ICH (e.g., vascular malformation, aneurysm, tumor). Patients who had a head CT scan performed more than 90 min after ED arrival or who were transferred from another facility to our hospital were also excluded.

Study Protocol

Our management protocol specified that patients judged to be eligible by the treating emergency physician should have their MAP lowered to a target of 100–110 mm Hg within 120 min of presentation to the ED and maintain this target MAP at this level for a minimum of 24 h. We also specified a goal of placing a functional arterial line within 90 min of patient arrival. Antihypertensive treatment before the placement of the arterial line was recommended.

The choice of antihypertensive agent was left to the discretion of the treating emergency physician. Doses for the hospital-preferred hypertensive medications (esmolol, fenoldapam, labetalol, nicardipine) and the hospital protocols for use of recombinant factor VIIa and human coagulation factor IX complex were provided.

All patients were monitored in a critical care room in the ED and admitted to the intensive care unit for cardiovascular and neurologic monitoring. A repeat head CT scan was obtained at 24 h, or sooner in cases of neurologic deterioration. CT scans were performed on a 512×512 matrix with a 5-mm slice thickness.

Measurements

We collected data using standardized collection forms. The ED data were collected by the treating emergency physician or, if present, the consulting neurologist or neurosurgeon. The initial data collection included patient demographic information, past medical history, risk factors for ICH, time of symptom onset, Glasgow Coma Scale (GCS) score, and National Institute of Health Stroke Scale (NIHSS) score. Time to obtain target MAP, time to functional arterial line placement, antihypertensive agent(s) used, and 24-h complications were also recorded. The consulting neurologist or neurosurgeon collected the inpatient data. This included antihypertensive agent(s) used or discontinued, GCS and NIHSS scores at 24 h and discharge, and in-hospital unexpected events. After the enrollment period, a board-certified neurologist, adept in the ability to measure intracranial

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