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Prognostic Significance of Hyponatremia in Acute Intracerebral Hemorrhage: Pooled Analysis of the Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial Studies.

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Abstract

OBJECTIVES:
To determine the association of hyponatremia at presentation with clinical and imaging outcomes in patients with acute intracerebral hemorrhage.

DESIGN:
Retrospective pooled analysis of prospectively collected data from 3,243 participants of the pilot and main phases of the Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trials 1 and 2 (international, multicenter, open, blinded endpoint, randomized controlled trials designed to assess the effects of early intensive blood pressure lowering in patients with acute intracerebral hemorrhage).

SETTING:
Clinical hospital sites in 21 countries.

PATIENTS:
Patients with predominantly mild-moderate severity of spontaneous intracerebral hemorrhage within 6 hours of onset and elevated systolic blood pressure (150-220 mm Hg) were included in the study.

INTERVENTIONS:
Patients were assigned to receive intensive (target systolic blood pressure, < 140 mm Hg within 1 hr) or guideline-recommended (target systolic blood pressure, < 180 mm Hg) blood pressure-lowering therapy.

MEASUREMENTS AND MAIN RESULTS:
Presentation hyponatremia was defined as serum sodium less than 135 mEq/L. The primary outcome was death at 90 days. Multivariable logistic regression was used to assess the association of hyponatremia with important clinical events. Of 3,002 patients with available data, 349 (12%) had hyponatremia. Hyponatremia was associated with death (18% vs 11%; multivariable-adjusted odds ratio, 1.81; 95% CI, 1.28-2.57; $p < 0.001$) and larger baseline intracerebral hemorrhage volume (multivariable adjusted, $p = 0.046$) but not with baseline perihematomal edema volume nor with growth of intracerebral hemorrhage or perihematomal edema during the initial 24 hours.

CONCLUSIONS:
Hyponatremia at presentation is associated with increased mortality in patients with predominantly deep and modest volume intracerebral hemorrhage through mechanisms that seem independent of growth in intracerebral hemorrhage or perihematomal edema.