Early Intracerebral Hematoma Expansion After Aneurysmal Rupture

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**Background and Purpose**—Intracerebral hematomas (ICHs) often increase in size in the initial hours. It is unknown whether expansion of ICHs after aneurysmal rupture in the acute phase is always a sign of rerupture of the original aneurysm.

**Methods**—We included patients with an ICH from a ruptured aneurysm who underwent computed tomography imaging within 24 hours of symptom onset and a repeat computed tomography within 48 hours. Hematoma growth was considered present when there was a 33% increase in hematoma volume, as assessed by the ABC/2 method. Clinical and radiologic characteristics were compared between patients with ICH growth, with and without clinical signs of rerupture. Rerupture was defined as a sudden deterioration in the level of consciousness in the absence of ventricular enlargement or a systemic cause.

**Results**—Hematoma expansion within 48 hours after onset occurred in 12 of the 49 included patients and was preceded by clinical evidence of rerupture in 6 of these 12 patients. Of the 6 patients without an evident rerupture, 3 had no clinical deterioration, 1 had respiratory failure due to pneumonia, another had temporal brain herniation, and the last had acute hydrocephalus.

**Conclusion**—Only half of the patients with early ICH expansion after aneurysmal rupture had clinical signs of rerupture of the aneurysm. Early ICH expansion after aneurysmal rupture can be caused by other mechanisms, which are possibly comparable to those responsible for hematoma expansion in spontaneous ICH. (Stroke. 2010;41:2592-2595.)

**Key Words:** subarachnoid hemorrhage ■ intracerebral hemorrhage ■ intracranial aneurysm

In 20% of patients with aneurysmal subarachnoid hemorrhage (aSAH), the hemorrhage extends into the brain parenchyma. The presence of an intracerebral hematoma (ICH) in patients with aSAH is associated with a high case fatality and poor functional outcome. Several studies have shown significant ICH expansion within 24 hours after onset of symptoms in approximately one third of patients with spontaneous ICH. Contrast extravasation has been observed up to 48 hours after symptom onset of spontaneous ICH, which suggests active bleeding.

In patients with ICH caused by a ruptured aneurysm, rerupture of the aneurysm is an obvious cause of expansion, but whether ICH expansion can also occur in the absence of a rerupture is unknown. We studied how often hematoma expansion in patients with ICH from a ruptured aneurysm is caused by rerupture of the aneurysm.

**Methods**

**Patients**

We included patients with ICH with or without SAH caused by a ruptured aneurysm from our prospectively collected database of patients with SAH admitted to the University Medical Center, Utrecht, The Netherlands, between January 2003 and November 2008. ICH had to be confirmed by noncontrast computed tomography (CT) within 24 hours after onset of symptoms. Patients had to have had a second CT within 48 hours after symptoms onset. The presence of an aneurysm had to be confirmed by CT angiography (CTA) or digital subtraction angiography. Exclusion criteria were age <18 years and craniotomy before the second CT. On the basis of the study protocol, the medical ethics committee of the University Medical Center Utrecht decided that no formal approval was needed for this study, because we only retrospectively reviewed data collected for clinical purposes, and we performed no additional tests or interviews of the patients.

We retrieved the following data for each patient: age, sex, daily use of antiplatelet agents, use of oral anticoagulants at the time of aneurysmal rupture (and if so, international normalized ratio at presentation), mean arterial pressure at presentation, clinical deterioration preceding the second CT scan, presumed cause of clinical deterioration, time interval between the first and second CT scans, location and size of the aneurysm, ICH location, and the presence of intraventricular or subdural blood. We defined rerupture as the cause of ICH expansion when a patient had a sudden deterioration in the level of consciousness in the absence of ventricular enlargement or a systemic cause.

**Measurement of ICH Volume**

ICH volume was calculated with the ABC/2 method. Perihematoma edema was not included in the measurements. Hematoma expansion was defined as an increase of ICH volume >33%, because changes in hematoma volume of <33% may reflect variability in CT imaging rather than actual hematoma expansion.

**Data Analysis**

For the “expansion group” versus the “no-expansion group” and the “rerupture group” versus the “no-rerupture group,” descriptive sta-
tistics were used for patient and ICH characteristics. We calculated overall proportions, mean values with standard deviation (SD) for normally distributed data, and median values with interquartile range for data with a skewed distribution. We refrained from a formal statistical comparison of patient and ICH characteristics between patients with ICH growth with and without rerupture because the numbers of patients were small.

Results

Of the 755 patients with aSAH admitted within the study period, 162 patients (21%) had an ICH, of whom 49 met our inclusion criteria (Figure 1). Patient and ICH characteristics are listed in the Table. In 12 patients (24%), the repeat CT showed ICH expansion within 48 hours of symptom onset. Median ICH volume increased from 13 mL (interquartile range, 7 to 24) on the admission CT to 26 mL (interquartile range, 12 to 65) on the second CT. In 3 patients with ICH expansion, no obvious clinical deterioration was present.

Six of the 12 patients with ICH expansion fulfilled our criteria for rerupture, but none of these 6 patients had a sudden aggravation of headache before the second CT was made. In 1 of the 6 patients with rerupture, CTA showed contrast extravasation from a middle cerebral artery aneurysm (Figure 2). In the 6 patients without signs of rerupture, 1 deteriorated due to respiratory failure from pneumonia, a second from temporal brain herniation, and a third from acute hydrocephalus. One of the 3 patients without obvious clinical deterioration was stable but comatose, with a Glasgow Coma Scale score of E1M5Vtube.6 The second patient was taking anticoagulants and was treated with a prothrombin complex concentrate (International Normalized Ratio = 1.11 on admission to our hospital; Figure 3). The third patient was a 41-year-old woman who presented with a Glasgow Coma Scale score of E3M6V5 and subtle sensory dysphasia at a regional hospital. Her clinical condition had remained unchanged at the time of the CT and CTA performed at our institution. The CT showed expansion of a left frontal hematoma (Figure 4) and a left middle cerebral artery aneurysm, which was coiled successfully. In both patients, no active extravasation of contrast from the aneurysm was observed.
Discussion

In only half of the patients with ICH expansion early after aneurysmal rupture, the expansion was explained by rerupture of the aneurysm; in patients with expansion but without rerupture, no clinical deterioration or obvious reasons other than rerupture were present.

Little is known about ICH expansion in patients with aSAH. In 1 study of ICH in various vascular lesions including aneurysms, the proportion of patients with hematoma expansion within a median time of 7 hours after presentation was 20%. However, in that study, the proportion of patients with a ruptured aneurysm who had ICH expansion and the proportion of patients with a rerupture were not reported. Another study did not find hematoma enlargement or contrast extravasation in 4 patients with ICH of a ruptured aneurysm who underwent a repeat CT and CTA within 24 hours after onset.

In patients with spontaneous ICH, hematoma expansion has been reported in approximately one third of patients within 24 hours after onset. The cause of hematoma expansion in these patients remains unclear. Ongoing bleeding from a single source, breakdown of the blood-brain barrier, and delayed bleeding from a second source have all been suggested to be involved in contrast leakage and subsequent hematoma expansion. Contrast extravasation has been observed up to 48 hours after symptom onset of a spontaneous ICH and has been shown to be an independent predictor of hematoma expansion and poor outcome. The location of contrast leakage within the hematoma can be solely central, peripheral, or mixed.

In patients with ICH after aneurysm rupture, rerupture of the aneurysm is an obvious cause of hematoma expansion. In this study, we found hematoma expansion in the absence of sudden clinical deterioration, suggesting that other mechanisms are responsible for hematoma expansion. Ongoing bleeding or bleeding from damaged vessels surrounding the hematoma, as suggested in spontaneous ICH, may cause hematoma expansion in aneurysmal bleeding in the absence of sudden clinical deterioration. Whether contrast extravasation is an independent predictor of hematoma expansion and poor outcome in aneurysmal ICH, similar to observations in spontaneous ICH, has not been studied.

Figure 2. A 53-year-old man was admitted with SAH and a right temporal ICH adjacent to the sylvian fissure (A and C; the first CT was performed at a regional hospital). Initially, his clinical condition was stable, with a Glasgow Coma Scale score of E3M6V3 and a left-sided paralysis. A few hours later, the patient had to be intubated because of sudden neurologic deterioration accompanied by progressive respiratory failure; he was then transferred to our hospital. Subsequent CTA showed active extravasation (E and F, second CT; the arrow indicates contrast leakage into the hematoma) from a middle cerebral artery aneurysm into the expanding ICH (B and D, second CT). His neurologic condition worsened to a Glasgow Coma Scale score of E1M1Vtube with bilateral, fixed, dilated pupils and brainstem dysfunction, and he died soon after admission.

Figure 3. A 48-year-old woman presented to a local hospital with sudden headache, mild right-sided hemiparesis, and dysphasia. She was taking oral anticoagulants because of a history of deep vein thrombosis associated with a factor V Leiden mutation. Noncontrast CT showed an SAH and a left frontotemporal ICH adjacent to the sylvian fissure (left). After normalization of her international normalized ratio by administration of a prothrombin complex concentrate, the patient was transferred to our hospital. On admission, her clinical condition was unchanged. A repeat CT scan performed 7 hours after the first CT showed marked ICH expansion with a mass effect (right), and CTA detected a left middle cerebral artery aneurysm without active extravasation. The hematoma was evacuated and the aneurysm was clipped. A year later, the patient’s right arm was still paretic, but she had regained independence in daily activities.
in CT imaging technique. The ABC/2 method that we used to assess ICH volume has an excellent interrater and intrarater reliability.5

In conclusion, a substantial proportion of patients with ICH due to a ruptured aneurysm show an expansion of ICH volume within 48 hours, without evidence of rerupture of the original aneurysm. Further studies are needed to more precisely determine the proportion of patients with aneurysmal ICH who develop ICH expansion and to define the effect of ICH expansion on clinical outcome. If patients at risk of hematoma expansion can be identified by, for example, the “spot sign,” further studies should determine whether the outcome of patients with a high risk of ICH expansion may be improved by immediately occluding the aneurysm in combination with evacuation of the hematoma or by other measures to prevent hematoma expansion.

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Disclosures
None.

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