Blood pressure in acute intra-cerebral hemorrhage

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Cerebrovascular disease represents a leading cause of mortality and morbidity worldwide, and the spontaneous non-traumatic intra-cerebral hemorrhage (ICH) is the stroke subtype characterized by the poorer prognosis. It affects more than 1 million people every year and to date no specific medical or surgical treatments are available to improve patient outcome.

The raise in blood pressure (BP) levels is very common after ICH and it results from the variable combination of multiple mechanisms including premorbid hypertension, increase intracranial pressure, activation of neuro-vegetative signaling and neuro-endocrine pathways. Elevated BP is a reliable outcome predictor and it has been strongly associated with early neurological deterioration, death and disability (1). Notwithstanding, the optimal strategy to lower BP during the hyper-acute stage of ICH is still debated and whether a more aggressive treatment could be better is still controversial (2). Two main conflicting perspectives face in this clinical dilemma. At one side, the acute hypertensive response could be protective, preserve the cerebral blood flow in the setting of raised intracranial pressure and prevent the development of ischemic injury. On the other, high BP levels increase the risk of edema formation and promote hematoma enlargement by enhancing on-going bleeding and re-bleeding.

The first issue that should be addressed is therefore represented by the safety of the anti-hypertensive treatment. With this respect, the evidence is reassuring: randomized clinical trials have clearly proved how the intensive BP-lowering treatment to a systolic target of 140 mmHg within the first hours after the ICH onset did not have negative effects on the neurological status and was not associated to serious adverse events (3-6). Recently, in the Antihypertensive Treatment of Acute Cerebral Hemorrhage-2 (ATACH-2) trial, a similar safety was observed even when the BP target was lowered to 110 mmHg (7). Accordingly, advanced neuroimaging did not found any reduction in peri-hematomal and hemispheric perfusion after BP reduction, and raised suspicions about the real existence of an ischemic peri-hemorrhagic penumbra area (6).

The second question is the efficacy of lowering BP in improving the patient prognosis. One of the main mechanism by which BP treatment is thought to influence the clinical outcome is the reduction of the hematoma growth. In large randomized trials comparing intensive and conservative BP-lowering strategies, the aggressive BP management resulted in significant attenuation of the hematoma enlargement and reduction of the risk of substantial hematoma growth within 24 hours after randomization. Unfortunately, any clear effect on the primary end-point of 3-month death or disability emerged. Nonetheless, secondary analysis has demonstrated improved functional recovery and better physical and mental well-being in patients aggressively treated (5). To date no definitive conclusions can be drawn about the real efficacy of the more intensive BP reduction, but a tendency toward a better prognosis has been observed. It is noteworthy that most patients enrolled in randomized clinical trials presented mild to moderate hematoma volume, were generally treated beyond the first three hours after the ICH onset and often failed to achieve the target BP level within one hour after randomization. All these factors may have confounded the final results and diluted the real effectiveness of the trial intervention.

Additionally, there is accruing evidence that not only

the absolute BP levels but even their variation over the time may affect the stroke outcome (8,9). Since fluctuations in BP levels may be influenced by both the intensity of treatment and the type of antihypertensive drug, and the benefit of early BP-lowering therapy is enhanced by ensuring stability in BP levels, further analysis on the BP variability are warranted.

In conclusion, as recommended by the recently revised American Heart and Stroke Association guidelines for the management of spontaneous ICH, the early intensive BP-lowering in patients with acute ICH and raised BP between 150 and 220 mmHg is safe and can be effective to improve clinical outcome (10). The assessment of the real effect size of the efficacy of the intensive treatment and the identification of the variables that may predict a more favourable response and a greater clinical benefit deserve further investigations and represent future challenges in the scenario of acute ICH treatment.

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Footnote

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