Induced hypothermia and neuroprotection effects for acute ischaemic stroke patients

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Stroke is responsible for 9% of deaths worldwide that makes it the second most common cause of mortality. More than 25% of stroke survivors become permanently disabled and lose independence in performing day-to-day activities (1). Approximately two million brain cells die every minute during a stroke which increases the risk of brain damage, disability, and death. The level of disability varies from patient to patient according to the type of stroke suffered, the part of the brain affected, and the size of the damaged area (2).

According to the American Stroke Association (ASA, 2014), strokes can be classified into two main categories: ischemic (87%) and hemorrhagic (13%). Ischemic strokes occur when the arteries connected to brain become narrowed or blocked, causing severely reduced blood flow (ischemia). Ischemic strokes can be further divided into thrombotic and embolic stroke. Hemorrhagic strokes occur when a blood vessel that supplies the brain ruptures and bleeds. When an artery bleeds, brain cells do not receive oxygen and nutrients. The most common type of stroke is ischemic type. That type may cause massive cerebral hemisphere infarction (MCHI). This event may result in lethal intracranial hypertension due to massive cerebral edema leading to herniation. Mortality rate in that event reaches 78% even after efficient medical management.

Therefore, decompressive craniotomy (DC) was suggested by neurosurgeons to reduce the increased intracranial pressure but the risk is still very high. As well, patients who traditionally take antplatelet aggregation and anticoagulants therapy are still at increased risk of developing cerebral hemorrhage (3).

Hypothermia has demonstrated neuroprotective actions in cerebral ischemia and should be induced as early as possible to induce maximum neuroprotection and edema releasing effect (4). Moreover, previous experimental studies demonstrated that hypothermia protects the brain from various insults such as stroke and brain trauma particularly when cooling is started within a few hours of onset due to its salutary actions such as decrease of cerebral metabolic activity, decrease release of glutamate, and increases flow of oxygen (5,6) and decreases cerebral edema by reducing permeability to inflammatory substances such as cytokines (7).

Despite evidence based and published guidelines that support the utilization of therapeutic hypothermia, implementation of hypothermia therapy has not been utilized. The common known barriers to implementation include lack of awareness of techniques of hypothermia and debates regarding the efficient method to reach aimed temperatures (8).

Accordingly, nurses will have evidence based practice regarding therapeutic hypothermia to be incorporated in the management of stroke patients during the different stages of implementation of therapeutic hypothermia that includes initiation, maintenance, rewarming, and normothermia (1). Therefore, specifying trained nurse practitioners who are well-versed in established guidelines can help integrate the multidisciplinary team that is needed to successfully implement therapeutic hypothermia. Thus, the current study is conducted to establish data base information regarding that issue to be incorporated into the future of care of acute ischemic stroke patients and might pay an attention for further researches in this area.

REFERENCES