Intracranial pressure monitoring for malignant stroke: It is too soon to call it off

Malignant cerebral infarction (MCI) is a large middle cerebral artery infarction with or without the anterior and posterior cerebral artery regions involved. MCI is associated with acute cerebral edema resulting in a space-occupying mass effect or brain herniation. The brain edema typically occurs within 48 h of the onset, and it may lead to significant disabilities or death in MCI. Because of the extent of infarction in MCI, intracranial pressure (ICP) is generally expected to rise. Treatment options for MCI are, thus, designed to lower ICP.

Several treatment options are available to manage increased ICP. The first-line therapy includes hyperosmolar therapy (e.g., hypertonic saline and mannitol). Mannitol is an osmotic diuretic that reduces brain volume by removing water from the brain parenchyma to the circulation. The free water is eventually excreted by the kidneys, leading to dehydration. Hypertonic saline lowers ICP by inducing hypernatremia. Hypertonic saline is often favored, given its minimal osmotic effect. Barbiturates decrease ICP by reducing the cerebral metabolic rate; however, they are associated with hypotension and unreliable neurological examination from the sedative effect. Hyperventilation removes carbon dioxide, a potent cerebral vasodilator. Because of occasional rebound vasodilation and subsequently increased infarction volume, hyperventilation is used as a temporizing measure.[1]

Although ICP is expected to rise in MCI, ICP monitoring has not been a standard of care. This is because clinical findings have not correlated clearly with ICP values. One study measured the initial ICP and subsequent values during the first 12 h of 19 patients who became stuporous from large hemispheric infarctions.[2] Four patients (21.1%) had elevated initial ICP and six (31.6%) showed increased ICP during the first 12 h. Eight patients died and five of these patients showed increased ICP in the first 12 h and the remaining three did not. In another study, intraparenchymal ICP sensors were placed in the ischemic hemisphere of 19 patients with MCI.[3] Twelve patients (63%) showed radiographic findings of uncal herniation or cistern effacement despite normal ICP. Four showed anisocoria and two of these patients had normal ICP. A prospective study of 48 patients showed similar results.[4] Clinical findings of herniation always precede increase in ICP. Radiographic findings did not correspond to ICP values.

These three studies concomitantly note two important factors: (1) ICP monitoring cannot supplant physical examination and radiographic findings and (2) significantly high ICP portends a poor prognosis. A few speculations have been discussed to explain the normal ICP in the setting of clinical brain herniation. Poca *et al*.[3] explain that the cerebral blood flow reduces in the ischemic hemisphere in the beginning though the reduced blood volume later gets overcompensated by increased extracellular water content. There may be a compensatory mechanism for changes in brain volume until the brain shift causes herniation-associated vascular/nerve compressions or brainstem distortion. Nonetheless, can these studies justify excluding ICP monitoring in MCI management?

Weaver *et al*.[5] presented four patients with a unilateral intracranial mass lesion. These patients showed ICP significantly different between the ipsilateral and contralateral hemispheres. Two of the four patients showed a midbrain-level examination without globally increased ICP. D'Ambrosio *et al*.[6] induced left hemispheric reperfused stroke in seven adult male baboons and measured ICP in both hemispheres. Interhemispheric ICP gradients were observed when the infarct volume became >20%. Wolfla *et al*.[7] demonstrated regional brain tissue pressure changes during an expansion of a right extradural temporal mass. Intraparenchymal ICP monitors were placed in the bilateral frontal and temporal lobes, midbrain, and cerebellum. While the most significant pressure changes were observed in the ipsilateral temporal lobe, the pressure change in the contralateral frontal lobe preceded that of the ipsilateral frontal lobe.

It still seems premature to conclude that ICP monitoring does not have a significant clinical value in managing MCI. The aforementioned studies[2-4] were largely based on unilateral ICP values. (In fact, one of these studies[5] measured bilateral ICP and noted differentials during the first 3 days of monitoring.) Pascal's law notes that a pressure change in an enclosed incompressible fluid gets equally transmitted throughout the fluid and to the walls of the container. However, the brain tissues are different in nature and too valuable to be treated as an "incompressible fluid." It appears to be presumptuous to assume that ICP is equally distributed in the brain with MCI. A prudent way to exclude the usage of

ICP monitoring in MCI is to include bilateral ICP measurements and differentials associated with clinical findings.

Future studies may clarify any significance of interhemispheric ICP differentials in MCI. In order to ideally study ICP differentials, locations for ICP monitor insertion should be varied and clinical findings must accompany to find any correlation. Until then, it is too soon to call off ICP monitoring in managing MCI.

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