

Intrathecal nicardipine

- Intrathecal nicardipine for cerebral vasospasm after non-traumatic subarachnoid hemorrhage: a meta-analysis
- Earlier onset of cerebral vasospasm in ruptured infectious intracranial aneurysms
- The Clinical Research Landscape of Intracranial Nicardipine for Aneurysmal Subarachnoid Hemorrhage: Insights From Bibliometric Analysis
- Effects of intrathecal administration of sodium nitroprusside and nicardipine on cerebral pial microcirculation, cortical tissue oxygenation, and electrocortical activity in the early post-resuscitation period in a porcine cardiac arrest model
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- Intrathecal Nicardipine as Treatment for Severe Cerebral Vasospasm After Aneurysmal Subarachnoid Hemorrhage: A Retrospective Clinical Study
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Data suggest that the [intrathecal](#) (IT) delivery of the [calcium channel blocker](#) (CCB) [nicardipine](#) may have a [role](#) in a reactive (rather than a preventative) approach to address [cerebral vasospasm treatment](#) and avoid [delayed cerebral ischemia](#) (DCI) ¹⁾. This approach has the advantage of avoiding deleterious systemic effects, i.e., decreased blood pressure, that is common with oral and intravenous CCBs ²⁾. An intermittent and titratable IT nicardipine regimen was shown to be associated with proximal vessel vasodilation, reduced risk for DCI, and improved long-term functional outcomes ^{3) 4)}. However, the macrovascular vasodilation itself, as quantified by a reduction in daily TCD blood flow velocity, was not associated with outcomes ⁵⁾.

Sathalingam et al. employed a non-invasive optical modality called diffuse correlation spectroscopy (DCS) to quantify the acute microvascular cerebral blood flow (CBF) response to [intrathecal nicardipine](#) (up to 90 min) in 20 patients with medium-high grade non-traumatic SAH. On average, CBF increased significantly with time post-administration. However, the CBF response was heterogeneous across subjects. A latent class mixture model was able to classify 19 out of 20 patients into two distinct classes of CBF response: patients in Class 1 ($n = 6$) showed no significant change in CBF, while patients in Class 2 ($n = 13$) showed a pronounced increase in CBF in response to nicardipine. The incidence of DCI was 5 out of 6 in Class 1 and 1 out of 13 in Class 2 ($p < 0.001$). These results suggest that the acute (<90 min) DCS-measured CBF response to IT nicardipine is associated with intermediate-term (up to 3 weeks) development of DCI ⁶⁾.

Case series

In this observational study, we prospectively employed a non-invasive optical modality called diffuse correlation spectroscopy (DCS) to quantify the acute microvascular cerebral blood flow (CBF) response to IT nicardipine (up to 90 min) in 20 patients with medium-high grade non-traumatic SAH. On average, CBF increased significantly with time post-administration. However, the CBF response was heterogeneous across subjects. A latent class mixture model was able to classify 19 out of 20 patients into two distinct classes of CBF response: patients in Class 1 ($n = 6$) showed no significant

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