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Hydration and Collateral Flow in Acute Stroke

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Hydration and volume status are intuitively linked with hemodynamics in acute ischemic stroke, yet numerous questions abound regarding such rudimentary and commonly encountered aspects of stroke care. Chang et al. are commended for underscoring the potential association of dehydration with indirect markers of collateral circulation in acute stroke.[1] They measured an array of clinical and laboratory variables, including the serum blood urea nitrogen/creatinine (BUN/Cr) ratio and analyzed the relationship with two previously described MRI markers of collateral circulation. Ipsilateral posterior cerebral artery laterality (iPCA) and FLAIR vascular hyperintensities (FVH) in the context of proximal or M1 MCA occlusion were linked with the presence of dehydration (defined as BUN/Cr ≥ 15). The vast majority (70%) of these acute stroke patients with proximal MCA occlusion excluded from thrombolysis were dehydrated. The lack of iPCA and less extensive FVH in dehydrated patients may provide indirect clues to the underlying collateral status yet a plethora of questions arise regarding the findings, discourse and implications suggested by the conclusions. The authors' inculcation regarding a purported causal *effect* of dehydration on the *development* of collaterals and resultant conclusions advocating intravenous fluid administration deserve scrutiny. Such profound conclusions goad the reader to carefully dissect rather than rapidly swig these curtly drawn summary statements about fluids and volume status. Is this saga plumb wrong?

The methodology and specific definitions used to plumb, or literally assess the volume status of water and depth of its impact, may be erroneous or “plumb wrong”. BUN/Cr values may be influenced by many factors and handled as either a continuous variable or utilize different dichotomous thresholds to define dehydration. Other hemodynamic variables, such as blood pressure, head positioning, fluid administration and cerebral blood volume (CBV) were relatively neglected.[2] Furthermore, the analyses reveal that dehydration is associated with lack of iPCA and less extensive FVH slow flow in collateral vessels downstream of M1 occlusion, yet NIHSS severity is worse with more extensive FVH. This novel quantification of FVH and implications remain unclear, as dehydrated patients appear to have lower NIHSS that defies all prior correlative studies that show higher NIHSS with worse

collaterals. Conjecture about CBV and actual collateral grade were also not substantiated by either perfusion imaging or angiography.[3]

Alternatively, are stroke patients with poor collaterals simply “plumb wrong” with feeble collateral circuits that are exquisitely frail and thereby dramatically altered by volume status or therapeutic fluid administration as the authors conclude? MRI was acquired within 3 days of M1 occlusion, without serial imaging or control for the timing of image acquisition. The temporal evolution and “development” of collaterals therefore remains unknown. The etiology of MCA occlusion, including underlying intracranial atherosclerosis and transient depletion of CBV, may have culminated in acute stroke. Selection bias is suspect in this cohort of patients with M1 occlusions ineligible for thrombolysis, with relatively milder NIHSS and ability to undergo MRI without evidence of completed MCA infarction up to 3 days from onset. These factors argue for relatively robust collaterals, despite the conclusion that 70% had poor collaterals due to dehydration. Is collateral sustenance (lack of completed MCA infarction due to M1 occlusion) up to 3 days from symptom onset truly a failure of collaterals?[4] Perhaps such patients may have benefited from endovascular therapies or alternative strategies to improve perfusion. Why were these patients not treated with thrombolysis at an earlier timepoint and how were confounding hemodynamic interventions such as head positioning, blood pressure manipulation and fluid administration controlled? A causal relationship of dehydration with indirect collateral markers and the development or temporal evolution of collaterals without knowledge of baseline collateral status cannot be ascertained.

The implications of this article are potentially extensive, arguing that hydration can promptly modify collateral grade. This refutes the concept that individual variability in the degree of collateral circulation exists amongst stroke patients or across animal strains where genetic variability has been established. If the authors are correct in this regard, then the strikingly potent association of collateral circulation in almost every multicenter endovascular stroke trial to date with angiographic proof of collateral grade may alternatively be purely due to differences in hydration or volume status. If collateral flow is merely a function of hydration, then the study of hemodynamics in stroke is indeed, paramount to all other investigations.

This intriguing article focuses on one of the most influential and generalizable topics in acute stroke. Wrong plumbing, or the differences in collateral grade and hemodynamics across individuals, emerges as one of the key questions in acute stroke. The effect of routine hemodynamic interventions, from prehospital care to emergency triage, bridging therapies for potential reperfusion and subacute ICU management, constitute the greatest unanswered quandaries in ischemic stroke.

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