Engorged Medullary Veins in Neurosarcoidosis: A Reflection of Underlying Phlebitis?

G. Bathla, N. Soni, T. Moritani and A.A. Capizzano

AJNR Am J Neuroradiol 2019, 40 (3) E14-E15
doi: https://doi.org/10.3174/ajnr.A5951
http://www.ajnr.org/content/40/3/E14

This information is current as of September 16, 2023.
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e read with interest the recent article describing the engorge
gen of deep medullary veins (DMV) in patients
with neurosarcoidosis (NS).1 The findings of DMV engorge
ment on susceptibility-weighted imaging have also intrigued
us, and we recently published our experience in such cases.2
We agree with some of the findings reported in this article,
such as occurrence of perivascular enhancement (PVE) in
about half of these patients, increased occurrence of microhe
morriages, and a tendency toward worse neurologic out
comes. Even though the latter was not statistically significant,
we suspect that it may, at least partially, be related to the small sample size.

Even though the authors mention that the DMV engorgement
is not secondary to downstream venous occlusion, they agree that
the pathophysiology of these findings remains unclear. This issue
is further compounded by the absence of any correlation with
neral tissue biopsy, conventional angiography studies, or any
changes in DMV with time, especially with regard to immunosup
pressive therapy, which limits any educated extrapolation or in
ference of the presented data.

We suspect that the engorged DMVs, as seen on SWI, reflect
underlying venous phlebitis. This is based on the previously re
ported postmortem literature, which showed that the venous in
volvement was most common in the paraventricular region, and
our own experience, in which we evaluated 4 patients with en
gorged DMVs.2,3 In all of our patients, we also had conventional
angiography data as well as neural tissue biopsy. The venous phase
of the angiographic studies confirmed the presence of tortuous and
engorged veins, and the brain biopsy also found a predomi
nant venous involvement.

Even though venous involvement in NS is fairly well-recog
nized in the postmortem literature, the under-recognition on
imaging is likely due to lack of sequences such as SWI, which
are more sensitive to tissue susceptibility. The introduction of
SWI as a routine clinical sequence has more recently led to
greater recognition of this imaging finding. In some ways, we
wonder if SWI has provided the missing link that connects the
postmortem literature and in vivo findings. Another reason for
our suspicion is based on our anecdotal experience with pa
tients with NS, in which mild cases of engorged DMVs do
improve when patients receive immunosuppressive therapy
and also appear worse when patients present with NS flare. In
patients in whom they have been present for a while, such
fluctuations, however, tend to be less frequent, possibly sec
ondary to irreversible injury. The presence of PVE in a similar
distribution also supports the possibility that there is a super
imposed component of perivascular inflammation. We agree
with the authors that this may be a combination of engorged
vessels and perivascular involvement. In fact, we recently per
formed vessel wall imaging in 1 such case and noted that the
cortical veins did show circumferential enhancement, corre
sponding to the SWI findings, a likely reflection of underlying
vascular inflammation.4

An interesting question here, if SWI does indeed reflect
venous involvement, is whether SWI findings can be used as a
surrogate biomarker for ongoing vascular inflammation in NS
cases. Even though this is open to further research, on the basis
of our experience with limited patients, we suspect that vessel
wall imaging and SWI are reflecting different components of
venous involvement. One, therefore, may not be substituted
for the other, especially because the SWI findings may become
irreversible in chronic cases. Nevertheless, the possibility that
SWI findings likely reflect underlying NS-associated phlebitis
does add a new dimension to the NS imaging spectrum. Even
though a lack of any neural tissue banks in NS would preclude
retrospective evaluation of imaging and postmortem data and the
overall uncommon nature of NS would limit a prospective single
center study, the significance of SWI findings may be better clar
ified through multicenter pooling of cases in which neural biopsy
and conventional angiography data are available.

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