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Subdural Hygroma versus Atrophy on MR Brain Scans: “The Cortical Vein Sign”

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PURPOSE: To determine if the position of the superficial cerebral cortical veins can be used to distinguish subdural hygroma from atrophy on MR brain scans. METHODS: Retrospective review of MR scans obtained in cases of extracerebral fluid collections, separating these into two groups, i.e., subdural hygroma or atrophy. FINDINGS: All cases of atrophy in this study showed cortical veins and their branches traversing widened cerebrospinal fluid spaces over the cerebral convexities. None of the subdural hygroma patients showed this finding. Cortical veins in hygroma patients were seen only at the margin of the displaced cortex, and did not traverse the fluid collections over the cerebral convexities. CONCLUSIONS: The authors call the visualization of cortical veins and their branches within fluid collections at the cerebral convexities “the cortical vein sign.” They believe this sign to be prima facie evidence of atrophy; its presence rules out the diagnosis of subdural hygroma in the region of interest.

Index terms: Cerebral hematoma, magnetic resonance; Cerebral atrophy, magnetic resonance; Veins, cerebral

Distinguishing between subdural hematoma and subdural hygroma by magnetic resonance (MR) imaging is usually not a problem since subdural hematomas usually contain blood products that alter the signal characteristics of the extracerebral fluid collection sufficiently that it may be recognized as different from cerebrospinal fluid (CSF) (1–4). This is true even in subdural hematomas of various ages that may be isodense to brain or CSF by computed tomography (CT) (2). However, when an extracerebral fluid collection is isodense to CSF on CT, various criteria have been utilized for predicting whether it represents the results of widening of the subarachnoid space due to atrophy versus a subdural fluid collection. These criteria usually are based on “mass effect” on the brain such as flattening of the cortical surface with obliteration of sulci with subdural hygroma, as opposed to abnormal prominence of the cortical sulci with atrophy.

These observations may be very subjective and inconclusive in many cases. The same difficulty arises on MR images when the extracerebral fluid collection is isointense to spinal fluid on all pulse sequences. This could occur in true “hygromas” that are the result of arachnoid tear or insufficiency allowing spinal fluid to enter the subdural space, or, theoretically, in subdural hematomas of sufficient age that blood products or their proteinaceous residual are not present in amounts adequate to allow differentiation of hygroma versus atrophy on the basis of differences in signal intensity.

We describe a method of distinguishing between subdural hygroma and atrophy that depends on objective observation of whether the cortical veins cross the fluid collection over the convexities at some distance from the sagittal sinus (atrophy) versus whether the cortical veins reach the brain surface immediately adjacent to the sagittal sinus and are not seen crossing the fluid collection over the convexities (hygroma).

We refer to the visualization of the cortical veins within the widened CSF spaces over the cerebral convexities in cases of atrophy as the “cortical vein sign” and we have developed a theoretical model to explain the basis of the sign.
(Fig. 1). The purpose of this retrospective study was to verify our conclusion drawn from the model, namely, that the cortical vein sign is specific for atrophy and excludes a diagnosis of subdural hygroma.

**Materials and Methods**

We retrospectively reviewed MR scans of patients with extracerebral fluid collections isointense to CSF utilizing standard radiographic criteria to separate these into two groups, i.e., subdural hygroma or atrophy. We accumulated cases until a total of 10 cases in each group was achieved. Criteria for inclusion in the subdural hygroma group were as follows (5, 6): 1) isointense semilunar or crescent shaped extracerebral fluid collection limited to a cerebral convexity; 2) mass effect manifested by flattening of cortical surface of the brain and relative lack of visualization of cortical sulcal spaces adjacent to the fluid collection; 3) unilaterality where applicable; and 4) ventricular compression.

Criteria for inclusion in the atrophy group were as follows (7, 8): 1) symmetrical enlargement of ventricles, basal cisternal spaces, and cortical sulcal spaces; and 2) lack of mass effect on the brain adjacent to the fluid collection.

We purposely eliminated historical data such as history of trauma or dementia, or the age of the patient, as criteria in an effort to be more objective on the basis of radiologic criteria alone.

We realize that this selection process is imperfect because of the fact that the diagnoses were not generally confirmed by surgery or autopsy in our series.

The relative location of where the cortical veins cross the fluid collection was reviewed in each case. That is, we analyzed whether the veins randomly crossed the CSF space even over the convexities and then followed a juxtcortical location, or whether the veins crossed the fluid collection in an immediate parasagittal location and assumed a juxtcortical location lateral to that point. In the latter group, the veins were not seen crossing the fluid space over the convexities.

Several additional cases of subdural hematoma at various stages of blood product degradation were reviewed to confirm the inwardly displaced cortical veins over the convexities in that condition previously described by Fobben et al (4).

The majority of the examinations were performed on a 1.5-T Signa scanner (GE, Milwaukee, WI) utilizing the standard head coil. Sagittal T1-weighted spin-echo images (800/20/1) and axial proton-density and T2-weighted images (2000/20,80/1) were utilized. Other parameters included 24.0-cm field of view, 5-mm section thickness with 2.5-mm gap, and 256 X 192 matrix. One scan performed outside this institution on a Picker 0.5-T scanner is also included in the illustrations.

**Results**

All cases of cerebral atrophy showed cortical veins or their branches within widened CSF-filled subarachnoid spaces over the hemispheric convexities (Fig. 2). These veins were noted at several
locations within the widened CSF spaces. Many of these veins appeared to be on a long traverse across the CSF space (Fig. 3) while others appeared to be "floating" or partially retracted from their normal sulcal positions. In particular, veins were frequently demonstrated on the "anticortical" (calvarial) surface of the widened CSF spaces on sagittal T1-weighted images. These "anticortical" veins lie at a constant depth and are presumably located within CSF just beneath the arachnoid membrane (Fig. 4).

All cases of subdural hygroma showed the fluid collections over the convexities to be void of vascular structures. Cortical veins in these cases could be seen only at the margins of the displaced cortex (Figs. 5 and 6).

All the cases of subdural hematoma with signal intensities different than that of CSF showed a location of the cortical veins in a similar location to the subdural hygromas, and none showed cortical veins randomly traversing the fluid collections over the convexities.

Discussion

On the convexities of the normal brain, the superficial cortical veins lie within the cortical sulci in close approximation to the underlying pia and the overlying arachnoid/dura membranes. These veins drain by branches that must first penetrate the overlying arachnoid and then the inner layer of the dura to reach a dural venous sinus. In the case of the cerebral convexity veins, this is the superior sagittal sinus (9).

To determine whether the position of the superficial cortical veins could be used to distinguish subdural hygroma from atrophy, the literature was reviewed and a theoretical model was developed (Fig. 1).

The model was based on the following lines of reasoning:

1. In cerebral atrophy, the brain cortex gradually recedes from the arachnoid/dura membranes. Since the arachnoid remains closely applied to the dura, recession of the cortex results in widening of the subarachnoid space (10). This space, however, retains its normal complement of cortical veins and branches. There is probably some "tethering" of these veins by their downstream arachnoid penetrations and dural attachments, near the sagittal sinus. Otherwise, the veins are obliged to continue to traverse the ever-widening subarachnoid space as the cortex recedes. These venous structures are, in fact, ubiquitous in the subarachnoid space and are easily visible on MR scans as "flow voids" or areas of high signal intensity depending upon the venous flow rate and the parameters used (11) (Figs. 2-4).

2. In the presence of subdural hygroma or hematoma, the subdural space is expanded by fluid between the dura and the arachnoid, forcing the arachnoid membrane against the cerebral cortex (4, 12, 13). This relatively closes the sub-
arachnoid space, trapping the cortical veins within it and displacing the arachnoid membrane and cerebral cortex as a unit away from the overlying dura. This leaves a homogenous fluid collection between the dura and arachnoid that is void of visible vascular structures (Figs. 5 and 6). The cortical veins that are visualized following the cortical surface of the brain at the cerebral convexities (Figs. 5 and 6) still have to reach the sagittal sinus, but they do so only far medially in the parasagittal area as may be observed on the venous phase of arteriograms with subdural fluid collections of any type (13). In addition, in sub-

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are no longer present could theoretically produce a similar appearance.

Morphologically, the recognition of veins along the margin of the displaced cortex in extracerebral fluid collections is a valuable sign long known to angiographers (13) and reported by others (4). However, the true significance of this sign is that the veins are seen only at the cortical margins in extracerebral fluid collections (Figs. 5 and 6). In atrophy, they are not only at the margins but also within the fluid and at the anticortical surface of the fluid.

Conclusion

The conclusion to be drawn from the model and this study is that, if cortical veins can be demonstrated deep within a convexity fluid collection, then that fluid must be CSF within dilated subarachnoid spaces and not subdural hygroma.

We have called the visualization of cortical veins and their branches deep within low signal intensity convexity fluid collections “the cortical vein sign.” We believe this sign to be prima facie evidence of atrophy and that the sign excludes the diagnosis of subdural hygroma in the region of interest.

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References