Measurement ofTransependymal Bulk CSF Flow in Communicating Hydrocephalus

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Introduction

Periventricular hyperintensities in FLAIR T2W images are often seen in hydrocephalous. It is associated with edema or CSF retention in the white matter. The origin for the increased interstitial water content could be local, either vascular or cellular, or ventricular by transependymal bulk CSF flow. Identifying the possible origin for these hyper intensities in a specific patient could help establish the correct diagnosis and treatment. We tested the hypothesis that ventricular-to-parenchyma transependymal flow is associated with reversed net CSF flow through the aqueduct (e.g., negative net aqueductal outflow). While measurement of net aqueductal flow is challenging due to the small net flow compared with the oscillatory flow component, Huang et al (1) demonstrated reliable measurements using high temporal and spatial resolution cine phase contrast combined with the pulsatility based automated lumen segmentation (PUBS) technique (2).

Method

Net aqueductal flow (NAF) was measured in 25 subjects with intact ventricles (no shunt) (five healthy, ten Chiari I malformation patients, and ten patients with communicating hydrocephalous of various causes) and in two pediatric patients who had an open extra ventricular drainage (EVD) during the scan. In these cases, the negative NAF was compared with their CSF drainage rate. In one of the healthy subjects scanning was repeated 4 times to establish reproducibility. CSF velocities were imaged using retrospectively gated cine phase contrast technique with FOV of 10-12 cm, slice thickness of 6 mm, matrix size of 384x190 interpolated to 512x512, velocity encoding of 10-20 cm/sec, and data reconstructed into 32 phases. Automated segmentation of the aqueductal lumen area was obtained using a software tool utilizing the PUBS technique (2). Additional anatomical imaging, including FLAIR, was obtained to assess for the presence of hyper intensities in the periventricular regions.

Results

Repeated measurement variability (%SD) was 11%. All healthy subjects and Chiari patients had positive net CSF outflow (0.41 ± 0.25 and 0.52 ± 0.35 mL/min, respectively). Negative net flow was found only in the hydrocephalous group in four of the 10 patients, in a patient who underwent radiation for pineal tumor (fig 2a), in two patients with suspected diagnosis of iNPH (fig 2b and 1c), and in a patient with posterior fossa cyst (fig 2d). The NAF ranged from -1.3 to -0.20 mL/min. Negative NAF was also found in the two pediatric patients with the open EVD (-0.12 and -0.86 mL/min, respectively). These patients' average CSF drainage rates were 10 and 20 cc per hour, respectively. Hyperintensities in the periventricular white matter regions were present in all the 4 patients with negative NAF.

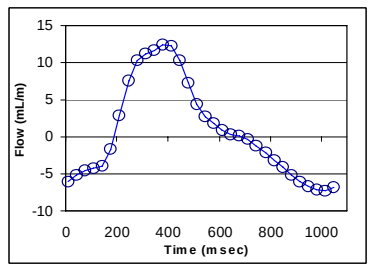


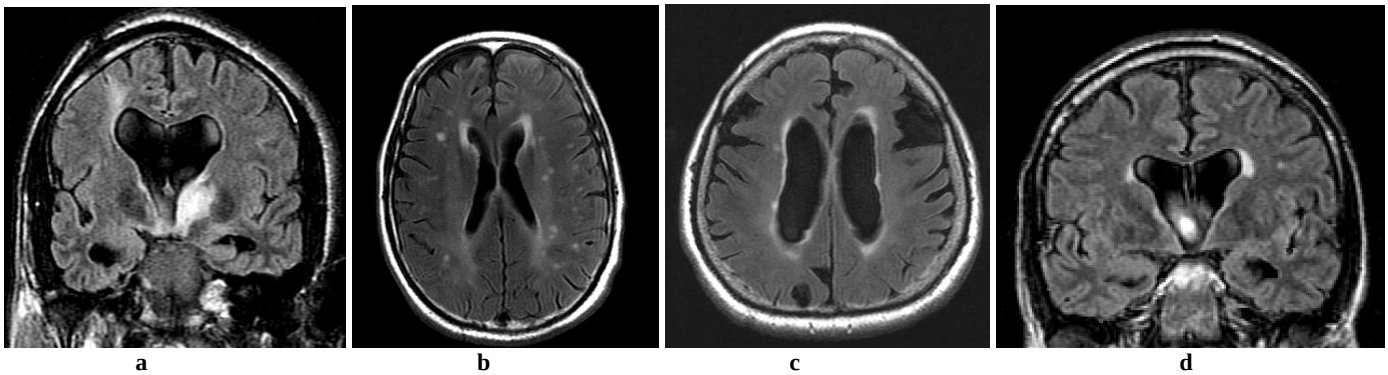
Fig. 1 High temporal resolution aqueductal CSF flow waveform

Conclusion

High temporal and spatial data acquisition and reliable automated lumen segmentation yield reliable measurement of the aqueductal bulk CSF flow component. A finding of negative NAF provides an indirect evidence for ventricular-to-parenchyma transependymal bulk flow. This measurement has potentially very low false negative rate as no negative NAF were found in the control subjects (healthy and the Chiari patients) Finding of negative NAF in the two patients with CSF drainage through an open EVD and the fact that all 4 patients with negative NAF had hyperintensities in the periventricular regions further support the reliability of this measurement.

References

1. Huang et al. Radiology 2004;233:603-608
2. Alperin and Lee. Mag. Reson in Med. 2003; 49:934–9443.



a **b** **c** **d**

Fig 2. FLAIR images of the patients with negative NAF. All demonstrated strong hyperintensities in the white matter periventricular regions.