

POST-CONTRAST FLAIR IMAGING IN A PATIENT WITH POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES)

I. Çelebi¹, M. Vural², B. Kahyaoğlu³, T. Güneysu⁴

We herein present a case of delayed enhancement of CSF on fluidattenuated inversion recovery (FLAIR) imaging in a patient with posterior reversible encephalopathy syndrome (PRES). In our case despite the settled clinical setting of PRES initial MR scan was negative and on repeated FLAIR imaging increased CSF signal intensity was more conspicuous than subtle cortical involvement.

Key-word: Brain, diseases.

Case report

A 65-year-old woman was admitted to the emergency service with sudden speech disturbance, difficulties in finding words, and numbness in right arm. Other findings of her neurologic examination were normal.

Blood pressure on admission was 196/98 mmHg. There was no evidence of renal dysfunction by laboratory tests. MR imaging of brain and contrast enhanced (0.1 mmol/kg gadodiamid) supraaortic MR-angiography were performed. MR findings of brain were unremarkable, apart from periventricular white matter signal intensity consistent with chronic microangiopathic ischemic changes (Fig. 1A). Diffusion weighted images and apparent diffusion coefficient maps demonstrated no restricted diffusion. On MR-angiography supraaortic arterial system was unremarkable. Transient ischemic attack was suspected on the basis of clinical presentation and essentially negative MRI of the brain and anticoagulant therapy was started.

Eight hours after the initial scan, MR examination of the brain was repeated because of persistent clinical statement. Unenhanced FLAIR images demonstrated elevated signal intensity throughout the subarachnoid spaces over both hemispheres and symmetric subtle cortical hyperintensity in the parietal and occipital lobes and inferior temporal-occipital junction, which had not been present on the initial scan (Fig. 1B). Subarachnoid hemorrhage was suggested as a possible explanation for the CSF signal elevation,

but subsequent CT examination was negative. The persistent clinical features and FLAIR imaging findings suggested PRES in the setting of hypertensive attack and anticoagulant therapy was replaced with intravenous Nimodipin therapy. Rapid clinical improvements were seen by the day two and follow-up FLAIR imaging revealed normal signal intensity of CSF and parenchyma (Fig. 1C).

Discussion

PRES is a neurotoxic state and typically characterized by headache, altered mental functioning, seizure and visual loss. The typical imaging finding is symmetric cortical and subcortical edema with a predominantly posterior distribution. The most common causes of PRES are preeclampsia/eclampsia, infection/sepsis, autoimmune disease, cancer chemotherapy, transplantation and hypertension. Although there has been significant research regarding the pathophysiology of PRES it continues to be debated. Two opposing hypotheses exist. The earlier theory suggests that overreaction of brain autoregulation results in reversible vasoconstriction, and subsequent reversible ischemia. The newer and more favorable second theory suggests that severe hypertension exceeds the limits of autoregulation resulting in injury to the capillary bed and subsequent hyperperfusion and hyperpermeability of blood-brain barrier. Hamilton et al. (1) presented a case of PRES with delayed enhancement of CSF and speculated that this finding provides additional

evidence in support of newer theory of hypertension/hyperperfusion.

In many institution FLAIR pulse sequence has become a part of routine MR examination protocol due to CSF nulling and heavy T2 weighting. It is well known that varied pathologies increasing the CSF protein level or cellularity may result in increased signal intensity in CSF on FLAIR MR imaging and this signal change is more conspicuous than on standart T1 and T2 weighted spin echo sequences. CSF signal intensity changes have been observed with major disruptions of the blood-brain barrier in studies involving neurologic disorders such as ischemic stroke, epilepsy, tumors, acute inflammatory/infectious causes and vascular malformations (2, 3). In the literature, it is reported that the weakly paramagnetic effect of supplemental oxygen results in reduction of CSF T1-weighted relaxation time and subsequent high signal intensity on FLAIR images (4). However, our patient did not receive supplemental oxygen therapy. Artifact-related causes of hyperintensity on FLAIR images include; CSF pulsation, vascular pulsation, magnetic susceptibility and motion artifact (5). It can not be CSF pulsation artifact, since such artifacts tend to occur in the basal, prepontine, and cerebellopontine angle cisterns and in sections containing foramina of the ventricular system. These artifacts are less common and less intense over the convexities of the cerebral hemispheres, where CSF flow is diminished (6). Susceptibility artifacts and resultant FLAIR subarachnoid hyperintensity most commonly occur in the presence of metal, but more subtle local incomplete nulling of CSF can be caused by air in the paranasal sinuses and the temporal bones. The hyperintensities of our patient is at parietal and occipital lobes away from temporal bones and paranasal sinuses. It can not be vascular or motion artifact since they are not

From: 1. Radiology Clinic, Sisli Etfal Training and Research Hospital, Istanbul, 2. Radiology Clinic, 3. Neurology Clinic, VKV American Hospital, Istanbul, 4. Radiology Clinic, Medmar Radiodiagnostic Center, Istanbul, Turkey.

Address for correspondence: Dr I. Çelebi, M.D., Radiology Clinic, Şişli Etfal Training and Research Hospital, 34377 Şişli, Istanbul, Turkey.
E-mail: irfancelebi@gmail.com

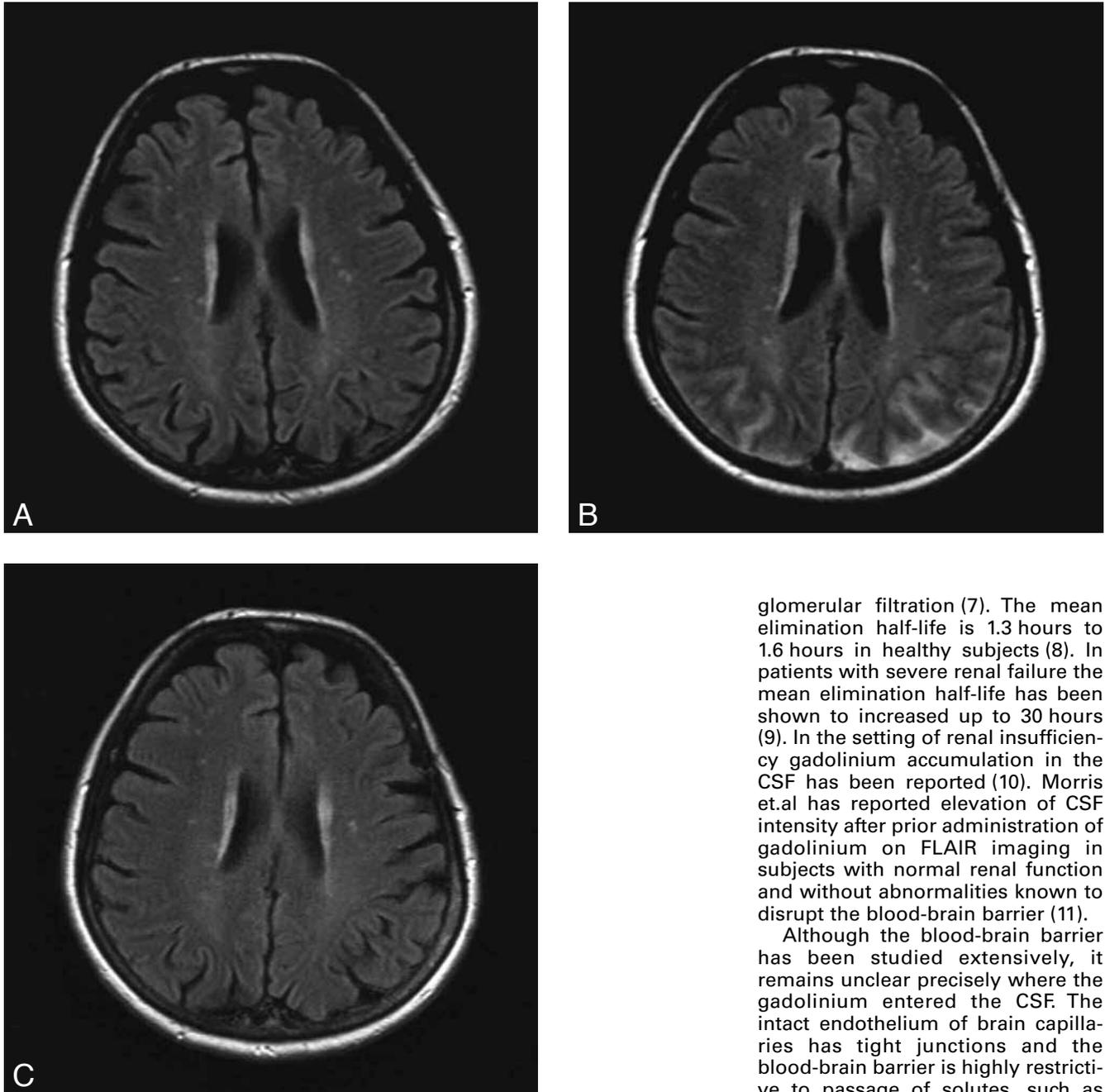


Fig. 1. — A. Initial axial FLAIR image is unremarkable apart from microangiopathic white matter intensities. B. Repeat FLAIR imaging reveals extensive CSF enhancement and mild cortical involvement with a predominantly posterior distribution. Cortical-based subtle lesions are difficult to appreciate owing to the adjacent hyperintense CSF. C. Follow-up FLAIR imaging obtained two days after the initial scan demonstrates complete resolution of lesions.

periodic with no synchrony between the phase-encoding steps.

In our case, the imaging which repeated 8 hours after the first evaluation was carried out using the same device and same technical parameters. The hyperintensity in posterior cerebrospinal fluid areas which was not seen in the initial evaluation and

appeared after 8 hours takes us away from the opinion of artefact-induced hyperintensity. In the third follow-up examination, the same parameters were used again and CSF hyperintensity was disappeared.

The main pathway of elimination of different gadolinium chelates is

glomerular filtration (7). The mean elimination half-life is 1.3 hours to 1.6 hours in healthy subjects (8). In patients with severe renal failure the mean elimination half-life has been shown to increased up to 30 hours (9). In the setting of renal insufficiency gadolinium accumulation in the CSF has been reported (10). Morris et.al has reported elevation of CSF intensity after prior administration of gadolinium on FLAIR imaging in subjects with normal renal function and without abnormalities known to disrupt the blood-brain barrier (11).

Although the blood-brain barrier has been studied extensively, it remains unclear precisely where the gadolinium entered the CSF. The intact endothelium of brain capillaries has tight junctions and the blood-brain barrier is highly restrictive to passage of solutes, such as gadolinium.

Blood-CSF barrier with fenestrated endothelium at the choroid plexus level and the circumventricular organs without the tight junctions in the capillary endothelium may allow the diffusion of gadolinium under certain conditions (12, 13). Ciliary body of eye has fenestrated capillary endothelium like choroid plexus and intraocular T1 shortening effect after contrast administration was reported (14, 15).

In our case despite the settled clinical setting of PRES initial MR scan was negative and on repeated FLAIR imaging increased CSF signal intensity was more conspicuous than subtle cortical involvement.

References

1. Hamilton B.E., Nesbit G.M.: Delayed CSF enhancement in posterior reversible encephalopathy syndrome. *AJNR*, 2008, 29: 456-457.
2. Levy L.M.: Exceeding the limits of the normal blood-brain barrier: Quo vadis gadolinium? *AJNR*, 2007, 28: 1835-1836.
3. Taoka T., Yuh W.T.C., White M.L., Quets J.P., Maley J.E., Ueda T.: Sulcal hyperintensity on fluid-attenuated inversion recovery MR images in patients without apparent cerebrospinal fluid abnormality. *AJR*, 2001, 16: 519-524.
4. Anzai Y., Ishikawa M., Shaw DW., Artru A., Yarnykh V., Maravilla K.R.: Paramagnetic effect of supplemental oxygen on CSF hyperintensity on fluid-attenuated inversion recovery MR images. *AJNR*, 2004, 25: 274-279.
5. Stuckey S.L., Goh T.D., Heffernan T., Rowan D.: Hyperintensity in the Subarachnoid Space on FLAIR MRI. *AJR*, 2007, 189: 913-921.
6. Rydberg J.N., Hammond C.A., Grimm R.C., et al.: Initial clinical experience in MR imaging of the brain with a fast fluid-attenuated inversion-recovery pulse sequence. *Radiology*, 1994, 193: 173-180.
7. Staks T., Schuhmann-Giampieri G., Frenzel T., Weinmann H.J., Lange L., Platzek J.: Pharmacokinetics, dose proportionality, and tolerability of gadobutrol after single intravenous injection in healthy volunteers. *Invest Radiol*, 1994, 709-715.
8. Joffe P., Thomsen H.S., Meusel M.: Pharmacokinetics of gadodiamide injection in patients with severe renal insufficiency and patients undergoing hemodialysis or continuous ambulatory peritoneal dialysis. *Acad Radiol*, 1998, 5: 491-502.
9. Shellock F.G., Kanal E.: Safety of magnetic resonance imaging contrast agents. *J Magn Reson Imaging*, 1999, 10: 477-484.
10. Rai A.T., Hogg J.P.: Persistence of gadolinium in CSF: a diagnostic pitfall in patients with end-stage renal disease. *AJNR*, 2001, 22: 1357-1361.
11. Morris J.M., Miller G.M.: Increased signal in the subarachnoid space on fluid-attenuated inversion recovery imaging associated with the clearance dynamics of gadolinium chelate: a potential diagnostic pitfall. *AJNR*, 2007, 28: 1964-1967.
12. Davson H.: Dynamic aspects of cerebrospinal fluid. *Dev Med Child Neurol(Suppl)*, 1972, 27: 1-6.
13. Carpenter M.B.: *Coe Text of Neuroanatomy*, (3rd ed) Baltimore: Williams & Wilkins, 1985: 1-19.
14. Kanamalla U.S., Boyko O.B.: Gadolinium diffusion into orbital vitreous and aqueous humor, perivascular space, and ventricles in patients with chronic renal disease. *AJR*, 2002, 179: 1350-1352.
15. Akın K., Agildere A.M., Sezer S.: Hyperintense signal in the subarachnoid space and ocular globes in brain MRI of a patient with acute renal failure. *Eur J Radiol*, 2005, extra volume 55 issue 2:41-45.