

tern was bilateral and fairly symmetric (Fig. 1). MRI could not be done due to his pacemaker. His blood pressure was subsequently brought down to 128/72 over a 24 hour period at which point there was significant improvement of his symptoms. Repeat CT Head with contrast done the following day showed diffuse white matter low attenuation with sulcal effacement involving, the splenium of the corpus callosum, without abnormal enhancement (Fig. 2). His aphasia continued to improve over several days at which time his speech



dysfunction. There is blood brain barrier breakdown and extravasation of fluid resulting in edema. In addition to these primary processes secondary events may occur which include focal cerebral vasospasm or thrombosis leading to brain infarction and hemorrhage. The most frequent MRI findings associated with PRES are hyperintensities on fluid-attenuated inversion recovery (FLAIR) images in the parieto-occipital and posterior frontal cortical and subcortical white matter. Less commonly, the brain stem, basal ganglia, and cerebellum are involved. Atypical imaging appearances would include contrast enhancement, hemorrhage and restricted diffusion on MRI [2].



Fig. (2). CT Head with contrast done the following day showed diffuse white matter low attenuation with sulcal effacement, involving the splenium of the corpus callosum, without abnormal enhancement.

showed only very mild circumlocutions and word finding difficulty. The right visual field deficit resolved. His mild neurologic deficits continued to improve and had resolved completely at two month follow up. Repeat CT head with and without contrast done four months after presentation showed that the symmetric white matter hypodensities had completely resolved. There was no evidence of enhancing masses or acute infarcts (Fig. 3).

Posterior Reversible Encephalopathy Syndrome (PRES) was first described in 1996, presenting with headache, seizures, changes in mental status, and various visual deficits [1]. It has been characterized on brain imaging as symmetric parieto-occipital edema but can occur with varying imaging appearances. It describes a usually reversible neurologic syndrome with a variety of presenting symptoms from headache, altered mental status, seizures, vomiting, diminished spontaneity and speech and abnormalities of visual perception and visual loss. Causes of PRES include hypertension, eclampsia, immunosuppressive medications, hypercalcemia, thrombocytopenic syndromes, amyloid angiopathy, systemic lupus erythematosus and renal failure. The pathophysiology of PRES is related to a hyperperfusion state whereby elevated capillary hydrostatic pressure leads to autoregulation

Fig. (3). CT head with contrast done four months after presentation showed that the symmetric white matter hypodensities had completely resolved.

It is important to note that the calcarine and paramedian occipital lobe structures are usually spared, a fact that distinguishes PRES from bilateral infarction of the Posterior cerebral artery distribution [1]. The reason that PRES tends to favor the posterior circulation is not clear. However, it may be due to a lack of sympathetic innervation at the level of the arterioles supplied by the vertebrobasilar system compared with the anterior circulation [2].

Our patient presented with elevated blood pressure, CT characteristics of PRES but a highly circumscribed neurologic syndrome (Wernicke's aphasia without hemiparesis) suggestive of a cardioembolic stroke affecting the left MCA territory. That is, PRES mimicked a focal stroke syndrome. The importance of recognizing this possibility is that his deficits resolved with blood pressure control, while other treatments, such as intensifying his anticoagulation would have been inappropriate. In addition, allowing his blood pressure to remain elevated as is often done in the setting of an acute stroke might have perpetuated the underlying pathophysiology of PRES leading to a worse clinical outcome. It is for this reason that PRES needs to be recognized quickly and treated appropriately.

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