Posterior reversible encephalophaty syndrome: a case related to blood transfusion

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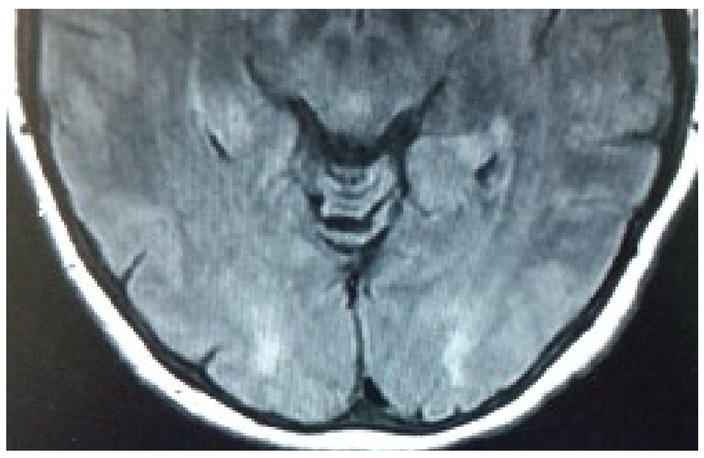
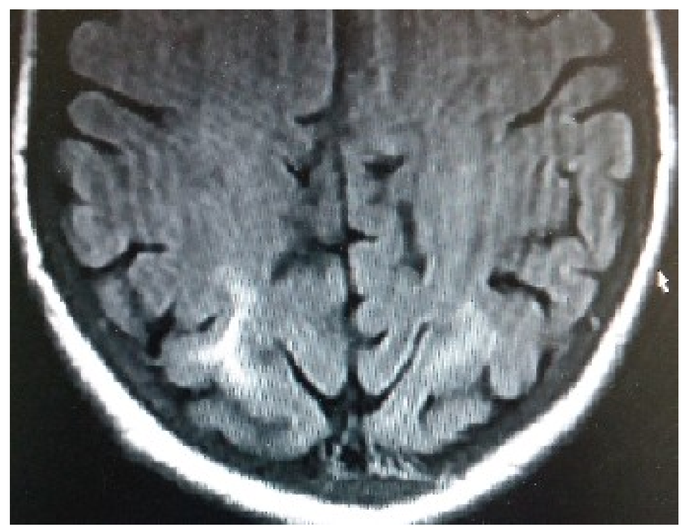
Posterior reversible encephalopathy syndrome (PRES) is a clinical radiological entity characterized by visual disturbances, epileptic seizure, headache and cerebral edema, especially in the posterior brain regions, often due to increased blood pressure. Two patogenetic hypothesis are proposed: loss to brain autoregulation due to hypertension and vasogenic edema or endothelial damage and cytotoxic edema.
Causes described are: autoimmune diseases (mostly systemic lupus erythematous), chemioterapic and immunosuppressant drugs, eclampsia, sickle cell disease, nephrotic syndrome, blood transfusion, iodized contrast administration.
The appearance of posterior reversible encephalopathy syndrome after blood transfusion is rare and has only been reported in ten reports to our knowledge. Most of the cases involved middle-aged female patients with chronic severe anemia who received large blood transfusion volumes within a short period of time. Symptoms of PRES often onset two to seven days after the blood transfusion.

We report the eleventh patient with PRES presumably secondary to blood transfusion.
A 60 years old hypertensive woman were admitted to medical division because of severe anemia (hemoglobin 3,5) and symptoms of acute ischaemia of legs due to peripheral hypoxia. She was treated with blood transfusion with restoration of hemoglobin values. After 2 days she accused mild occipital headache and visual disturbances. The neurological examination revealed only right incongruous homonymous hemianopia. The blood pressure was 160/90. A CT scan of brain showed mild hypodensity of left occipital lobe. The following day the patient presented two generalized epileptic seizures, treated with Levetiracetam. Fluid-attenuated-inversion-recovery MRI imaging revealed high intensity areas in the bilateral occipital and parietal lobes, compatible with vasogenic edema. Neurological disturbances completely recover after a week, without seizure recurrence. We think that, in this case, PRES was due not to hypertension (that was mild) but to an abrupt increase of hematocrit after chronic severe anemia.

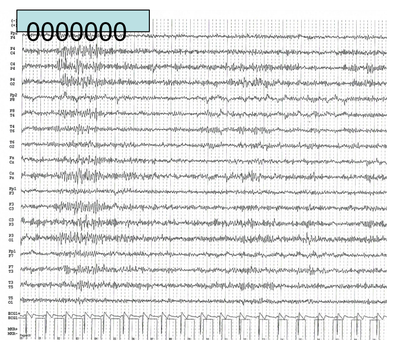
Pathogenesis of PRES after blood transfusion is not clear. It has been proposed that hypoxia causes elevation in brain capillary density sustained by increase of VEGF. That can increase endothelial permeability and could predispose to vasogenic edema. Blood transfusion seems to act as trigger to activate neutrophils and exacerbate dysfunction of the vascular endothelium, causing disruption of the blood brain barrier, and leakage of brain capillary resulting PRES. Another important element is considered the loss of hypoxic vasodilatation after hemoglobin restorage, that can drive to endothelial damage then vasogenic edema.
That changes are typical of rapid correction of chronic anemia and may not occur after transfusions in patients with acute blood loss. That suggests to consider caution with aggressive transfusions in setting of chronic anemia.



TWO MRIT2 weighted Images of posterior brain edema



EEG few days after seizures



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