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# Myoclonic seizures in patient with cortical laminar necrosis due from hypoglycaemia and anaemia

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**Introduction:** Cortical laminar necrosis is necrosis of neurons in the cortex of the brain in situations when supply of oxygen and glucose is inadequate to meet regional demands. This is often encountered in cardiac arrest, brain ischemia, global hypoxia, hypoglycemia, anemia, status epilepticus etc. Early cytotoxic oedema causes high signal seen on DWI. CLN is a permanent brain injury, radiologically characterized by high intensity cortical lesions on T1 and Flair weighted MRI image. Myoclonic seizures are brief, shock-like jerks of a muscle or a group of muscles. Usually they don't last more than seconds. There can be just one, but sometimes will occur within a short time. In epilepsy, myoclonic seizures usually cause abnormal movements on both sides of the body at the same time.

**Case presentation:** 61 years old woman, with many years history for arterial hypertension and diabetes. Two month before, patient was hospitalization on the Peja Regional Hospital, diagnosed with lacunar pontine infarct (by CT head scan). Laboratory parameters in that time were: SE=60/h, Urea=6.8, glycaemia=10.4, Triglycerides=1.3, Cholesterol=5.7, AST=16, ALT=15, RBC=4.66, WBC=10.4, HGB=136, HCT=38.8. Ten days before hospitalization in our clinic, she manifested conscious disturbance for about an hour, result of glycaemia showed that this was a hypoglycemic crisis (1 mmol/l). From that day continually she become more unconscious, tired, gait disability, loss of speech. At our clinic: SE=120/h. Glycaemia=6.48…7.98. Creatinine=91…113, Urea=18.2…14.7, Cholesterol=4.06, Triglycerides=1.59, Total bilirubin=8.8…3.03, AST=38…37, ALT=16…16, LDH=826…681, CK=61…44, Albumin=24.6…28.4, Total proteins=60…55.43, Calcium=2.1, Potassium=3.5, Natrium=150, Clor=114, CRP=41…37.4, Procalcitonine= , INR=1.1, PT=81%, PTT=29", Iron=20, RBC=3.3…3.4, WBC=15.2…16.3, HCT=27.4…28.3, HGB=8.5…8.8, PLT=340…320. Ct head scan: Irregular hypodense area in the pons and cerebellar region. MRT head scan: hypersinjal on flair FP bill, with water restriction sings on DWI – cortical laminar necrosis; lacunar chronic ischaemic lesion on the right of the pons (see images). Consulting: Hematologist: Dg. Anemia. Cardiologist: Dg.CVS stabile. Nephrologist: Dg.Oliguria. Endocrinologist: Dg.DM. ORL: nasogastric sound. During hospitalization at our clinic, patient begin to manifest myoclonic seizures, especially on both arms and face (see video). We treated her with isotonic solutions, vitamins, anticoagulants, antibiotics, antipyretics, substitution therapy, bicarbonates, hypertonic solutions, diuretics, antiepileptic and oxygen.

**Discussion:** We chose to present this case for some reasons: because Cortical Laminar Necrosis is relatively rare disease; at our patient we had three etiologic factors which can cause CLN (hypoglycemia, anemia, brain ischemia); and also associations between myoclonic seizures and frontal cortical leas ions (necrosis).

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