Toxic Encephalopathy

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Even if neurotoxic exposure is common the toxic encephalopathy is difficult to diagnose and can be difficult to treat. A good clinical history, proper clinical examination, relevant laboratory testing and to be open for the possibility of toxic exposure is crucial when dealing with these patients. Neuroimaging are in general neither sensitive nor specific for toxic causes of encephalopathy, but can in same situations help in rule in or rule out a toxic cause. In the evaluation of encephalopathy conventional MRI is the best modality to demonstrate the cerebral pathology. The examination should include sequences such as T1-weighted, T2-weighted and FLAIR and, of course, diffusion weighted imaging, and sometimes contrast administration will be helpful. The findings may vary depending on the toxic agent and the underlying condition. Abnormal findings might be seen predominately in the cortex, even if rare, or predominately in the white matter or be mixed. Typical findings are for example encephalopathy with diffuse brain edema which can be seen in different toxic exposures. Typical imaging findings of diffuse brain edema can be effacement of sulci, poor differentiation between gray and white matter, and effacement of the ventricles, which can be seen on both CT and MRI (Fig 1). Basal ganglia are often involved in heavy metals exposure especially manganese and bilateral basal ganglia involvement should always raise a suspicion of toxic etiology. Other findings are diffuse non-enhancing small white matter lesions or more confluent areas of white matter hyperintensity. The later can for example be seen in later stages after carbon monoxide (CO) exposure in which the patient present with bilateral white matter hyperintensities (on T2-weighted imaging) with or without basal ganglia involvement. Meningeal contrast enhancement or focal contrast enhancing lesions in the brain parenchyma are unusual. In posterior reversible encephalopathy syndrome (PRES), which have been reported with toxic exposure especially in children on multi chemotherapy, presents often with focal symmetrical parenchymal edema predominately in the parietal and occipital lobes but can also be present in the frontal lobes, and in the cerebellum (Fig 2a-b). Treatment with cyclosporine is often associated with PRES. The clinical symptoms may also vary depending on the degree of toxic exposure and if the exposure is acute or more chronic. Acute toxic encephalopathy present with symptoms and signs that includes mild confusion, attention deficits, seizures and coma. Patients with more chronic toxic encephalopathy may have present with milder insidious symptoms like mood disturbances, fatigue and cognitive dysfunction. Other common complains are memory disturbances and headache. Also the cerebral injury might give additional symptoms like injury to the basal ganglia may result in parkinsonism, injury to the cerebellum may result in ataxia and gait disturbances, and even if rare apraxia, agnosia, and aphasia can be noted in patients with focal cortical injury. Vincristine, organic mercury, interfereon and other chemotherapy agents can cause the syndrome of cortical blindness.

In the present lecture different toxic agents will be presented, their clinical symptoms and typical, if any, imaging findings will be described.
Fig 1. Axial CT in 23 year old male after Tylenol overdose demonstrates effacement of the sulci and lateral ventricles and poor differentiation between gray and white matter.

Fig 2a-b. Axial FLAIR images demonstrates the typical findings in a patient with PRES. Increased signal in the parietal and occipital lobes as well as signal abnormalities in the subcortical frontal white matter.