Letter to the editor

Neurogenic pulmonary edema – the Takotsubo syndrome of the lungs

With interest we read the article by Cho et al. about a 31 years old male who experienced an inhalation injury and CO-poisoning after a fire in his apartment (1). During hyperbaric oxygen therapy (HBOT), applied for the CO-poisoning, he experienced a single generalised tonic-clonic seizure followed by lung edema (1). The case report has a number of shortcomings and raises concerns.

We do not agree with the conclusion that lung edema resulted from HBOT (1). Acute lung edema after a generalised seizure may be the direct consequence of the seizure, as has been previously reported (2). Acute lung edema immediately after an acute, severe neurological injury, such as acute meningitis, subarachnoid bleeding, seizures, ischemic stroke, intracerebral bleeding, epidural bleeding, traumatic brain injury, multiple sclerosis exacerbation, or vertebral artery dissection, is termed neurogenic pulmonary edema (NPE) (3).

An argument in favour of NPE as the consequence of a stress-reaction to a serious cerebral event is the simultaneous occurrence of NPE and TTS in a patients with meningioma (4), with ischemic stroke and thrombolysis (5), in patients with brainstem strokes (6), in a patient with epidural hemorrhaghe (7), in patients with subarachnoid bleeding (8), and in patients with brainstem hemangioblastoma (9).

A further shortcoming of the study is that no MRI of the brain was carried out. CO-poisoning is well known to cause hyperintensities on diffusion weighted imaging (DWI). In a retrospective study of 387 patients experiencing CO-poisoning, 26.9% had DWI hyperintensities on cerebral MRI (10). DWI lesions could easily explain why the patient developed a seizure. DWI lesions may not show up on cerebral CT scans why carrying out an MRI is obligatory. Since CO-poisoning goes along with cerebral lesions, they are more plausible to eplain the development of seizurs than excessive oxygen consumption.

The main therapeutic options for treating NPE are supportive measures and lowering of the intracranial pressure (11). Thus we should be informed if the index case had developed any clinical features or features on imaging of increased intracranial pressure. In case the patient had experienced an increase in intracranial pressure we should be informed which kind treatment was applied.

In a recent study it has been shown that endothelin-1 may be elevated in the cerebrospinal fluid (CSF) of patients with NPE due to infection with enterovirus-71 (12). We should be informed if endothelin-1 was elevated in the CSF of the index case. Diagnosing NPE is still challenging but transpulmonary thermodilution studies can be helpful to establish the diagnosis. We are interested to know if this diagnostic approach was applied and how NPE was diagnosed in the index case. Missing in the report is a description of the EEG.

Overall, this interesting case report could be more meaningful if an MRI of the brain had been carried out in the acute stage of CO-poisoning, if NPE triggered by the tonic-clonic seizure would have been more extensively discussed, and if treatment of NPE would have been more extensively described.

Josef Finsterer, MD, PhD
Krankenanstalt Rudolfstiftung, Messerli Institute,
Veterinary University of Vienna, Vienna, Austria
E-mail : fifigs1@yahoo.de

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Letter to the Editor Response

The editor recommends that the author respond to the reader's comments, but did not receive a response by the deadline.

Hiroshi Sakaue
Editor-in-Chief