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Case Report
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Cerebral Microbleeds in a Teenage Patient after Respiratory Failure

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Abstract

From Posttraumatic Stress Disorders (PTSD) to somatic disorders, when black dots show the difference.

Long term ventilation because of respiratory insufficiency with saturation problems is commonly seen on intensive care units. We report about a young adult who was admitted to the ICU after respiratory failure and was ventilated for almost two weeks. After extubation the patient felt depressed, showed a panic facial expression and was clinically diagnosed with posttraumatic stress disorder. The following performed cMRI showed multiple microbleeds bihemispheral and were not caused by hypertension or cerebral amyloid angiopathy which are common causes for cerebral microbleeds in elderly patients.

Cerebral microbleeds with predominance of the corpus callosum like they were seen in the above mentioned patient can typically be found in younger patients with diseases requiring intensive care, such as respiratory failure and are classified as critical illness-associated microbleeds.

Although the connection between these hemorrhages and respiratory failure is not completely understood there are several theories regarding the aetiological cause.

The following case report shows the patient‘s history with clinical findings and typical radiological ones of this rarer cause of cerebral microbleeds in comparison to the aetiology of microbleeds of known risk factors.

Nevertheless it points out the importance for radiologists of knowing about the diversity of cerebral microbleeds.

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Introduction

Cerebral microbleeds are a commonly seen phenomenon that is regularly detected as an incidental finding when interpreting cranial Magnetic Resonance Imaging (cMRI), as a result of the fact that they may persist for many years. In particular current bleeding-sensitive SWI sequences (susceptibility weighted imaging), with high sensitivity for the magnetic properties of blood, iron and calcifications, have led to a marked increase in sensitivity for the detection of cerebral microbleeds. Radiology is now confronted with the task of providing evidence for the aetiology of these microbleeds on the basis of specific patterns. A common cause of cerebral microbleeds is, for example, hypertension left untreated for many years or cerebral amyloid angiopathy. Here, MRI reveals typical patterns of hemorrhaging, with the basal ganglia and the thalamus predominantly being affected in the case of hypertensive microbleeds. The following case report describes the case of a young patient with multiple cerebral microbleeds that are not of the origins mentioned above [1,2].

Case Presentation

An 18-year-old male patient was admitted with severe respiratory insufficiency and evidence of pronounced bilateral infiltrates. An exogenous allergic alveolitis was surmised. Cortisone, high-intensity ventilation and repeated placement in the prone position ultimately led to stabilization of the condition. Weaning proved to be difficult. Oxygen denaturation down to 50% occurred, in reaction to which sedation and myorelaxation were induced and the patient had to be intubated. Over the further inpatient course, ventilation deteriorated repeatedly due to secretion obstruction and diffuses pulmonary hemorrhages. Permissive hypercapnia was tolerated. The patient was extubated after a total of 13 days (Figure 1a and 1b).

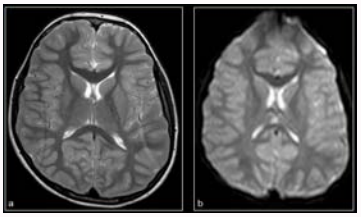

Figure 1: Axial T2wTSE (a) 11 years previously, without pathological findings. No increased signal intensity in isoB1000 (b) as evidence of a diffusion disorder of the brain tissue.

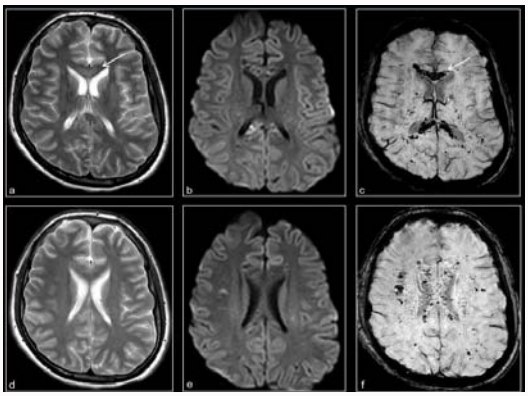

Figure 2: Axial T2wTSE (a, d) with demonstration of signal enhancements in the corpus callosum (arrow), hyperintense compared with the white matter. No increased signal intensity in isoB1000 (b, e) as evidence of a diffusion disorder of the brain tissue. On the susceptibility-weighted images (SWI) (c, f), the T2-weighted signal increases correspond to a signal cancellation (arrow). Demonstration of multiple susceptibility artefacts in the periventricular white matter. The changes correspond to multiple hemosiderin deposits and are interpreted as critical illness-associated microbleeds.

Neurologically, flaccid tetraparesis and reduced algesia were seen after extubation, and suspicion of a mild critical illness polyneuropathy was confirmed. In addition, the patient was suffering from hyperreactive delirium. Three days after extubation, the patient's mood was depressed and he was autonomically impaired, with a tendency to latent hypoventilation and a look of panic. Suspicion of a posttraumatic stress disorder was expressed, but cranial Magnetic Resonance Imaging (cMRI) was performed to exclude a somatic cause. In SWI of the cMRI, multiple bihemispherical hemosiderin deposits were seen, with a predominance in the corpus callosum. The T2-weighted images (Figures 2a-2f) showed an accompanying vasogenic edema. In a subsequent neuropsychological examination, impairments in the areas of speech, linguistic divergent thinking, memory and briefly delayed recall were seen, so that a cognitive disorder resulting from a disease, injury or functional disorder of the brain according to ICD-10 [F07.8] was assumed.

Discussion

Chronic hypertension and cerebral amyloid angiopathy are the two most common pathologies responsible for cerebral microbleeds in elderly patients [1].

However, other causes should also be taken into consideration, particularly in younger patients. Cerebral microbleeds with a predominance for the corpus callosum have also been described in younger patients with diseases requiring intensive care, such as disseminated intravascular coagulopathy, respiratory failure and viral infections, and in this connection are classified as critical illness-associated microbleeds [2,3].

The young age of the patient is also to be stressed in the case reported here. Patients aged between 27 and 63 years with similar findings can be found in the literature [2].

There are different theories regarding the aetiology of critical illness-associated cerebral microbleeds [2]:

Since hemorrhages caused by high altitudes show a similar distribution pattern [4], it stands to reason that hypoxia might be the point of origin. In most of the cases described, the patients were suffering from respiratory insufficiency [2-6], which means that an intermediate hypoxia is probable. Also in the patient described here, denaturation repeatedly occurred over the inpatient course. The hypoxia may, through hydrostatic or chemical effects, lead to a disturbance of the blood-brain barrier and thus enable the escape of erythrocytes [1].

A further attempt to explain the bleeding is Disseminated Intravascular Coagulopathy (DIC). In previously described cases, it was not always possible to provide information about a possible presence, but there were certainly signs pointing in this direction, such as a thrombocytopenia. However, this is not the case in our patient.

A true causal connection has yet to be established.

The clinical consequences of cerebral microbleeds often remain unnoticed. In a case control study, however, it was indeed shown that they are associated with cognitive dysfunctions [7].

Conclusion

The present case presentation shows the diversity of cerebral microbleeds with regard to the distribution pattern and their underlying aetiology. The detection and classification of cerebral microbleeds is an integral part of radiological diagnostics, as therapeutic concepts regarding the aetiology, e.g. in the case of chronic hypertension, result for the patient. The case described, with its critical illness-associated microbleeds, illustrates a rarer cause of the presence of multiple cerebral microbleeds.

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