A case of Marchiafava-Bignami disease
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Case Type: Clinical Cases
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Patient: 62 years, male

Clinical History:

A 62-year-old man with previous alcohol abuse was admitted after an episode with state of confusion and behaviour alterations. A MRI scan was performed.

Imaging Findings:

A 62-year-old man with a previous 20-year history of alcohol abuse developed alteration of consciousness with state of confusion and aggressivity. Four months later, after interruption of alcohol abuse, psychiatric evaluation diagnosed major depression with defects in memory for past events and concentration.

He underwent a MRI examination that showed small lacunar areas that involved the splenium and genu of corpus callosum (Fig 1, 2, 3) in presence of mild perilesional hyperintensity. Comparing the clinical history of the patient to neuroimaging findings, a diagnosis of Marchiafava-Bignami was suggested.

Discussion:

Marchiafava-Bignami disease (MBD) is a rare complication of chronic alcoholism (especially red-wine drinkers) mainly characterized by symmetrical demyelination and necrosis of the corpus callosum. The pathogenesis is unclear but the most accepted etiologic factor is the deficiency in vitamin B complex. Clinical manifestations can be different according to the rate of clinical progression.

In the acute state, seizures, alterations of consciousness and death may occur. The subacute state is usually characterized by mental confusion, behavioural disorders, memory deficits, cerebellar signs and interhemisferic disconnection. In the chronic state mild dementia may be diagnosed.

Clinicoradiologic subtypes of MBD have been recently identified in: Type A form characterized by severe alterations of consciousness and diffuse swelling of the entire corpus callosum on MRI, and a Type B with mild impairment of consciousness and small callosal lesions associated with favourable outcome.

Several studies demonstrated that CT and MRI are essential for the diagnosis, the differentiation of subtypes and severity, and the prediction of prognosis in vivo. As recognition and early treatment can improve clinical outcome and symptoms are nonspecific, neuroimaging findings can help to differentiate MBD from other reversible callosal lesions and also from alcohol related disorders as Wernicke encephalopathy (excluded in our patient, Fig. 4) and Korsakoff syndrome.

Reversible lesions of corpus callosum are mainly due to viral encephalitis, hypoglycaemic encephalopathy and hydrocephalus.

The common characteristic of these lesions is the magnetic resonance imaging pattern that disappears after the resolution of the causative factor. On MRI images, they show round small areas with high signal in FLAIR and T2 sequences and low or no signal in T1 sequences without contrast enhancement. In the acute phase diffusion weighted images (DWI) may appear restricted because of probably excitotoxic brain oedema caused by increasing of extracellular glutamate that binds NMDA (N-methyl-D-aspartic acid) receptors inducing calcium entry and finally
apoptosis without brain ischemia. To differentiate MBD lesions to other possible causes of callosal damage we have to consider their specific localization inside the corpus. The genu is usually the most involved structure followed by the splenium, lesions are symmetrically distributed. The entire corpus callosum may also be altered. In case of chronic damage corpus callosum degenerates and separates into three layers with necrotic cavities mainly in the middle layer (“sandwich necrosis”). On neuroimaging studies with MRI, lesions reveal high signal intensity in T2, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted image (DWI) with low apparent diffusion coefficient (ADC). Chronic lesions appear, in cases without administration of vitamin B, when necrosis occurs and show hyperintense signal on T2-weighted images and hypointense on T1-weighted images that can assume a lacunar aspect. The usefulness of contrast media remains unclear. Cortical involvement is extremely rare and when present, it is usually localized in lateral–frontal regions. In conclusion even if it is not possible to identify pathognomonic characteristics of MBD lesions, the clinical aspects and neuroimaging pattern may result helpful for the diagnosis.

**Differential Diagnosis List:** Marchiafava-Bignami disease type B in chronic phase.

**Final Diagnosis:** Marchiafava-Bignami disease type B in chronic phase.

**References:**


Description: Sagital T1 weighted image. Focal low signal intensity (arrow) in the splenium (left) and genu (right) of the corpus callosum. Origin:
Figure 2

Description: Axial fluid attenuated inversion recovery (left) shows focal hypointensity with perilesional hyperintensity in the splenium of corpus callosum (arrow). T2 weighted image (right) show focal hyperintensity in the same region. Origin:
Description: Axial fluid attenuated inversion recovery (left) shows focal hypointensity with perilesional hyperintensity in the genu of corpus callosum (arrow). T2 weighted image (right) show focal hyperintensity in the same region. Origin:
Description: Axial fluid attenuated inversion recovery demonstrate absence of any pathological lesions around the third ventricle and mammillary bodies, excluding the diagnosis of Wernicke encephalopathy.
Origin: